

Cranial Electrotherapy Stimulation

Its First Fifty Years, Plus Three

A Monograph

By

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A Summary Look at Studies of Cranial Electrotherapy Stimulation

by Ray B. Smith, Ph.D.

Executive Summary. Five meta-analyses were computed in order to summarize most of the studies of Cranial Electrotherapy Stimulation (CES) appearing in the U.S. scientific literature. They are categorized as follows:

<u>Syndrome Studied</u>	<u>No. of Studies</u>	<u>No. of Subjects</u>	<u>Average Improvement</u>
Insomnia	18	648	62%
Depression	18	853	47%
Anxiety	38	1,495	58%
Drug Abstinence	15	535	60%
Cognitive Dysfunction	13	648	44%

The **mechanism of action** is increasingly being seen as that of an adaptogen in that in a basic way CES appears to reduce stress that underlies many emotional and physical disorders.

A generic **clinical protocol** for the use of CES is to stimulate patients from 45 minutes to 1 hour daily, for three weeks. Stimulation should be at 100 pulses per second (pps) when that is available on the CES device being used. Otherwise follow the manufacturer's instructions.

No **placebo effect** in CES treatment has ever been found when that effect was specifically controlled for, with the possible exception of a negative placebo effect that one study found in non treated patients.

Introduction

Cranial Electrotherapy Stimulation provides small pulses of electric current across the head of patients for the FDA recognized treatment of depression, anxiety and insomnia. CES has been in clinical use in the U.S.A. since 1963 and in Europe since 1953. Hundreds of thousands of patients have been treated with CES over the years, and thousands presently use these prescription devices in their homes.

The original FDA grandfathered CES device, the Neurotone 101, pulsed at 50 and 100 Hz, biphasic, on a 20% duty cycle. There was no dc bias. While several different CES devices were used in the studies analyzed here, the FDA has declared them all to be substantially equivalent to the original grandfathered device, so the devices used in the various studies are not given in this report.

Meta-analysis is a way of combining the results of many separate studies to see the effectiveness of a treatment when different types of patients are studied, under different study conditions, with different study protocols, and who came to the various studies with differing symptoms accompanying their presenting problem.

The goal of clinical studies is always to first test the effectiveness of a potential treatment and secondly to discover which patients the treatment may be most effective in treating. Meta-analysis has the effect of allowing us to essentially study a larger number of patients than can

often be assembled for a single study, and the larger the combined study sample, the greater is the confidence that can be placed in the study outcome: that the study findings are true and accurate. Also, the more diverse the study group is in the combined sample, the more confident one can be in generalizing the study outcome to larger groups of patients outside the study. That is, it increases the range of potential types of patients that one can predict will be effectively treated with CES.

A total of 67 studies, involving 2,910 patients were analyzed in the five meta-analyses reported below. Some studies and some patients are represented in more than one analysis if in a given study of both anxiety and depression, for example, the symptoms were tested and evaluated separately. At the beginning of each analysis given below, the number of studies and the number of patients involved are provided for each of the symptoms or syndromes analyzed.

To many readers, the study design in which improvement scores are obtained is important. A variety of study designs were present in each of these analyses. These are broken down separately into sections, such as double-blind studies, open clinical studies and so forth for the reader who has this interest.

An important technical detail about meta-analysis is that some researchers measured depression in several ways pre- and post-study, such as patient's self rating scales, clinician's rating scales, physiological stress measures of one kind or another such as blood pressure, pulse rate, or even electroencephalograms in some cases. The patients responded to each of these at one or another level in terms of improvement following treatment. In the resulting analysis, all of these improvement scores were added and a mean improvement score is presented. Only the mean score is finally reported in the analyses given below.

Parenthetically, it might be noted that percent improvement scores cannot be added and divided directly to get a mean, in that they represent r correlation scores, which do not have a normal distribution. The improvement scores have to be converted to Z_r scores and a mean obtained, then that mean Z_r score converted back into a mean percent improvement score, which is reported at the end of each section of each table.

One last technical detail the reader should know is that most authors do not report change scores obtained in a study as percent improvement scores. They sometimes publish the statistical probability of the difference they obtained in scores pre- and post-treatment, such as $p=.05$, or $p=.01$ and so forth. Others print out F test scores or t scores, while others report out other statistics to show what happened in the study. Fortunately, along with the data reported, and given the number of subjects in the study, it is possible statistically to convert these various scores to r correlation scores, or percent improvement scores.⁴² The exception is the rare author who reports only the percent of their patients who improved by the end of the study. That cannot be converted to an r score, unfortunately, and therefore cannot be included in meta-analyses.

To reduce the amount of technical detail with which the reader will have to contend, only the next to last step in this conversion, the Z_r score, is shown in the tables below. Any supporting information the reader wishes to pursue can be found in the original published reports, which are referenced in the right hand column of each table.

Analysis of Studies

Sleep Studies. Eighteen studies, in which a total of 648 patients with various types of sleep disorders were treated with CES, were meta-analyzed in order to get a more confident look at the effectiveness of CES for treating this condition. The result of the analysis showed that the overall effectiveness of CES was 62% improvement, and when the studies were weighted in terms of the rigorousness of the study design employed, the improvement was found to be an even stronger 67%. The results also indicated that a wide range of sleep disorders can be expected to respond to CES treatment.

In the table below is a summary of the studies that were combined in the meta-analysis.

Table 1. CES Sleep Studies Completed Over the Past 43 Years

Study Design	Zr Score^a	No. Subjects	Measure Used^b	Reference
Double Blind	.388	27	EEG, Clinician's Rating	21
Double Blind	.908	30	Self Rating Scale	26
Double Blind	.875	60	Self Rating Scale	27
Double Blind	.590	18	Clinician's Rating	39
Double Blind	.448	21	Self Rating Scale	41
Double Blind	1.127	22	Clinician's Rating	44
Double Blind	1.528	10	EEG, Self Rating Scale	77
Totals	5.864	188		
Average	.838			
Effect Size^c	r = .69			
Single Blind	1.650	28	PRN Sleep Meds	16
Single Blind	.448	28	Clinician's Rating	18
Single Blind	.693	18	Clinician's Rating	47
Totals	2.791	74		
Average	.930			
Effect Size	r =.73			
Crossover	.678	19	Clinician's Rating	10
Crossover	.343	34	Self Rating Scale	33
Crossover	.343	34	Clinician's Rating	67
Totals	1.364	87		
Average	.455			
Effect Size	r =.43			
Open Clinical	.511	28	Clinician's Rating	11
Open Clinical	.633	186	Self Rating Scale	38
Open Clinical	.549	9	Clinician's Rating	46
Open Clinical	1.071	20	Self Rating Scale	72
Open Clinical	.590	56	Self Rating Scale	71
Totals	3.335	299		
Average	.671			
Effect Size	r =.59			

SUMMARY, ALL SLEEP STUDIES REPORTED ABOVE			
Grand Total	13.354	648	
Average	.724		
Total Effect Size	r =.62		

^a Since percent improvement scores can not legally be averaged, they are converted into Zr scores, averaged, and then converted back to percent improvement (effect size.)

^b Most of the rating scales, both by the patients and the clinicians were of published reliability and validity, though some were Likert Scales with face validity and assumed test-retest reliability, since the error variance within each patient was assumed to be relatively constant, and therefore carry over between pre and post testing.

^c Effect size, here, is a statistician's basic estimate of the overall percentage improvement by the patients as a result of the treatment

Discussion of the Sleep Studies

In several of the studies, sleep was but one symptom within a larger syndrome, so that in almost one fourth of the patients, fibromyalgia was the presenting syndrome, while in another fourth of the studies the drug abstinence syndrome was the presenting diagnosis. In the most, however, insomnia, alone, was the presenting diagnosis. It is also interesting that the two studies that blinded the patients had the best results. If we omit the results obtained in the difficult cross over designed studies, the average treatment effect rises to an impressive 67%.

As will be noted later (see Study Designs, page 16), we learned early on in CES work to stay clear of the cross over design, after we discovered that the improvement begun by a week or so of CES treatment continues after treatment is stopped. Many otherwise good studies were lost early on due to that effect, and one can see in the table above that two of the three crossover studies produced the lowest response rate of that found in any of the other studies.

One clinical detail we learned early on, is that patients who have not been sleeping well when they enter a study – most of them, by definition – sometimes make up for lost REM sleep during CES treatment and have the most vivid, most colorful dreams they have ever had. We learned to warn study participants of this in advance, since some earlier patients associated this with incipient schizophrenia or some other serious mental condition. Once alerted to the possibility they have always looked forward to the effect with real anticipation, and are disappointed if it doesn't happen.

Depression Studies

Eighteen studies were analyzed, in which a total of 853 patients were treated with CES for depression. The result of the analysis showed that the overall effectiveness of CES was 47% improvement. The results indicated that various types of depression, which accompany a wide range of clinical syndromes can be expected to respond, sometimes dramatically to CES treatment.

In the table below is a summary of the 18 studies that were combined in the meta-analysis.

Table 2. CES Depression Studies Completed Over the Past 36 Years

Study Design	Zr Score^a	Presenting Group	No. Subjects	Measure Used^b	Reference
Double Blind	1.099	Substance Abuse	29	Clinical Rating Scales	2
Double Blind	.283	Substance Abuse	20	Self Rating Scale	24
Double Blind	.255	Psychiatric Inpatients	11	Clinical Rating Scale	26
Double Blind	.310	Closed Head Injured	21	Self Rating Scale	64
Double Blind	.321	Fibromyalgia	60	Self Rating Scale	28
Double Blind	.511	Psychiatric Outpatients	18	Clinician's Rating	43
Double Blind	.900	Insomnia, Anxiety	17	Clinician's Rating	33
Totals	3.679		176		
Average	.526				
Effect Size^c	r = .48				
Single Blind	.486	Psychiatric Outpatients	22	Clinical Rating Scales	44
Single Blind	.881	Substance Abuse	72	Self Rating Scale	61
Totals	1.367		94		
Average	.684				
Effect Size	r =.60				
Crossover	.219	Psychiatric Inpatients	23	Clinician's Rating	10
Crossover	.929	Insomnia, Depression	28	Self Rating Scale	18
Totals	1.148		51		
Average	.574				
Effect Size	r =.52				
Open Clinical	.354	Graduate Students	54	Self Rating Scale	30
Open Clinical	.365	Fibromyalgia	20	Self Rating Scale	70
Open Clinical	.266	Fibromyalgia	60	Self Rating Scale	71
Open Clinical	.662	Pain, Depression	318	Self Rating	58
Open Clinical	.350	Psychiatric Outpatients	9	Clinical Rating Scale	45
Open Clinical	.549	Chronic Pain, Depression	48	Serum Analysis	54
Open Clinical	.332	ADHD	23	Self Rating Scale	57
Totals	2.878		532		

Average	.411				
Effect Size	r =.39				
SUMMARY, ALL DEPRESSION STUDIES REPORTED ABOVE					
Grand Total	9.072		853		
Average	.504				
Total Effect Size	r =.47				

^a r correlation scores, representing percent improvement, are obtained mathematically from the study outcomes presented by the authors.

^b Most of the rating scales, both by the patients and the clinicians were of published reliability and validity.

^c Effect size, here, is a statistician's basic estimate of the overall percentage improvement by the patients as a result of the treatment

Discussion of Depression Studies

In many of the studies, as noted above, depression was but one symptom within a larger presenting syndrome. For example in many of the patients, fibromyalgia was the presenting symptom, while in another large group of studies substance abuse (drug abstinence syndrome) was the presenting diagnosis. The presenting syndrome or type of patient is given in column three of the table. In all of the studies, however, depression was a major diagnosis within the presenting syndrome or group.

Anxiety Studies

Thirty-eight studies were analyzed, in which a total of 1,495 patients were treated with CES for anxiety. The result of the analysis showed that the overall effectiveness of CES was 58% improvement. The results indicated that various types of anxiety, which accompany a wide range of clinical syndromes, may be expected to respond, often dramatically to CES treatment.

Table 3. CES Anxiety Studies Completed Over the Past 36 Years

Study Design	Zr Score^a	Presenting Group	No. Subjects	Measure Used^b	Reference
Double Blind	.950	Substance Abuse	47	Clinical Rating Scales	2
Double Blind	.412	Outpatient Psychiatric Abuse	32	Self Rating Scale	14
Double Blind	.365	Substance Abuse	28	Self Rating Scale	16
Double Blind	.549	Outpatient Psychiatric	28	Clinical Rating Scale	18
Double Blind	.720	Outpatient Pain Patients	20	Physiological Measures	19

Double Blind	.604	Outpatient Pain Patients	30	Physiological Measures	20
Double Blind	.563	Psychiatric Prisoners	28	Clinical Rating Scale	22
Double Blind	.625	Substance Abuse	20	Self Rating Scales	24
Double Blind	1.099	Psychiatric Inpatients	11	Self Rating Scale	26
Double Blind	.233	Psychiatric Inpatients	60	Self Rating Scales	37
Double Blind	.693	Substance Abuse	21	Self Rating Scale	40
Double Blind	.775	Psychiatric Inpatients	24	Self Rating Scale	47
Double Blind	.618	Psychiatric Inpatients	20	Self Rating Scale	48
Double Blind	.405	Psychiatric Outpatients	80	Clinical Rating Scales	49
Double Blind	.365	Substance Abuse	60	Self Rating Scales	50
Double Blind	.693	Closed Head Injured	21	Self Rating Scale	64
Double Blind	.549	Normal Volunteers	30	Physiological Measures	68
Double Blind	.567	Prison Sex Offenders	105	Self Rating Scale, Physiological Measures	73
Double Blind	.618	Substance Abuse	24	Self Rating Scale	75
Double Blind	.633	Dental Patients	33	Self and Clinician Rating Scales	77
Double Blind	.811	Psychiatric Outpatients	22	Clinical Rating	43
Totals	12.847		744		
Average	.612				
Effect Size^c	r = .55				
Single Blind	.497	Substance Abuse	72	Clinical Rating Scales	61
Totals	.497		72		
Average	.497				
Effect Size	r = .46				
Crossover	.321	Psychiatric Inpatients	23	Clinician's Rating	10
Crossover	.080	Insomnia, Anxiety	28	Clinician's Rating	13
Crossover	.365	Outpatient	17	Clinician's Rating	33

		Psychiatry			
Crossover	1.757	Outpatient Psychiatry	10	Self, Clinician's Ratings	72
Totals	2.523		78		
Average	.631				
Effect Size	r =.56				
Open Clinical	.563	Psychiatric Outpatients	25	Clinician's Rating	11
Open Clinical	.523	Psychiatric Outpatients	12	Clinician's Rating, Physiological Measure	31
Open Clinical	.973	Psychiatric Inpatients	20	Clinician's Rating	29
Open Clinical	.621	Graduate Students	54	Self Rating Scales	30
Open Clinical	.640	Psychiatric Outpatients	182	Physiological Measures	35
Open Clinical	1.344	Substance Abuse	32	Self Rating Scale, Physiological Measure	36
Open Clinical	.973	Substance Abuse	186	Clinician's Rating	38
Open Clinical	.510	Psychiatric Outpatients	9	Clinician's Rating	44
Open Clinical	.604	Psychiatric Outpatients	12	Clinician's Rating	45
Open Clinical	1.039	Psychiatric Outpatients	23	Self Rating Scales	57
Open Clinical	.436	Phobic Outpatients	31	Self Rating Scale	63
Open Clinical	1.099	Prison, Sex Offenders	15	Self Rating Scale	74
Totals	9.325		601		
Average	.777				
Effect Size	r =.65				
SUMMARY, ALL ANXIETY STUDIES REPORTED ABOVE					
Grand Total	25.192		1,495		
Average	.663				
Total Effect Size	r =.58				

^a r correlation scores, representing percent improvement, are obtained mathematically from the study outcomes presented by the authors.

^b Most of the rating scales, both by the patients and the clinicians were of published reliability and validity.

^c Effect size is a statistician's basic estimate of the overall percentage improvement by the patients as a result of the treatment

Discussion of Anxiety Studies

There were more psychiatric inpatients and outpatients in this group of studies than in most of the others, though at one time patients in addiction treatment centers were regarded as psychiatric patients (See Abstinence Syndrome below.) The presenting syndrome or type of patient is given in column three of the table. In all of the studies, however, anxiety was a major diagnosis within the presenting syndrome or group.

Cognitive Function

Thirteen studies, in which a total of 648 patients with various types of cognitive dysfunction were treated with CES, were combined statistically in order to get a more confident look at the effectiveness of CES for treating this condition. The result of the analysis showed that the overall effectiveness of CES was 44% improvement. When the 7 studies of patients with substance abuse and the 3 studies of fibromyalgia patients were analyzed separately it was found that the substance abuse patients averaged a 60% improvement, while the fibromyalgia patients gained a modest but significant 17%.

Table 4. Studies of Cognitive Function Completed Over the Past 31 Years

Study Design	Zr Score ^a	Presenting Group	No. Subjects	Measure Used ^b	Reference
Double Blind	.1020	Substance Abuse	60	Profile Of Mood States	51
Double Blind	.829	Substance Abuse	60	Psychological Tests	50
Double Blind	.151	Fibromyalgia	60	Profile of Mood States	28
Totals	2.000		180		
Average	.667				
Effect Size^c	r = .58				
Single Blind	.604	Substance Abuse	72	Profile of Mood States	61
Single Blind	1.293	Substance Abuse	227	Psychological Tests	60
Single Blind	.388	Substance Abuse	24	Profile of Mood States	75
Single Blind	.234	Substance Abuse	100	Psychological Test	56
Totals	2.519		423		
Average	.630				
Effect Size	r =.56				
Open Clinical	.172	Graduate Students	54	Profile of Mood States	30

Open Clinical	.412	Post Traumatic Syndrome	2	Neuropsychiatric Texts	6
Open Clinical	.497	Substance Abuse	15	EEG	4
Open Clinical	.203	ADHD	23	Psychological Tests	57
Open Clinical	.182	Fibromyalgia	20	Profile of Mood States	70
Open Clinical	.182	Fibromyalgia	60	Profile of Mood States	71
Totals	1.648		299		
Average	.275				
Effect Size	r =.27				
SUMMARY, ALL COGNITION STUDIES REPORTED ABOVE					
Grand Total	6.167		648		
Average	.474				
Total Effect Size	r =.44				
SUMMARY OF SUBSTANCE ABUSE PATIENTS ONLY					
Totals	4.865		558		
Average	.695				
Effect Size	.60				
SUMMARY OF FIBROMYALGIA PATIENTS ONLY					
Totals	.515		140		
Average	.172				
Effect Size	.17				

^a Since percent improvement scores can not legally be averaged, they are converted into Zr scores, averaged, and then converted back to percent improvement (effect size.)

^b The Profile of Mood States is of published reliability and validity, as were each of the psychological tests used in the above studies.

^c Effect size, here, is a statistician's basic estimate of the overall percentage improvement by the patients as a result of the treatment

Discussion of Cognitive Function Studies

In most of the studies, cognitive confusion was but one symptom within a larger syndrome. While all presented symptoms of cognitive confusion of some type, it is obvious from the above secondary analysis, that the cognitive dysfunction among the substance abuse patients was very likely of a different etiology than that of the fibromyalgia patients, who may have been experiencing cognitive distraction due to the stress of the chronic pain of their condition.

Drug Abstinence Syndrome

Fifteen studies were analyzed, in which a total of 535 patients were treated for the drug abstinence syndrome with CES. The result of the analysis showed that the overall effectiveness of CES was 60% improvement.

Researchers earlier received a strong impetus to study CES in substance abuse patients when in the 1970s it was found that the abstinence syndrome, including such features as depression, anxiety and insomnia, was seen to come under control very quickly with CES. Serendipitously it was also discovered that what had up until the 1970s been termed “permanent brain damage” in these patients responded to three weeks of CES treatment by bringing these patients back within their normal functioning range (see Table 4, above)

Table 5. Studies of the Drug Abstinence Syndrome with CES

Study Design	Zr Score^a	Presenting Group	No. Subjects	Measure Used^b	Reference
Double Blind	.987	Poly Substance Withdrawal	18	Clinical Rating Scales	2
Double Blind	.397	Cocaine Withdrawal	17	Treatment Responses	5
Double Blind	1.029	Methadone Withdrawal	28	Treatment Records	16
Double Blind	.415	Alcohol Withdrawal	20	Self and Clinical Rating Scales	24
Double Blind	.403	Alcohol Withdrawal	20	Self and Clinical Rating Scales	31
Double Blind	.780	Poly Substance Withdrawal	49	Psychological Tests	51
Double Blind	.671	Poly Substance Withdrawal	60	Self Rating Scales	50
Totals	4.682		212		
Average	.669				
Effect Size^c	r = .58				
Single Blind	.360	Alcohol Withdrawal	85	Psychological Tests	56
Single Blind	.772	Alcohol Withdrawal	47	Self Rating Scales	61
Single Blind	.725	Alcohol Withdrawal	47	Self Rating Scales	60
Single Blind	.737	Alcohol Withdrawal	24	Self Rating Scales	75
Totals	2.594		203		
Average	.649				
Effect Size	r =.57				
Open	.678	Alcohol	53	Physiological Measure	59

Clinical		Withdrawal			
Open Clinical	.775	Smoking Cessation	20	Reduced Smoking	3
Open Clinical	.549	Poly Substance Withdrawal	15	EEG	4
Open Clinical	1.065	Marijuana Withdrawal	32	Self Rating Scales, Physiological Measure	36
Totals	3.067		120		
Average	.767				
Effect Size	r =.65				
SUMMARY, ALL ADDICTION STUDIES REPORTED ABOVE					
Grand Total	10.343		535		
Average	.690				
Total Effect Size	r =.60				

^a Most studies utilized several (up to 7) improvement measures. The average improvement on all measures reported for each study.

^b The Self Rating and Clinical Rating Scales used in the studies all have published reliability and validity measures.

^c Effect size, here, is a statistician's basic estimate of the overall percentage improvement by the patients as a result of the treatment

Discussion of Drug Abstinence Syndrome

The variety of substances of abuse involved in the above studies were quite varied, and included alcohol, heroin, cocaine, marijuana, and nicotine, among possibly others hidden within the poly substance groups. The measures used in evaluating the response to treatment were also greatly varied. Some involved published clinician's ratings scales, other utilized published patient's self-rating scales, while others used psychological tests of various kinds, while yet others combined these along with physiological measures, such as EEG or EMG recordings. While in one study a clinician's rating of treatment response was among the lower measures obtained, in another study the analysis of patient records, both during and following treatment, was among the highest. Also among the strongest responders to the CES treatment were methadone and marijuana patients.

Two of the studies compared the treated and control patients on AMA rates in which the patients left the program against medical advice, and on recidivism rate which measures the number of times a patient returns for additional treatment (not shown here). In both cases, they found that both the AMA and recidivism rates were reduced by one-half or more in the CES treated patients.^{5,51}

Explanation of Study Designs

In the **open clinical** study, the patients know they are being actively treated, the clinicians know who is being treated, and the statistician who summarizes the study data also knows, since there is only one group of patients.

In the **single blind** study, the patients do not know which are getting treated and which are getting sham treatment, but the clinician providing the treatment knows which are the treated patients. In the single blind study, the clinician doing the post study evaluation of the patients is often blinded to treatment conditions when he completes his evaluation. The statistician is usually blinded also, so that he is given two sets of scores to compare, and doesn't know which of them received the treatment. This study design was used earlier on before treatment-blinding devices came on stream. In such studies, the treatment was administered sub sensation threshold, in which the clinician turned up the current intensity until the patient just felt it, then turned it back down until the patient said he could no longer feel the stimulation. At that point, the clinician either left the current at that level or turned the unit off (down to, but not including the final click). Because the patients and the statistician are both blind to the study conditions, some authors have unwittingly published this design as a double blind experiment. But that term is generally reserved for the true double blind experimental design as described next.

The **double blind** study, the gold standard of science, is usually confined to studies in which neither the patient nor the clinician knows who is being studied. Those designs became available when a double blinding box could be inserted between the patient and the CES device. The double blinding box often had three, four or more settings in addition to a "0" setting in which current flowed freely between the CES unit and the patient. Among the other settings available, some passed current to the patient and some blocked it entirely. The clinician would begin the double blind treatment session by setting all double blinding boxes to the "0" position, would connect the patient to the CES electrodes, turn the current up slowly until the patient signaled he could just feel it, then reduce the stimulus level until the patient signaled that he could no longer feel it. At that point, the clinician set the double blinding box to one of the other settings available and left the patient on the device for 30 minutes to an hour, with neither the patient nor the clinician now knowing who was and who was not being treated.

In a good double blind experimental design, such as was the case in the majority of those reported in the tables, the persons who were responsible for measuring or rating patient improvement were also blind as to who was treated, as was the statistician who was given anonymous groups of data to analyze. Note that, in effect, that makes such studies quadruple blind, but that term is not used in science.

In the **crossover design**, half the patients get treated the first week or two of the study, while the other half receive sham treatment. In the second half of the study, the formerly treated patients now receive sham treatment while the formerly sham treated patients now receive treatment. If the crossover does not involve a sham treatment condition, then the crossover study is treated as an open clinical trial where all patients and staff know who is being treated at each cross of the study. That design is often referred to as a study with "wait in line" controls, in that the patients waiting to begin treatment are tested before and at the end of the waiting period

before going into treatment. That is thought to control for environmental factors such as unusual stressors on the 10 O'clock news, any local dramatic weather changes, and so forth.

We learned early on in CES work to stay clear of the cross over design in CES studies after we discovered that the improvement begun by a week or so of CES treatment continues after treatment is stopped. That is, the patients continue to get better as time goes on following treatment. One can imagine what that does to the statistical analysis when at the end of the study, both groups have improved significantly, but the patients treated first are no longer behaving as good controls should, but are getting even better than the final treatment group is showing. Many otherwise good studies were lost early on due to that effect, and one can see in tables 1, 2 and 3, that the crossover patients did the least well from CES treatment than any other groups when the statistical analysis was completed. In the tables above, where available, we included only the scores obtained in those studies following actual treatment, but before the crossover phase of each study.

Table 6 is a combined summary of all the studies of each given study design. This is not at all a typical type of meta-analysis but is designed to compare the effectiveness of the various designs in ferreting out the treatment effectiveness of CES, no matter what the conditions measured within each type of design.

Table 6. Summary by Study Design

Study Design	No. Studies	No. Subjects	Mean Improvement	Mean Range of Improvement
Double Blind	31	1,076	56%	23% - 81%
Single Blind	8	519	62%	29% - 93%
Open Clinical	22	1,162	56%	27% - 83%
Crossover	6	153	57%	8% - 95%
Totals/Ave.	67	2,910	58%	22% - 90%

From the above table it can be seen that the average (mean) improvement was very similar in all of the study designs, and no significant differences were found among them by statistical analysis. Looking at the variability of improvement scores found in the various studies, however, the extreme variability of the results in the crossover studies can be seen in that among those 6 studies can be found both the lowest and the highest scores. Also, to reiterate, only the score following the first treatment periods for each group in the crossover were included when possible, otherwise the combined totals as given in the published reports are presented.

It is perhaps of interest to persons attempting to evaluate the effectiveness of CES that the majority of the studies were in the highly prized double blind format. Patients in those studies fared equally as well as those in the open clinical trials, which may indicate that the highly touted, yet much more expensive double blind research format is not that terribly necessary in order to discover the merits of CES treatment.

As is apparent when one looks at Table 7, a serendipitous discovery from this unusual approach to meta-analysis is the finding that no matter which symptom is studied, if it is an

emotionally negative symptom associated with stress of one kind or another, CES can be expected to yield a significant improvement of at least 22% on average, and as high as 81% on average. No known medical treatment has been found to have that effectiveness, and certainly not without significant negative side effects (also see the section on adaptogens in the Mechanism of Action section below).

Table 7. Double Blind Studies

Study Design	Percent Improvement	Presenting Group	No. Subjects	Measure Used	Reference
Double Blind	37%	Insomnia	27	EEG, Clinician's Rating	21
Double Blind	64%	Insomnia	30	Self Rating Scale	26
Double Blind	70%	Insomnia	60	Self Rating Scale	27
Double Blind	53%	Insomnia	18	Clinician's Rating	39
Double Blind	42%	Insomnia	21	Self Rating Scale	41
Double Blind	81%	Insomnia	22	Clinician's Rating	44
Double Blind	79%	Insomnia	10	EEG, Self Rating Scale	77
Double Blind	78%	Substance Abuse	29	Clinical Rating Scales	2
Double Blind	41%	Substance Abuse	20	Self Rating Scale	24
Double Blind	46%	Closed Head Injured	21	Self Rating Scale	64
Double Blind	23%	Fibromyalgia	60	Self Rating Scale	28
Double Blind	58%	Psychiatric Outpatients	18	Clinician's Rating	43
Double Blind	72%	Insomnia, Anxiety	17	Clinician's Rating	33
Double Blind	39%	Psychiatric Outpatients	32	Self Rating Scale	14
Double Blind	60%	Substance Abuse	28	Self Rating Scale	16
Double Blind	50%	Psychiatric Outpatients	28	Clinical Rating Scale	18
Double Blind	62%	Outpatient Pain Patients	20	Physiological Measures	19
Double Blind	54%	Pain Patients	30	Physiological Measures	20
Double	51%	Psychiatric	28	Clinical Rating	22

Blind		Prison Inmates		Scale	
Double Blind	23%	Psychiatric Inpatients	60	Self Rating Scales	37
Double Blind	60%	Substance Abuse	21	Self Rating Scale	40
Double Blind	65%	Psychiatric Inpatients	24	Self Rating Scale	47
Double Blind	55%	Psychiatric Inpatients	20	Self Rating Scale	48
Double Blind	38%	Psychiatric Outpatients	80	Clinical Rating Scales	49
Double Blind	55%	Substance Abuse	60	Self Rating Scales	50
Double Blind	50%	Normal Volunteers	30	Physiological Measures	68
Double Blind	51%	Paroled Sex Offenders	105	Self Rating Scale, Physiological Measures	73
Double Blind	55%	Substance Abuse	60	Self Rating Scale	75
Double Blind	72%	Substance Abuse	60	Self Rating Scale	51
Double Blind	38%	Cocaine Withdrawal	17	Treatment Responses	5
Double Blind	38%	Substance Abuse	20	Self and Clinical Rating Scales	31
Number Studies	Number Subjects	Average Score	Zr	Mean Improvement	Range of Improvement
31	1,076	.625		56%	23% - 91%

Treatment Stimulus Level

The question often arises how much intensity of stimulation is needed to get changes in the symptoms. Or conversely, it is sometimes assumed that if a small amount of stimulation gets results, a more intense stimulation will get better results, and perhaps faster. Looking at table 6 below, it should be noted that only in the open clinical studies were patients stimulated above their level of sensation. In all the blinded studies (67%), patients received stimulation that they could not feel. The resulting stimulation very likely was not enough to light a flashlight battery, and yet they did equally as well or better than the 33% of patients who were stimulated above their sensation threshold.

To have a specific look at this, in the 1970s I did an unpublished study in which 100 patients each received stimulation pre set 0.19 mAmp, 1.0 mAmp or at a stimulation level set by the patients to their comfort level. I tested them for anxiety, depression and cognitive function. They were all stimulated daily for 45 minutes, Monday through Friday for three weeks. To my

surprise patients stimulated at the very low and very high levels did significantly less well than did the patients who were stimulated at their comfort level, even though on retrospective analysis that level varied considerably among them. It is very likely that controlled studies in which the treatment intensity is set just below the patient's sensation threshold is closer to what would otherwise be found to be their comfort level than would a very high or very low setting otherwise.

Safety of Cranial Electrotherapy Stimulation

In 1974 the FDA funded a study of CES by the National Research Council's Division of Medical Sciences (FDA Contract 70-22, Task Order No. 20 (NTIS PB 241305 pp. 1-54), in which the National Research Council reported back to the FDA that there could be no possibility of harm to a human subject from this level of electrical stimulation (up to 4 milli-amp). From that point on, the FDA's official position has been that CES is not a significant risk device.

It is interesting to note that not one problem from significantly negative side effects was reported in any of the CES studies analyzed here, involving 2,910 patients. None of the patients has raided the fridge during the night and gained weight. None has complained of grogginess or suicidal ideation the next day. None has complained of headaches or a foggy feeling following treatment. When asked, CES patients have reported instead feeling more rested, more alert, and less tired following treatment. Perhaps better still, if they went into the studies caught up in stress related cognitive confusion, or even a chemically engendered cognitive dysfunction, if the study lasted at least 2 or 3 weeks, their cognitive function returned to their normal functional level.

Possible Mechanisms of Action

When a new medical treatment comes on line, there is pressure to explain how it works. While CES has been in medical use for the past 53 years (it came into being as "electrosleep" in Europe in 1953), there has been no definitive, settled explanation of its mechanism of action. To explain its mechanism satisfactorily, a treatment has to be understood within one of the accepted explanatory concepts currently in vogue in medical science. Some of the theories that are more or less active at present are as follows:

The Nervous System.

The major present concept is that the body functions via a more or less hard wired nervous system. In this theoretical system, the body is neuronally wired to receive incoming stimuli via its afferent neurons, send them to the central nervous system, which then sends out response stimuli via its efferent neurons. One touches a finger accidentally to a hot surface and the finger is immediately jerked away from the hot stove, for example.

Since the neurons don't ordinarily physically touch, the neural wiring functions via synaptic endings on the neurons in which the pre synaptic membrane discharges neurochemicals from stored vesicles into the synapse between the neurons and these stimulate receptors on the post synaptic membrane (the receiving membrane of the neuron next in line to fire) and that neuron fires the next neuron or the sensitive membrane on a muscle receptor, and so forth.

To work as efficiently as it was designed to work, all the neurons must be intact, and all the neurochemicals that are involved in the neurological firing patterns have to be in balance with all the others. If one neurochemical is out of balance, either it over fires or under fires the system for which it is responsible, in which case physical or emotional symptoms of one kind or another arise. For example, if there is not enough dopamine, Parkinson like symptoms develop. If there is not enough serotonin, depression results, etc.

If CES is to be effective within this system, then it must be shown that CES acts to bring back into balance neurotransmitters that are out of balance with their associates. Pozos and his associates completed a series of interesting experiments with canine subjects that looked at this possibility. They examined the adrenergic-cholinergic balance in the brain.

His research group theorized that if CES actually stimulated neurons to fire, as CES salesmen were claiming, he could give some of the dogs reserpine plus CES stimulation and the reserpine would block the reuptake of any newly CES generated dopamine into the presynaptic vesicles. That would reduce the future amount of dopamine available in the presynaptic vesicles to fire the postsynaptic neurons on the adrenergic side, and the cholinergic system would gain the upper hand. He did that, and discovered that in doing so he had thrown the adrenergic system out of balance with the cholinergic system and the dogs began to show Parkinson like symptoms.

Pozos was not directly measuring dopamine in the dog's brain, however, so he thought he would do some more experiments to make sure he was in the right ballpark. He decided to block the uptake of acetylcholine on the other side of the adrenergic-cholinergic equation. It was the acetylcholine uptake that was firing that side of the equation and instigating the Parkinson like symptoms. To do this, he gave his CES stimulated dogs, which were still in Parkinson like tremors, some atropine to block the acetylcholine uptake. The tremors ceased.

He decided that since he was not, after all, measuring either dopamine or acetylcholine, he would check the system further by taking another group of CES treated, tremoring dogs, remove the atropine from the cholinergic side and add physostigmine instead. Physostigmine would actually stimulate the cholinergic nerves to fire more rapidly than normal. He did this, and the dogs showed the most pronounced tremors ever.

Pozos was reasonably certain that he was manipulating the correct system and had shown that CES could effectively stimulate an increase in the manufacture of dopamine. But wait. If that were the case, then CES should be able to put the terribly out of balance system in the dog's brain back to normal when all provoking chemicals were removed. To that end, he removed the drugs from all the dogs' systems, gave all of them regular food and water for the following week. A third received L-Dopa, and another third of them received CES stimulation.

The non treated animals returned to normal, non-Parkinson like states within three to five days. The CES treated animals, however, returned to normal, non-Parkinson like states within 3 to 7 hours, as did the third of the animals receiving L-Dopa.⁴¹

A similar study was run in human narcotics addicts in whom the depletion of endorphin by the narcotics had presumably thrown off the balance between the endorphin and norepinephrine systems in the locus ceruleus of the brain. Once the narcotics, which had been mimicking endorphin and thereby down-regulated that system, were removed, norepinephrine got the upper hand and physiological withdrawal symptoms began. The researchers knew that they could block the post synaptic receptors to norepinephrine with alpha methyl dopa and thereby stop the withdraw symptoms, but thought they would also try CES stimulation on half of the patients to see if CES could stimulate increased endorphin production, and thereby rebalance the system.

They found that both CES and the drug treatments worked equally well, so that the physicians who monitored the double blind research could never tell which patients were treated with alpha methyl dopa and which were receiving CES. Until after the study, that is, when the drug patients went into rebound depression (their norepinephrine production had down-regulated due to the blocked uptake by alpha methyl dopa) and the CES treated patients did not.¹⁵

(In a somewhat interesting reversal of research form, 7 and 8 years following Gold's report of using CES for withdrawal in human subjects, Dougherty and his associates at the University of Texas studied CES in the withdrawal of rats. In one they found that CES attenuated the severity of naloxone-precipitated morphine withdrawal,⁷ and in the other they found a time-dependent effect of CES treatment in reducing the severity of motor hyperactivity associated with abrupt morphine withdrawal.⁸)

If Pozos found evidence that CES stimulates dopamine production in canine subjects, and Gold found evidence that CES stimulates endorphin production in his human subjects, we may have an insight into the mechanism of the very positive response of numerous fibromyalgia patients to CES.

Wood assumed that chronic stress was an underlying mechanism of fibromyalgia. He noted that the brain's response to acute stress is an analgesic response mediated by activation of mesolimbic dopamine neurons arising from the ventral tegmental area and projecting to the nucleus accumbens. Chronic stress results in a down-regulation of dopamine output to the nucleus accumbens and a persistent hyperalgesia. He theorizes that this stress-related dysfunction of mesolimbic dopaminergic activity may be the basis for other fibromyalgia associated symptoms as well (see tables 2 and 4 above), and predicts that treatment strategies aimed at boosting dopaminergic function would have superior efficacy in patients suffering from fibromyalgia.⁷⁸

Another study actually counted the number of presynaptic vesicles in presynaptic membranes of squirrel monkeys before, after several minutes of CES stimulation, and for a time following the cessation of stimulation. Their findings convinced them that CES acted essentially to stimulate the vesicles to empty their contents, thus reducing them in number. But CES then acted to dramatically increase the number of new vesicles formed in the presynaptic membrane as stimulation continued. Once the stimulation had ceased, the number of vesicles tended to gradually return to their pre stimulus levels over an hour or so.⁵⁵

Acupuncture Theories

Following President Nixon's visit to China, acupuncture treatment came into a sort of vogue in the U.S. and still plays a role in some medical circles. The theory behind acupuncture is that the body works on an energy homeostasis and at times, and for any number of reasons that system can be thrown out of balance. It was for that reason that earlier on CES was thought by many to perhaps have its effects by stimulating this system to increased energy balance when insufficient energy was present in the system to keep the body working normally.

Energy is known to flow through the collagen connective tissues of the body, and some areas of the body are more sensitive to energy incoming to that system than others. These sensitive areas are known as acupuncture points, and CES may well supply energy to that system, though not necessarily by stimulating those points directly.

CES electrodes are placed at various places on the head so that the stimulating current is allowed to pass through the head. CES current has been shown to spread around the head and scalp while also going through the entire brain, though canalizing along the limbic, or "emotion" brain.²³

As anyone knows who has placed CES electrodes on the mastoid processes behind the ears and turned the current up, one tends to get an involuntary grin when the current spreads to the facial muscles, and similarly, there can be light flashes keeping time with the CES pulse as the energy passes through the ocular apparatus in the eyes. For this reason, it is very likely that any acupuncture points on or about the head would receive sufficient stimulation, wherever they are located, to respond to CES stimulation. For example, in some therapeutic strategies, several of those points on the face are said to be dramatically activated by merely softly tapping on them with the finger tips.¹² Not enough is known about the acupuncture system by the present author to speculate further on just how CES may effect bodily changes via the acupuncture route, but it may be shown to do so in the future.

There are other energy flow systems that are active throughout the body, such as **the vascular system as an electrical transmission system**. Nordenstrom has shown that the vascular system acts as a biologically closed electric circuit in which energy flows readily, pulling and pushing electrically charged blood components so as to keep the body in functional homeostasis. That system is active both in the arteries and veins, whose walls act as insulation, and in the vascular-interstitial spaces.³⁴ To date, no known studies of how CES effects or interacts with this system exists, though theoretically it should.

Nor are any CES studies known to exist regarding **the perineural electrical system** which Becker has shown also acts throughout the body as an electrical system acting peripherally to, but separately from the nervous system, though again, theoretically it should.¹

EEG studies.

Numerous EEG studies, MRI studies and the like have been done with CES stimulated subjects, several of which are reported in the sleep studies and addiction studies sections. Several

of those studies are ongoing, and new ones are being planned as this is being written. In looking through the various studies that have been done over the years, it can be stated that CES is invariably found to work changes in the brain's neural firing pattern. While the effects of those changes can be difficult to decipher, none has been thought to have a negative impact of any sort on the patients studied. For example, no seizures have ever been detected accompanying CES treatment, even among known seizure patients.⁶⁴

Neurohormonal Studies

Several studies have been completed, showing the ability of CES to effect a return to more normal conditions hormones that are out of balance in depressed patients,⁵⁴ and those such as DHEA, testosterone, estrogen, and IGF-1 in older subjects in whom those hormone levels were low.⁶²

Synchronicity Theory

The science of spontaneous order, as the synchronicity field is now being called, appears to be rising fast as a new way of describing medically related phenomena.⁶⁷ In this theory, every part of the body is seen to be functionally synchronized with every other part, and each organ is specifically functionally synchronized within itself. The entire body is also synchronized with the external environment. Illness results when any part of the body becomes desynchronized on any of the three synchronicity levels.

Synchronicity can easily be seen on TV nature channels when thousands of schooling fish dart first in one direction and then another, quite spontaneously and never hitting another nearby fish when attacked by a feeding shark. Large flocks of migrating birds can be seen wheeling at high speeds overhead in first one direction and then another without ever colliding even though there may be hundreds of birds flying in the close formation.

Similarly, in the human body, all liver cells have to be functioning in sync for the liver to get its work done. The same is true of the pancreas, the heart muscles, the adrenal glands, and so forth. Further, all the various organs, even while entraining their separate rhythms, have to work in synchrony with all the others if the body is to function properly.⁸⁰

The master clock that regulates circadian rhythm in the rat has been found to be a monosynaptic neural pathway from the retina in the eye to the two small suprachiasmatic nuclei in front of the hypothalamus. This pathway, while originating in the eye, has nothing to do with vision, nor does it fire into any visual centers of the brain. It appears to be dedicated specifically to the purpose of regulating the synchronicity between the rat and its external environment.

Researchers are still looking for the master clock within each of our body's organs and in the body as a whole. The future may well show that the incoming, timed rhythmic pulsations of electric energy involved in CES treatment acts in some way to reset a desynchronized body back into normal synchronicity and thereby produces a more healthful functioning.

CES as an Adaptogen

Stress is thought by many to underlie as much as 90% of the emotional and physical disorder that plagues modern society. There are numerous reasons given for this, as can readily be found in much of the medical literature. Earlier research by Hans Selye⁵³ indicated that not only is stress a problem that interferes with health in many persons, but that when an individual experiences chronic stress the problem is made worse.

Selye noted that the normal, inbred human (and other animal) reaction to threat is to fight or flee. Underlying this fight or flight response is a host of bodily changes that shifts the blood supply and body's nutrients to the muscles and away from the interior of the body. The endocrine glands secrete emergency supplies of hormones that help narrowly orient the person to the source of the threat and prepare him to utilize stored energy in the most efficient manner. The heart rate increases, the blood pressure increases, and the adrenal glands excrete large amounts of cortisol into the blood stream. The entire physiological mechanism is geared up to support the response to the emergency.

Once the emergency is over, the body then gradually returns to normal functioning. The blood returns to the intestines so food can be digested, the cortisol level returns to normal, the "hair standing on end" reaction subsides as elevated hair on the skin and scalp return to their normal position. The heart rate and blood pressure return to normal.

A major contribution to this field was Selye's finding that in the presence of chronic stress, a version of the fight or flight reaction remains in place as the body habituates to the chronic presence of implied threat. In that case, the body never returns to its normal, pre stress level, and a host of physiological and emotional symptoms may develop over time due to the chronic elevated adjustment of the physiology of the organism, remaining as it does in the emergency response mode.

Everyone is familiar with sources of chronic stress. Watching the morning or evening news broadcasts on TV in which murders, rapes, arson, bombings, and so forth are invariably enumerated to catch you up on every stressful event that has taken place while you slept or were at work. Driving to work in the impossible traffic that is an ever more frequent condition in America's modern cities is an awful stressor, and the threat to your well being is real. And you may do it five or six days a week.

Even shopping for food in modern supermarkets has become laden with stress as new information continues to come on stream about the horrors of various fats, of many harmful food additives and preservatives, of fruits and vegetables that may contain insecticide residues, or milk and meat that may contain added hormones that can cause cancer.

We worry constantly about the amount of TV we and our children are watching, and the vicarious killing, maiming and general destruction our children engage in with their hand held electronic game devices on a daily basis.

Chronic stress is like high blood pressure. We can have it but no longer feel it. We can know of its presence only by the pathology that it generates in our emotions or physical health. The problem becomes what to do about it.

Enter adaptogens. A Russian Scientist, Dr. Nicolai Lazarev,²⁵ was studying medicinal herbs that seemed to act almost like panaceas, in that some of them appeared to be an effective treatment for an embarrassingly wide assortment of disorders. In studying them further, he found that they apparently acted to increase the body's resistance to adverse influences not by any one specific action but by a wide range of physical, chemical and biochemical factors that had a normalizing effect on the body. Their action appeared to somehow alleviate stress, and in the process improve all kinds of conditions that had been generated by that stress.

He termed this reaction the adaptogenic response and referred to the herbs as adaptogens. Among other responses to this reduction in stress were improved sleep, reduction in fatigue, the alleviation of depression, and improved cognitive performance. The mechanism of one of the first adaptogens that he studied, *rhodiola rosea*, was found to be apparently related to its ability to influence levels and activity of biogenic monoamines such as serotonin, dopamine, and norepinephrine, among others in the brain.

Before long, other, non herbal remedies were also found to behave like adaptogens such as COBAT,¹⁷ a combination of two amino acids, taurine and beta-alanine that were originally intended to improve the immune function and thereby act as an anti-cancer treatment. In researching its mechanism of action, it was discovered that it apparently acted to regulate the group of chemicals called cytokines. Cytokines are produced by some of the white cells making up the immune system, and often act as messengers between the cells, stimulating them to make more cytokines when the body is threatened. The problem is that these threats and perhaps other stressors can trigger this system to over react, triggering a so-called cytokine cascade. That leads to the cytokine syndrome in which fatigue, fever, cognitive confusion, muscle pain and depression are among its better known symptoms. COBAT may have its adaptogenic effect by normalizing the cytokine response and thereby avoiding or reversing the cytokine syndrome in many patients.

Viewed in this light, we can now see CES as behaving very much like an adaptogen. The mechanism of action is similar in some ways to that postulated for *rhodiola rosea*.⁷⁹ It acts to normalize the biogenic monoamines in the brain and bring them back into balance.^{15,41,54}

And while no work has been done on CES in relation to the cytokines as far as is known, CES is often thought to be one of the few treatments, if not the only one presently available, that can reset the chronic stress response back to an earlier, normal stress reaction. For example, there are two types of anxiety studied in some of the CES studies given in Table 3 above.^{47,50,61} One is so-called state anxiety, which is a reaction to stressors in the environment to which one is presently reacting. The other is trait anxiety which one carries within him at all times, even when lying on a sunny beach while on vacation or picnicking in the woods with his family. Trait anxiety is thought to reflect the chronic stress syndrome as outlined by Selye. In studies of anxiety, both state and trait anxiety come back to within normal levels by three weeks of daily

CES treatment. No other treatment has been shown to have this kind of effectiveness in so short a time frame, and at less cost.

Earlier, when I listened to CES marketers talk among themselves, the great difficulty in presenting CES in such a way that it was not seen as a panacea was often discussed. The panacea claim, or snake oil cure was well known by them to be a kiss of death when marketing medical treatments. Their problem can be seen in a new light if one conceptualizes CES as an adaptogen. In the chronic stress syndrome, the biogenic monoamines have developed a new, stress response type of balance to each other. Physical and emotional symptoms result. If CES acts to bring these relationships back into a more normal pattern, then symptoms of fatigue, pain, sleeplessness, cognitive confusion, and so forth would be alleviated as the body returns towards normal.

In Table 7, as one looks at the various double blind studies that researched at least 5 different stress related syndromes, it can be seen that all of these symptoms do respond significantly to CES treatment. To the extent that CES acts as an adaptogen, it would explain why CES manufacturers have always had such difficulty in writing up the directions for use in their packaging. As we have noted, stress can underlie pain, it can underlie depression, it can underlie anxiety states, it can underlie fatigue, and so forth. But the amount of stress that produces each of the above may vary in different individuals over time, and produce one or more or all of those symptoms at any given time. And while most patients can be seen to respond to CES, they may well respond at different levels of treatment or rates of improvement. Unlike penicillin, the package insert for an adaptogen must, by the very nature of an adaptogen, remain rather vague and generalized.

To aid clinicians and help them get around this potential barrier to the use of CES, the directions given for the clinical use of CES, below, is geared to treat the majority of patients effectively, no matter what the stress level with which they begin.

Summary of Mechanisms

CES has been shown to travel throughout the brain, and in the process bring back to normal neurohormonal systems that have been deliberately thrown out of balance by researchers or by patients themselves, in the case of narcotics addicts. It has been shown to bring back to more youthful levels several hormones that are typically reduced in aging.

CES, then, is thought to act as an adaptogen to balance physiological systems that have become unbalanced by whatever means, but certainly by chronic stress. And if stress can underlie 90% of our emotional and physical symptoms, then CES as an adaptogen that effectively reduces stress should logically be expected to have a positive effect on the 90% of disorders caused by that stress.

An interesting corollary to that is the often obtained clinical finding that once a patient is back in balance – these are sometimes seen in the drug abstinence syndrome, for example – CES ceases to have an effect and the patient stops using it. For that reason it is known to not be

addicting or habit forming in any way. And in none of the studies to date has a significant negative side effect been reported.

The Clinical Protocol for the Use of CES.

Executive Summary:

Plan to treat patients from 45 minutes to one hour daily for three weeks. Set the unit to pulse 100 pps when that is available, and stop treatment if patients signal they have reached their maximum level of improvement before the three weeks are up.

Discussion

Clinicians new to CES understandably want to know how to apply CES effectively, should they wish to use it with their patients. Ideally the FDA would like all CES manufacturers to state in their instruction booklet how CES should best be used, but that is difficult to do based on the presently available research. Among the studies cited in this monograph, use instructions were never researched as such. But one can infer much by reading the study protocols and seeing the results of the several different treatment strategies.

Rosenthal tried perhaps more different treatment protocols than most other researchers. He began looking at “electrosleep” to see if anything at all happened to his inpatient and outpatient psychiatric samples. He gave an early group 30 minutes of stimulation every other day over a five day (three treatment) period. He obtained results that while very limited, suggested that something of importance might be going on, so a subsequent group received 30 minutes of stimulation for 5 days in a row, Monday through Friday. That group did better still. In his later studies, Rosenthal typically progressed to a protocol in which each patient received 10 days of at least 30 minutes stimulation. He reasonably felt that if a significant treatment effect was going to show up, it should show up by the end of 10 days of stimulation. His reports reflected that his surmise was in the right ballpark.

A few years later, when the CES manufacturer, Ray Gilmer visited a new research center with his Neurotone 101, the researchers found that his unit could stimulate at either 50 pps or 100 pps (pps is the same as Hz, the two terms often being used interchangeably). When they asked Gilmer which should be used, and what the treatment protocol should be, he encouraged them to use the 100 pps setting, and treat patients for 1 hour per day for three weeks. In a latter day scrutiny of the literature available up to that time, it seemed obvious that he was leading more by intuition than by published study results.

Nonetheless, the present reviewer began researching the unit in a busy inpatient facility in which therapies lasted approximately 50 minutes so the patients could be released from one and be on time at the beginning of the next. The facility was also minimally staffed on weekends, with no clinicians available for research treatments. Subsequently, over the next several years, CES was studied in that treatment center with an effective protocol of 45 minutes (it took 5 minutes to get the devices hooked up to the patients) per day, Monday through Friday for 3 weeks. That was more treatment than Rosenthal had used, so after several hundred patients were

run through the 3 week protocol, it was decided to check the next research group going through on the Profile of Mood States every day following treatment to see when the greatest treatment effect occurred. It was discovered that while the majority of patients came around nicely by the end of the 10th session, the remainder did not show maximum improvement until sometime between treatment number 10 and 14. For that reason all subsequent studies in that facility utilized the 15 treatment paradigm to be reasonably sure that response was obtained from as many patients as would respond. That protocol is still being widely followed at the time of this writing, some 34 years later, as one will see when reading some of the later referenced studies.

An important question clinicians have always had: Is CES addictive or habit forming. Since this author's original work was in a 600 bed addiction treatment hospital that question weighed heavily on our staff. Since we didn't know the answer earlier on to that question, the thought was often stated, "better they be addicted to CES than to the horror we are trying to get them off of." That being ultimately unsatisfactory, we finally designed a study to find out if CES is addicting. Following their treatment with CES in a California addiction treatment center, more than 100 patients were prescribed CES units for home use and told they could use them as they felt the need.

One year later they were called back to the treatment facility to see how many were abusing their CES device or showing signs of habituation or even addiction to them. None of them showed any sign of even moderate, ongoing use. In fact, most of them had used it for only a week or two during the year, and only when under unusual stress, according to their reports to the researchers. One of the patients said, "I don't even know where my CES device is, but you can bet I'll go straight to it if I get into some bad stress." The others nodded in agreement.

Another question is that of the possibility of CES provoking seizure activity in seizure prone patients. While there is at least one research protocol out which is designed to answer that question, I have seen no publication regarding the subject to date. We have inferential information regarding the subject, however. Studies involving migraine patients – a type of cerebral seizure activity according to some clinicians – have never induced migraines in persons who are migraine prone and are not having an episode when CES is applied.⁹

Since substance abusers can sometimes have seizures when undergoing abstinence, earlier on clinicians in the District of Columbia withdrawal facility chose to use CES on patients only when they had finished the acute withdrawal phase of their treatment. On the other hand, in the mid 1970s I was told in a phone call to Charity Hospital in New Orleans, that they routinely applied CES to patients who were undergoing acute withdrawal in order to prevent seizures, and that that was proving successful.

In the 1980s I visited an inpatient addiction treatment center in Florida in which CES was the treatment of choice for patients who were suffering delirium tremens. I watched on in amazement at the physical process required by the staff to get CES electrodes connected to such patients and keep them on them for the approximately 5 minutes required for the calming effect to kick in. They reported no seizures associated with this treatment approach.

As for age groups who respond to CES, the studies reported above involved children at the sub teen level to elders in their 70s and 80s. One study that specifically looked at the age response of their patients, whose age varied over a wide range, found that they could detect no age group correlation with response to treatment. All the patients did similarly well.⁵⁰

That same group, as have others, completed placebo controlled studies to see what is the placebo effect of CES treatment. They either found none,^{51 61} or a negative placebo effect when non treated patients reacted with apparent additional stress to the fact that this new medical treatment was useless in their treatment.²⁸ An earlier study deliberately looked for placebo response prone patients by separating their treatment group into patients who had tested as high suggestibility vs. low suggestibility on psychological tests. They theorized that more suggestible patients would show the strongest placebo effect from CES treatment. They found that neither of the two groups showed a CES treatment placebo effect.⁴⁷

Clinicians are sometimes faced with the challenge that I had earlier when confronted with a CES device that stimulated at either 50 pps or 100 pps. Other available units stimulate at frequencies as high as 5,000 pps and have shown successful treatment effects. The late Margaret Patterson's device stimulated at 4 or more different frequencies, and her clinical staff encouraged the use of different pps settings for different syndromes being treated. At this writing, that unit has not become available in the U.S., but clinicians should know that those theories are out there. Several studies have compared pulse rates that were available on CES devices and their findings suggested that when 100 pps is available on a unit it could rationally be used as a first choice.^{69, 68, 52}

In summary, while isolated studies have shown a response to CES from sometimes as little as 20 minutes of treatment,⁶⁵ to be on the safe side, most of a clinician's patients can be expected to have their greatest response within three weeks of treatment, 45 minutes a day. Receiving treatment past the point of their final treatment response will not harm them in any way and they will not habituate to the stimulation. Or as one teenaged cocaine patient said to me one day, "Dr. Smith, do I have to keep wearing this thing? It did really great things for me the first two weeks, but for the last five days nothing has happened." His treatment was terminated immediately, and that is perhaps a good suggestion for any clinician who is going to use CES outside of a research paradigm, in which case a specific stimulation protocol will need to be followed.

References

1. Becker, R.O., and G. Selden (1985) The Body Electric; Electromagnetism and the Foundation of Life. New York: Williami Morrow. pp 79-117.
2. Bianco, F. Jr. (1994) *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.
3. Boertien, A.H. (1967) The electrosleep apparatus as a device in an antismoking therapy. In: Wageneder, F.M. and St. Schuy (Eds) Electrotherapeutic Sleep and Electroanaesthesia. Amsterdam: Excerpta Medica Foundation. International Congress Series No.136 pp. 103-104.
4. Braverman, E., R.B. Smith, R. Smayda and K. Blum (1990) Modification of P300 amplitude and other electrophysiological parameters of drug abuse by cranial electrical stimulation. *Current Therapeutic Research*. 48(4):586-596.
5. Brovar, A. (1984) Cocaine detoxification with cranial electrotherapy stimulation (CES): a preliminary appraisal. *International Electromedicine Institute Newsletter*. 1(4):1-4.
6. Childs, A., and M.L. Crismon (1988) The use of cranial electrotherapy stimulation in post-traumatic amnesia: a report of two cases. *Brain Injury*. 2:243-247.
7. Dougherty PM, Dafny N (1989) Trans-cranial electrical stimulation attenuates the severity of naloxone-precipitated morphine withdrawal in rats. *Life Sciences*. 44(26):2051-6.
8. Dougherty PM, Dong WQ, Faillace LA, Dafny N (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.
9. England RR (1976) *Treatment of migraine headache utilizing cerebral electrostimulation*. Master of Science Thesis, North Texas State University, Denton, Texas.
10. Feighner, J.P., S.L. Brown, and J.E. Olivier (1973) Electrosleep therapy: A controlled double-blind study. *Journal of Nervous and Mental Disease*. 157(2):121-128.
11. Flemenbaum, A. (1974) Cerebral electrotherapy (electrosleep): an open clinical study with a six month follow-up. *Psychosomatics*. 15(1):20-24.
12. Flint, G.A. (2001) Emotional Freedom; Techniques for Dealing with Emotional and Physical Distress. Vernon, British Columbia: NeoSolterric Enterprises. pp 25-26.
13. Frankel, B.L., R. Buchbinder, and F., Snyder (1973) Ineffectiveness of electrosleep in chronic primary insomnia. *Archives of General Psychiatry*. 29:563-568.

14. Gibson, T.H. and D.E. O'Hair (1987) Cranial application of low level transcranial electrotherapy vs. relaxation instruction in anxious patients. *American Journal of Electromedicine*. 4(1):18-21.
15. Gold, M.S., A.L.C. Pottash, H. Sternbach, J. Barbaban, and W. Annitto (1982) *Anti-withdrawal Effects of Alpha Methyl Dopa and Cranial Electrotherapy*. Paper presented at The Society for Neuroscience. 12th Annual Meeting, October.
16. Gomez, E. and A.R. Mikhail (1978) Treatment of methadone withdrawal with cerebral electrotherapy (electrosleep). *British Journal of Psychiatry*. 134:111-113.
17. Health Sciences Institute (2005) *Who wouldn't want 52% more energy: What the "goldilocks effect" can do for fatigue and your immune System*. The HIS: Baltimore, pp 1,2.
18. Hearst, E.D., C.R. Cloninger, E.L. Crews, and R.J. Cadoret (1974) Electrosleep therapy: a double-blind trial. *Archives of General Psychiatry*. 30(4):463-466.
19. Heffernan, M. (1995) The effect of a single cranial electrotherapy stimulation on multiple stress measures. *The Townsend Letter for Doctors and Patients*. 147:60-64
20. Heffernan, M. (1996) comparative effects of microcurrent stimulation on EEG spectrum and correlation dimension. *Integrative Physiological and Behavioral Science*. 31(3):202-209.
21. Hozumi, S., H. Hori, M. Okawa, Y. Hishikawa, and K. Sato (1996). Favorable effect of transcranial electrostimulation on behavior disorders in elderly patients with dementia: a double-blind study. *International Journal of Neuroscience*. 88:1-10.
22. Jamelka, R. (1975) *Cerebral electrotherapy and anxiety reduction*. Master's Thesis, Stephen F. Austin State University.
23. 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.
24. Krupitsky, E.M., A.M. Burakov, G.F. Karandashova, J. Katsnelson, V.P. Lebedev, A.J. Grinenko, and J.S. Borodkin (1991). The administration of transcranial electric treatment for affective disturbances therapy in alcoholic patients. *Drug and Alcohol Dependence*. 27:1-6.
25. Lazarev, N (2006) *Adaptogen*. As reported in the Wikipedia Encyclopedia, <http://en.wikipedia.org/wiki/adaptogen>.
26. Levitt, E.A., N.M. James, and P. Flavell (1975) A clinical trial of electrosleep therapy with a psychiatric inpatient sample. *Australian and New Zealand Journal of Psychiatry*. 9(4):287-290

27. Lichtbroun, A.S., Mei-Ming Raicer, R.B. Smith (1999) *The use of Cranial Electrotherapy Stimulation in the Treatment of Fibromyalgia*. Presented at the 15th Annual International Symposium on Acupuncture and Electro-Therapeutics., Columbia University, New York City, October 21-24.
28. Lichtbroun, A.S., M.C. Raicer, and R.B. Smith (2001) The treatment of fibromyalgia with cranial electrotherapy stimulation. *Journal of Clinical Rheumatology*. 7(2):72-78.
29. Magora, F., A. Beller, M.I. Assael, and A. Askeknazi (1967) Some aspects of electrical sleep and its therapeutic value, in Wageneder, F.M. and St. Schuy (Eds.) Electrotherapeutic Sleep and Electroanaesthesia. (Amsterdam: Excerpta Medica Foundation) International Congress Serious Noi. 136. pp. 129-135.
30. Matteson, M.T., and J.M. Ivancevich (1986) An exploratory investigation of CES as an employee stress management technique. *Journal of Health and Human Resource Administration*. 9:93-109.
31. McKenzie, R.E., R.M. Costello, and D.C. Buck (1976) Electrosleep (electrical transcranial stimulation) in the treatment of anxiety, depression and sleep disturbance in chronic alcoholics. *Journal of Altered States of Consciousness*. 2(2):185-196.
32. McKenzie, R.E., S..H. Rosenthal, and J.S. Driessner, (1976) Some psycho-physiologic effects of electrical transcranial stimulation (electrosleep). In Wulfsohn, N.L. and Sances, A. (Eds) The Nervous System and Electric Currents. (New York: Plenum) pp 163-167.
33. Moore, J.A., C.S. Mellor, K.F. Standage, and H.A. Strong (1975) A double-blind study of electrosleep for anxiety and insomnia. *Biological Psychiatry*. 10(1):59-63.
34. Nordenstrom, B.E.W. (1983) Biologically Closed Electric Circuits; Clinical, Experimental and Theoretical Evidence for an Additional Circulatory System. Stockholm:Nordic Medical Publications. pp 269-317.
35. Overcash, S.J. (1999) A retrospective study to determine the efficacy of cranial electrotherapy stimulation (CES) on patients suffering from anxiety disorders. *American Journal of Electromedicine* 16(1):49-51.
36. Overcash, S.J. and A. Siebenthal (1989) The effects of cranial electrotherapy stimulation and multisensory cognitive therapy on the personality and anxiety levels of substance abuse patients. *American Journal of Electromedicine*. 6(2):105-111.

37. Passini, F.G., C.G. Watson, and J. Herder (1976) The effects of cerebral electric therapy (electrosleep) on anxiety, depression, and hostility in psychiatric patients. *Journal of Nervous and Mental Disease*. 163(4):263-266.
38. 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.
39. Patterson, M., N.V. Flood, and L. Patterson (1992) Neuroelectric therapy (NET) in addiction detoxification. *Subtle Energies* 3(3):1-22.
40. Philip, P. J. Demotes-Mainard, M. Bourgeois, and J.D. Vincent (1991) Efficiency of transcranial electrostimulation on anxiety and insomnia symptoms during a washout period in depressed patients; a double-blind study. *Biological Psychiatry* 29:451-456.
41. 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. Sjorberg, (Eds) Neuroelectric Research. Springfield, Charles Thomas. pp 221-225.
42. Rosenthal, R. (1991) Meta-analytic procedures for social research. Newbury Park, Sage Publications. Pp 13-20.
43. Rosenthal, S.H. (1972) Electrosleep; a double-blind clinical study. *Biological Psychiatry* 4(2):179-185.
44. Rosenthal, S.H., and N.L. Wulfson (1970) Electrosleep: A clinical trial. *American Journal of Psychiatry* 127(4):175-176.
45. Rosenthal, S.H. and N.L. Wulfsohn (1970) Electrosleep: A preliminary communication *Journal of Nervous and Mental Disease*. 151(2) 146-151.
46. Rosenthal S.H., and N.L. Wulfsohn (1970) Studies of electrosleep with active and simulated treatment. *Current Therapeutic Research*. 12(3):126-130.
47. Ryan, J.J. and G.T. Souheaver (1976) Effects of transcerebral electrotherapy (electrosleep) on state anxiety according to suggestibility levels. *Biological Psychiatry* 11(2):233-237.
48. Ryan, J.J. and G.T. Souheaver (1977) The role of sleep in electrosleep therapy for anxiety. *Diseases of the Nervous System*. 38(7):515-517.
49. Sausa, A.D. and P.C. Choudbury (1975) A psychometric evaluation of electrosleep. *Indian Journal of Psychiatry*. 17:133-127.

50. Schmitt, R., T. Capo, and E. Boyd (1986) Cranial electrotherapy stimulation as a treatment for anxiety in chemically dependent persons. *Alcoholism: Clinical and Experimental Research*. 10(2):158-160.
51. Schmitt, R., T. Capo, H. Frazier, and D. Boren (1984) Cranial electrotherapy stimulation treatment of cognitive brain dysfunction in chemical dependence. *Journal of Clinical Psychiatry*. 45:60-063.
52. Schroeder MJ, Barr RE (2001) Quantitative analysis of the electroencephalogram during cranial electrotherapy stimulation. *Clinical Neurophysiology*. 112(11):2075-83.
53. Selye, H. (1978) *The stress of life*. New York:McGraw-Hill.
54. Shealy, C.N., R.K. Cady, R.G. Wilkie, R. Cox, S. Liss, and W. Clossen (1989) Depression: a diagnostic, neurochemical profile and therapy with cranial electrical stimulation (CES). *Journal of Neurological and Orthopaedic Medicine and Surgery*. 10(4):319-321.
55. 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, kpp 31-33.
56. Smith, R.B. (1982) Confirming evidence of an effective treatment for brain dysfunction in alcoholic patients. *Journal of Nervous and Mental Disease*. 170:275-278.
57. Smith, R.B. (1999) Cranial electrotherapy stimulation in the treatment of stress related cognitive dysfunction, with an eighteen month follow up. *Journal of Cognitive Rehabilitation* 17(6):14-18.
58. Smith, R.B. (2001) Is microcurrent stimulation effective in pain management? An additional perspective. *American Journal of Pain Management*. 11(2):62-66.
59. Smith, R.B., A.E. Burgess, V.J. Guinee, and L.C. Reifsnider (1979) A curvilinear relationship between alcohol withdrawal tremor and personality. *Journal of Clinical Psychology*. 35(1):199-203.
60. Smith, R.B., and E. Day (1977) The effects of cerebral electrotherapy on short-term memory impairment in alcoholic patients. *International Journal of the Addictions*, 12:575-562.
61. Smith, R.B. and L. O'Neill (1975) Electrosleep in the management of alcoholism. *Biological Psychiatry*. 10(6):675-680

62. Smith, R.B. and C.A. Ryser (2000) *Important Things we Learn When Research Goes Awry*. Paper presented at the International Oxidative Medicine Association conference, Denver, Colorado.
63. Smith, R.B. and F.N. Shiromoto (1992) The use of cranial electrotherapy stimulation to block fear perception in phobic patients. *Current Therapeutic Research*. 51(2):249-253.
64. Smith, R.B., A. Tiberi, and J. Marshall (1994). The use of cranial electrotherapy stimulation in the treatment of closed-head-injured patients. *Brain Injury*. 8(4):357-361.
65. Solomon S, Elkind A, Freitag F, Gallagher RM, More K, Swerdlow B, Malkin S (1989) Safety and effectiveness of cranial electrotherapy in the treatment of tension headache. *Headache*. 29(7):445-50.
66. Straus, B., A. Elkind, and C.A. Bodian (1964) Electrical induction of sleep. *American Journal of Medical Sciences*. 248:514-520.
67. Strogatz, S. (2003) *Sync; the Emerging Science of Spontaneous Order*. New York: Hyperion books. pp 70-100.
68. Taylor, D.N. (1991) *Effects of cranial transcutaneous electrical nerve stimulation in normal subjects at rest and during stress*. PhD Dissertation, Brooklyn College of the City University of New York.
69. Taylor DN, Lee CT (1992) Frequency-dependent effects of sine-wave cranial transcutaneous electrical nerve stimulation in human subjects. *Acupuncture and Electrotherapeutic Research*. 17(3):221-7.
70. Tyers. S., and R.B. Smith (2001) A comparison of cranial electrotherapy stimulation alone or with chiropractic therapies in the treatment of fibromyalgia. *The American Chiropractor*. 23(2):39-41.
71. Tyers, S. and R.B. Smith (2001) Treatment of fibromyalgia with cranial electrotherapy stimulation. *The Original Internist* 8(3):15-17.
72. Von Richthofen, C.L. and C.S. Mellor (1980) Electrosleep therapy: a controlled study of its effects in anxiety neurosis. *Canadian Journal of Psychiatry*. 25(3):213-229.
73. Voris, M.D. (1995) An investigation of the effectiveness of cranial electrotherapy stimulation in the treatment of anxiety disorders among outpatient psychiatric patients, impulse control parolees and pedophiles. *Dallas:Delos Mind/Body Institute*, pp 1-19.

74. Voris, M.S. and S. Good (1996) Treating sexual offenders using cranial electrotherapy stimulation. *Medical Scope Monthly*. 3(11):14-18.
75. Weingarten, E. (1981) The effect of cerebral electrostimulation on the frontalis electromyogram. *Biological Psychiatry* 16(1):61-63.
76. Weiss, M.F. (1973) The treatment of insomnia through use of electrosleep: an EEG study. *Journal of Nervous and Mental Disease* 157(2):108-120.
77. Winick, R.L. (1999) Cranial electrotherapy stimulation (CES): A safe and effective low cost means of anxiety control in a dental practice. *General Dentistry*. 47(1):50-55.
78. Wood PB (2003) Stress and dopamine: implications for the pathophysiology of chronic widespread pain. *Medical Hypotheses*. 62:420-4.
79. www.adaptogensonline.com
80. Yamazaki, S. et al (2000) Resetting central and peripheral circadian oscillators in transgenic rats. *Science* 288: 682-685.

Biographical Sketch of the Author

After serving in the U.S. Military as a Field Medical Officer on the General's Staff of the 4th Division Infantry Artillery at Ft. Lewis, Washington, Dr. Smith Completed his Ph.D. in Physiological Psychology at the University of Texas at Austin.

He taught at The State University of New York in Cortland, and served as Senior Research Scientist at American University's Center for Research in Social Systems, before NIMH sent him on a grant to serve as Chief of Research and Clinical Training at the District of Columbia's 600 bed Rehabilitation Center for Alcoholics. It was there he first became acquainted with CES

In 1972 he began his first research with one of America's original CES devices, the Neurotone 101, which the FDA later grandfathered and which still remains the standard CES device against which all newly developed CES units must be compared for substantial equivalency prior to entering the U.S. market.

In researching the CES claims for the effective treatment of depression, anxiety and insomnia with hundreds of hard core drug and alcoholism patients, Dr. Smith serendipitously discovered that the treatment totally reversed the "permanent brain damage" he and other psychologists at the Center had been diagnosing.

He joined NeuroSystems, the manufacturer of the Neurotone 101, in 1980, and has since worked in the CES field with several CES companies in all parts of the U.S., aiding them with start up and development. In addition to publishing more than 20 studies of his own, he has continued to assist others in research design and implementation, thereby earning the title, Mr. CES, in widespread CES circles.

Dr. Smith presently resides on the Chesapeake Bay in Maryland, where at this writing he is engaged in research projects from Seattle, and Santa Barbara on the West Coast, to Pennsylvania on the East.