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The efficacy of cranial electrotherapy stimulation (CES) for the relief of anxiety and depression among polysubstance abusers in chemical dependency treatment

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THE EFFICACY OF CRANIAL ELECTROTHERAPY STIMULATION (CES) FOR THE RELIEF OF ANXIETY & DEPRESSION AMONG POLYSUBSTANCE ABUSERS IN CHEMICAL DEPENDENCY TREATMENT

by
FAUST BIANCO, JR.

A dissertation submitted in partial fulfillment of

the requirements for the degree of Doctor of Philosophy

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PSYCHOLOGY

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ABSTRACT

Bianco, Faust, Jr. (Doctor of Philosophy in Psychology)

The Effectiveness of Cranial Electrotherapy Stimulation (CES) for the Relief of Anxiety and Depression Among Polysubstance Abusers in Chemical Dependency Treatment

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(345 words)

For the last two decades there has been a controversy over the effectiveness of cranial electrotherapy stimulation (CES) for the treatment of the withdrawal syndrome of chemical dependency, especially affective symptoms. Several researchers claimed any effectiveness of CES was due to placebo effect. The literature pertaining to the use of CES with chemically dependent individuals was subjected to meta-analysis as a pilot study and showed some evidence for effectiveness of CES. For the present study, 65 subjects originally were recruited. The subjects were polysubstance abusers referred for treatment by the State of Oklahoma . They were randomly assigned to three experimental groups. Twenty-nine subjects completed the full course of CES treatments. All three groups received identical medical detoxification protocols and treatment milieus at two treatment centers. In addition, one group received active CES treatment, another received sham CES treatment, and the

third group received no CES treatment. The experimental design was completely double blinded and methodological measures were taken to control for placebo effects throughout the course of the investigation. Identical anxiety and depression pretreatment and posttreatment assessment was completed by all groups, consisting of three self-report measures (Beck Anxiety and Depression Inventories, and the Symptom Check List) and two observer-rated measures (Structured Interview Guide for Hamilton Anxiety and Depression Scales) administered by two researchers. Analysis of variance revealed significant group differences. Scheffe post hoc tests showed a statistically significant improvement among the active CES treatment group over the sham CES and no-CES control groups as measured by the observer-ratings. However, the self reports showed no statistical differences between groups. No statistically significant placebo effects were demonstrated as a component of CES treatments. Effects sizes were also calculated regarding the efficacy of CES treatment relating to the improvement of anxiety and depression for this experiment. The impact of the results is discussed, especially the differential results between self-report and observer-ratings and how these differences relate to future studies of CES relating to chemical dependency. Also discussed are the possible mechanisms of CES and alternative uses of CES in the treatment of substance abuse and affective disorders.

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CHAPTER I

INTRODUCTION

A Brief History of Electricity and Electromagnetic Fields as a Clinical Intervention

Cranial electrotherapy stimulation (CES), the use of low intensity, low frequency electrical impulses applied transcutaneously to the cranial area, represents one of the more contemporary if not controversial clinical applications of electricity in medicine and psychology. Electricity and electromagnetic fields for clinical interventions have a long and varied background. Their origins date back several millennia to the time of the Greeks when Aetius prescribed the shock of the electric fish, the "torpedo", as a treatment for gout (Reynolds, 1971). According to Tyler (1990) the torpedo was used by medieval Arab physicians for the relief of such diverse maladies as migraine headaches, sleeping disorders, seizures, and melancholia. "This use of the electric fish represents an ancient precursor of electroshock therapy for severe depression" (Tyler, 1990, p. 145). Also, during the middle ages Paracelsus, the foremost physician of his time, believed that magnets held the secret of healing for all disease (Reynolds, 1971).

In 1780 Luigi Galvani first observed that an electrical impulse caused contractions in the muscle of a frog. "From the famous experiments of Luigi Galvani (1723-98) came the 'Ramsden' type of electrogenerator, used for defibrillation, as well as other uses." (Bentall, 1990, p. 3). The first book published on electrotherapy was that of Christian Gottlieb

Krantzenstien. This physician proposed reducing swelling in certain patients by inducing an electrical field around the patient, through the revolution of a glass globe. He explained his results stemmed from driving out excess blood from the affected tissue (Reynolds, 1971). There is an interesting side note to Krantzenstien's observation that an electrical field could reduce edema. In the first paper relating the use of CES to relieve drug withdrawal, Wen and Cheng (1972) state that they noticed the serendipitous relief of withdrawal while applying CES to opiate addicted neurosurgery patients solely for reducing edema.

In the late 19th century electrotherapeutics became commonplace. The static electricity machine, in particular, was used as a treatment for many ailments such as: anemia, pain, numbness, constipation, spasms, and paralysis. Much of what then constituted electrotherapy has been debunked by modern medicine. However, remember that the use of X-ray came out of the same research trend. Also, techniques of electronic muscle stimulations have become specialized because of this movement, as is exemplified by the development of the pacemaker (Reynolds, 1971). The use of diagnostic tools such as evoked potential devices and electromagnetic imaging devices can be viewed as a direct extension of this early work in electrotherapy.

Electric convulsive therapy (ECT), previously known as electroshock, was seen as a panacea by psychiatry when it was first introduced in Italy during the 1930's by Cerletti and Bini (Davison & Neale, 1986). Later ECT fell into disrepute because of overuse and its seizure related side effects (Davison & Neale, 1986). However, in recent years ECT has come back into favor with some psychiatrists for the relief of acute depression. This increased use of ECT is due to advances in the application of ECT, particularly the use of

modern anticonvulsants and muscle relaxants (Davison & Neale, 1986).

In the 1960's neurophysiology and psychology were aided by advances in electronics in the development of a popular technique called Biofeedback (Davison & Neale, 1986). With this technique the client is afforded the possibility of a modicum of control over his/her autonomic nervous system. This is done though either visual or auditory stimuli that are linked to autonomic nervous system monitors attached to sensors at key positions on the client's anatomy.

Also, during the 1960's the international research community gained interest in what was called "electrosleep," later called cranial electrotherapy stimulation (CES). This international interest in electrosleep was sparked by the publication of proceedings held at the International Symposia for Electrotherapeutic Sleep and Electroanesthesia in Graz Austria in 1966 and 1969 (Wageneder & Schuy, 1967; 1970).

Simultaneously, the transcutaneous electrical nerve stimulator (TENS) was developed for the stimulation of peripheral nerves. The use of TENS developed after Melzack and Wall (1965) proposed the gate-control explanation for pain sensitivity. Positive results were demonstrated using the TENS for the treatment of acute and chronic pain. This resulted in the production of various portable TENS devices (Shealy & Mortimer, 1971). Many studies showing pain reduction in the 50% to 75% range, depending on the type and location of pain, have led to the wide use of TENS (Ebersold, 1977; Loeser, et al., 1975; Melzack, 1975). TENS devices are sometimes used in the same way as CES (being placed on the cranium for relief of disorders other than pain), and such use is generally termed cranial TENS (Taylor et al., 1991).

The History of CES

The roots of CES go back to the very beginning of the 20th century. Leduc was the first to extensively study the use of "electroanesthetic currents" in 1902. Leduc used an intermittent current of 0.5 milliamperes and 12-80 volts. The technique produced narcosis in animal and human subjects that allowed surgery to be performed on subjects with no pain or discomfort (Dodge, 1967).

The conception of "electrosleep" began with a proposition from the fertile mind of Ivan Pavlov in the 1920's. His by now classic observation of the conditioned salivation reflex in dogs led him to propose that internal inhibition and sleep were essentially an identical process. He proposed that sleep was the spread of internal inhibition over the entire cortical and subcortical structures. Pavlov hypothesized that monotonous cyclical stimuli, be they auditory, visual, or electronic would produce internal inhibition and thus sleep (Pavlov, 1927).

Following Pavlov's aforementioned proposition, Soviet researchers in electroanesthesia found a way to induce sleep by spontaneously applying pulsed currents of 0.5-3.0 milliamperes and 100 Hertz to the central nervous system. Over the next forty years many studies (primarily in the then Soviet Union) reported the utility of electrosleep for intervention in various psychological and psychosomatic disorders. These Soviet reports were received with skepticism in the Western research community. Critics pointed to a lack of methodological controls, flaws within experimental designs, and a lack of consistent and reliable diagnostic criteria (Iwanovsky & Dodge, 1966; Von Richthofen & Mellor, 1979; Wageneder et al., 1969). Dodge (1967) indicated that the relative inaccessibility of Eastern

Bloc data, the paucity of Western research in comparison to the abundance of Soviet research, and an innate suspicion of anything Soviet led to a further mistrust of the technique itself.

According to Dodge (1967), the technique of "elektroson" (Russian for electrosleep) was established by Liventsen, Giljorovsky, Ye, Segal and Kirillova in 1949. However, the Russians Banshchikov, Kulikova, and Ye (1970) say that the first description of electrosleep was published by M. O. Gurevich in 1946 while working at the Institute of Psychiatry. Simultaneously, S. S. Korsakov was working on elektroson impulses at the First Moscow Medical Institute. Whatever the truth may be, clearly the Russians were the first to conceptualize and operationalize the treatment of electrosleep. Dodge (1967) lists 30 separate institutes in the Soviet Union where electrosleep or CES research was conducted between 1949 and 1966. Iwanovsky and Dodge (1968) describe an extensive research and use of CES in the Soviet Union and Eastern Block countries during the 1950s and 1960s. After the first of the symposia on electrotherapeutic sleep and electroanesthesia held in 1967 and 1969 in Graz, Austria (Wageneder & Schuy, 1967; Wageneder & Schuy, 1970), Western interest was sparked by several reviews published in the Foreign Science Bulletin (Dodge, 1967; Iwanovsky & Dodge, 1968; Wageneder Iwanovsky, & Dodge, 1969; Wageneder & Schuy 1967; 1970).

The application of transcutaneous electrical stimulation applied to the cranium for the relief of anxiety related disorders began in the Soviet Union during the 1950s. The intervention involved the use of a pulsed direct current of low frequency (100 Hz or less) and intensity (less then three milliamperes). This was done to normalize and calm the central

nervous system. The treatment was originally called "electrosleep" because of the observations that patients often feel asleep during the treatment. The restorative effect of this artificially induced sleep was believed to be at the core of the treatment's effectiveness (Frankel et al., 1973). Electrosleep as a name for this treatment was changed to "cerebral electrotherapy" (CET) shortly after the International Symposia for Electrotherapeutic Sleep and Electroanesthesia. This was due to reports suggesting electrosleep was not a reliable means of sleep induction (Frankel et al., 1973) and because it appeared that sleep was not a necessary aspect of successful treatment (Wageneder, Iwanovsky, & Dodge, 1969).

In subsequent years the treatment has been termed "Transcerebral Electrotherapy," "Electric Transcranial Stimulation," and "Neuroelectric Therapy." Later, because the electric stimulation was applied transcutaneously to the cranial area, the name again was changed to "cranial electrotherapy stimulation" (Smith, 1985). These terms often have been used interchangeably in the field. However, the term cranial electrotherapy stimulation is currently favored by the FDA (Smith, 1985). For the sake of clarity, during the remainder of this paper, the treatment is referred to only as cranial electrotherapy stimulation and abbreviated as "CES."

As mentioned, there was initially much criticism in the Western scientific community of the early Eastern Bloc research (Dodge, 1967; Iwanovsky & Dodge, 1968; Van Posnak, 1969; Von Richthofen & Mellor, 1979; Wageneder et al. 1969;). Responding to these criticisms, Western researchers designed studies with double blind procedures, randomized subject pools, control subjects, relevant outcome measures, descriptive statistics and placebo procedures. However, it must be noted that few of these Western studies included all of the

above. Specifically, regarding the improvement of substance abuse withdrawal symptoms, it can be said that none of the studies reviewed by meta-analysis (O'Connor, Bianco & Nicholson, 1990) included all of the above criteria. This does not allow for more than a cautious interpretation of results.

Smith (1985) notes that most of the American research has concentrated on the effectiveness of CES in clinical trials as opposed to investigating the mechanisms by which it may work. Most of this research has related to the amelioration of mood disorders, stress, insomnia, drug effects, and cognitive dysfunction (O'Connor, Bianco, & Nicholson, 1990). The conclusions drawn from this body of research can be generally categorized into one of three general categories: 1) CES is effective in the alleviation of anxiety, depression, chemical withdrawal effects, stress, insomnia, and cognitive dysfunction in clinical trials; 2) CES is not an efficacious treatment for symptoms; and 3) any positive effects of CES can be explained as due to suggestion or placebo effect. Because of the questionable methods of most of the studies done in this field, any conclusion must be looked at judiciously. This circumspect attitude is necessary because of the questionable implementation of procedures and sampling techniques, the subjectivity and invalidity of many of the outcome measures, poor or inadequate research protocols and other methodological errors evident in many of these studies, especially in the area of drug withdrawal (O'Connor, Bianco & Nicholson, 1990). Consequently, the current study intends to rectify the shortcomings of methodologies used in previous CES drug withdrawal studies.

CHAPTER II

OVERVIEW OF CES RESEARCH

Early Eastern Bloc Studies

Lewis (1966) reports a case study of insomnia, done by Rabinovich in 1914, as the first instance of electroanesthesia being used as a CES therapy. Rabinovich placed electrodes on the patient's forehead and hands and passed 0.75 milliamperes (mA) of electrical current transcutaneously. This treatment resulted in the patient falling asleep within a few minutes. Rabinovich reports that the patients had no harmful side effects and awakened feeling rested and refreshed. It appears this was the beginning of the clinical application of CES. After Rabinovich's work, there was not much work in the CES field for the next three decades. One might presume both World Wars and the Russian Revolution created more pressing priorities for Soviet research. The next research done in CES was that of Giljorowski and his colleagues done in the late 1940s (Dodge, 1967; Lewis, 1966). Lewis notes that Giljorowski and his colleagues began to move toward low frequency and low current treatments. Based on Pavlov's (1927) findings that weak stimulation created sleep, Giljorowski hypothesized that electrical stimuli of very low frequency and current might induce natural inhibition and therefore sleep (Lewis, 1966). Giljorowski observed states resembling deep relaxation or sleep during these experiments with low frequency and current.

As stated, the Eastern Bloc literature on CES is characterized by a paucity of rigorous scientific methodology. Many studies were observations of clinical experience, and were based on case studies. The following comment of Wageneder et al. (1969) exemplifies the reaction of western researchers to the eastern Block CES and electroanesthesia studies:

> The general opinion, however, is that the available Soviet publications do not provide a sufficiently objective view of either technique. In particular, electrosleep therapy is being widely publicized by the Soviets. However, the pertinent publications frequently provide only general statistics, of little clinical value, in the number of "successes, " and do not deal with the physiological mechanism of electrosleep or include an objective evaluation of clinical results. Although there is no doubt that the Soviet reports are authentic, there is considerable dissatisfaction among U. S. researchers and clinicians with the general character of these studies. (p. 21)

The Soviet studies will be only briefly summarized for the sake of succinctness and due to their lack of scientific rigor. Suffice it to say, the clinical experience of the Soviet researchers led to their belief that CES was quite effective. It was considered efficacious for most psychological disorders and for physiological problems such as hypertonia, eczema, dermatitis, and nocturnal enuresis (Andreyeva, 1967; Putan, 1967; Turayeva, 1967; Vish, 1967).

Most of the Eastern investigations continually related positive psychological changes in affect, mood, self-image, and self-relevant beliefs. The two following statements by Banshchikov, Routenburd, and Lulikov (Lebedeva), (1970) and Banshchikov, Kulikov (Lebedinskaya), Ye, Dezdenezhnykh and Sinissina (1970) as also noted by Snodgrass (1977) exemplify the Soviet's positive attitude concerning the efficacy of CES:

> Their nocturnal sleep improved, they began to feel rested after sleeping, their mood was more even, and their efficiency improved. In the second half of the electrosleep course, the patients grew self-confident and confident in their recovery. (Banshchikov, Roytenburd, & Kulikova (Lebedeva), 1970, p. 20)

> Along with better nocturnal sleep, the patient's general condition also improved. They were less irritable, there were fewer headaches, less fatigability, more even mood, paler delusions and fewer phobic manifestations, less marked fluctuations in their condition, and less anxiety. (Banshchikov, Kulikov (Lebedinskaya), Ye, Dezdenezhnykh & Sinissina, 1970, p. 14)

As mentioned, Western research findings relating to CES can be grouped into three categories of results: 1) efficacious 2) non- efficacious and 3) placebo effect related results. The remainder of this chapter will be an exposition of samples of the research fitting into each of these categories. This author believes that these exemplars represent the body of CES literature relating to mood disorders, insomnia, physiological effects and substance withdrawal. The body of literature on CES is rather extensive (over 180 CES studies were reviewed for this study). Yet much of the methodology is flawed, particularly in the area of effective controls for the analysis of placebo effects that may confound the conclusions drawn from the results of the studies.

CES Results Considered Clinical Efficacious

Foster, Post, and Benton (1963) may be the first Western investigators to show the efficacious clinical use of CES. These investigators were interested in muscle spasm control during the rehabilitation of 17 patients suffering from central nervous system traumas. These 17 patients were compared to 6 healthy controls. Electrodes were placed on the frontal and occipital areas of the cranium and the passage of current was from posterior to anterior. The researchers used direct current (DC) pulsed at a frequency of 20-40 Hertz (Hz) with an intensity of 0.5 to 0.8 milliamperes (mA). The investigators described a reduction in muscular rigidity and spasticity, pulse rate, blood pressure, and respiration. The authors believed that

CES could be a valuable tool in the rehabilitation of patients with CNS traumas. In addition, the reduction of physiological signs associated with anxiety lead the authors to speculate CES might also be beneficial in the reduction of anxiety disorders. Unfortunately, the lack of blinding procedures, statistical analysis, standard treatment protocols, no treatment controls and placebo controls attenuate the authors' conclusions.

The first double blind study in the CES area was performed by Straus, Elkind, and Bodian (1964). Thirty-four in-patient subjects suffering from insomnia were used in a cross-over design to compare CES to 100 mgs. phenobarbital and a sham CES procedure paired with 100 mgs. of phenobarbital. The CES was done with electrodes placed similarly to the Foster et al. design with the 30-40 Hz of rectangular DC current passing from posterior to anterior at an unspecified intensity. The investigator observed that the active CES sleep induction approached the effectiveness of the phenobarbital and was more effective than the placebo. Again, one should accept their conclusion cautiously due to methodological problems. Primarily, the problem lies in the outcome measures used to evaluate sleep onset, intensity, and duration. The outcome measures were nurse progress notes based upon intermittent visual observation of the subjects with no self-report given by the patients. When subjects were later asked to give their subjective evaluation of sleep, there was little convergence with the nurses' observations. The subjects reported little difference between the active and the sham CES treatments. There is an additional problem relating to placebo effect control. No sensation of treatment was felt by the sham CES group while the active group felt an electrical sensation throughout the treatment. It is possible that the sham CES group was not convinced that they were receiving the same treatment as the active CES. This

skepticism might be particularly true if the sham and active groups engaged in between group communication relating to specifics of their respective treatments or members of one group had knowledge of the specifics concerning the treatment effects or sensations of the other group. This knowledge might differentiate the groups' expectations for improvements (Frankel et al. 1973). The differential sensation of treatment would be likely would be true because the cross-over aspect of the experimental design would allow each subject to have various treatment sensations during the different phases of treatment. This perceived treatment difference may have been related to the technicians carrying out the treatment, thus compromising the double-blind procedure. In addition, no inter-subjects variability statistics relating to the treatment course are presented. The number of treatments given to individual subjects ranged from six to twelve treatments depending on the discharge parameters of each patient. These methodological confounds challenge the authors' positive treatment conclusions.

Wageneder et al. (1969) treated 386 elderly persons suffering from primary insomnia with an average of twenty 90 minute CES sessions of unspecified current and intensity. Based on clinical observation, the investigator concluded that 272 subjects improved immediately after treatment because they "appeared" to be sleeping. Wageneder interpreted this finding to be highly significant and cautioned skeptical clinicians not to underestimate the potency of CES. However, due to the subjectivity of the outcome measures and flaws in control procedures, statistical analysis, and the description of treatment parameters, it is difficult to be as enthusiastic about the results of this study as are the original investigators.

Rosenthal and Wulfsohn (1970a) used more than 40 (the exact number of subjects not

mentioned) non-randomly chosen neurotic subjects suffering with symptoms of anxiety, depression and insomnia. These subjects were outpatients attending a mental health clinic for a period from four months to many years. They were considered resistant to the standard treatments used at the facility. CES was given with electrodes placed on the frontal and occipital area of the cranium. A pulsed rectangular DC current of 100 HZ with current intensity of 0.5 to 1.0 mA was used. The number of treatments subjects received ranged from seven to ten. The initial treatment was 10 minutes, the second treatment was 20 minutes, and all subsequent treatments were 30 minutes in duration. After five to ten treatments two-thirds of the subjects were deemed to have significant reduction of their symptoms by subjective report and clinical observation.

Encouraged by these results Rosenthal and Wulfsohn (1970b) conducted a second study with six outpatients and three inpatients using the same treatment parameters described in their earlier study. Outcome measures consisted of a pre-post treatment self-report of depression, subjective sense of well-being, clinical evaluation of anxiety, depression, and sleep disturbance, and clinical interviews conducted with the subject's relatives. Evaluation of the subjects showed a significant improvement, according to the authors. Five of the out-patient subjects and one in-patient had remission of their symptoms. However, neither of these experiments had controls for normal treatment or placebo effects, nor were there any blinding procedures.

A third study by Rosenthal and Wulfsohn (1970c) reported on 12 outpatients with the same symptomatology. The same treatment parameters and outcome measures were used as in the previous study (Rosenthal & Wulfsohn, 1970b). Because 10 subjects substantially

improved in less than 6 treatments, the authors felt that the role of suggestion relating to treatment outcome begged investigation. To examine this question, 6 additional patients with similar symptoms were given 5 treatments identical to the previous subjects with the exception that cranial electrodes were not attached to the machine. Four of the 6 subjects in this sham treatment showed partial remission of symptomatology, but none showed the "dramatic" remission of symptoms seen with the active CES. This may be the first indication that a placebo effect was involved in the positive findings shown for CES.

Koegler, Hicks, and Barger (1971) used 15 subjects with a variety of mood and personality disorders with concomitant anxiety, depression, and insomnia. The investigators wished to study CES's effect on sleep patterns. The study was an uncontrolled pilot study. Each patient was given 15 treatments over a three-week period (no treatments were given on weekends). CES was done with electrodes placed on the frontal and occipital areas of the cranium using a rectangular DC current of 40 Hz with an intensity of 1.0 mA. Fourteen of the 15 subjects completed the study. Twelve subjects were reported to have significant improvement of symptoms. Unfortunately, the specifics of outcome measures for evaluation were not specified. Some patients maintained complete remission after a four-month follow-up, while others had a partial relapse. This study was uncontrolled for no-treatment or normal treatments and placebo effects. Caution should be taken in sharing the author enthusiasm for their positive finding in a study with no mention of the specific dependent variables, statistical analysis, blinding procedures, or controls.

In a double blind study Rosenthal (1972) administered either an active CES or a sham CES. In the sham treatment, the electrodes were disconnected from the device. Twenty-two

individuals suffering from neurotic and personality disorders with concomitant anxiety, depression, and insomnia were used as a subjects. The subjects were given half hour CES treatments for five consecutive days. The electrodes were placed on the frontal and occipital cranial areas and the current was passed from posterior to anterior. Treatment consisted of a rectangular DC pulse of 100 Hz frequency and an intensity of 0.5 to 1.2 mA. An unspecified number of subjects received medications throughout the course of their treatment, yet evidently some subjects did not. Outcome measures consisted of pre-post treatment psychiatric interviews. These showed a significant reduction of sleep disturbance and anxiety among the active CES subjects. There was no improvement of depression for the active CES subjects. None of the sham CES subjects showed improvement for any symptomatology. As with the Strauss et al. (1964) study, the active CES group felt a "tingling" throughout the treatment, but the sham CES group did not. As stated earlier, it is believed such a procedure invalidates not only the placebo control, but possibly the blinding procedure. Beyond the solely qualitative nature of the dependent measures, the lack of statistical analysis, or even descriptive statistics makes the acceptance of the study's positive findings difficult.

Weiss (1973), in a double-blind, placebo controlled study of 10 nonclinical subjects, looked at the induction of sleep in "normals." Fifteen minute CES sessions of an unspecified frequency, intensity and polarity were administered to two groups of five subjects each for twenty-four consecutive days. One of these groups received active CES treatments while the other received sham CES treatments. Electroencephalograms (EEG) were taken before and after sleep induction, as an outcome measure. The results showed a significant reduction of

sleep onset latency, percentage of in-bed wakefulness, and total time in stage one sleep among the active CES as compared to the sham CES group. The findings are again suspect for several reasons: 1) the sample is small 2) the MMPI was used to screen for psychotics, and by chance the placebo group scored significantly higher on the Anxiety, Mania, Psychopathic deviance, and Schizophrenia scales than did the active CES group. This difference may represent a heterogeneous sampling error between groups; 3) The placebo control and the blinding procedures were compromised because the active group felt an electrical sensation throughout the treatment while the sham CES group felt a tingling sensation only for a few moments.

Fenighner, Brown, and Oliver (1973) investigated the efficacy of CES in a double blind study of twenty-three chronic psychiatric out-patients. All the subjects had predominant symptoms of anxiety, depression and insomnia. The subjects received ten consecutive, daily, 30 minute treatments. The transcutaneous electrodes were placed on the frontal and occipital cranium, with a rectangular DC current of 100 Hz and 0.5 to 1.0 mA. This study showed CES improved anxiety and insomnia, but was correlated with an exacerbation of depression. Individual psychiatric ratings were used as a dependent measure in a pre-post treatment design. Again, the double-blind and placebo control procedures were compromised because the active CES subjects felt an electrical stimulation throughout the treatment while the sham CES subjects did not. The positive results are suspect because the subjects who had a "massive worsening" of depression were eliminated from the statistical analysis. In addition, all the active CES patients who initially improved for depression, but relapsed within a month after the treatment ended were eliminated from the statistical analysis.

In a noncontrolled, unblinded study Flemenbaum (1974) studied 25 out-patients with symptoms of anxiety, depression, and insomnia. These subjects were considered resistant to many traditional treatments, and thus, in the researcher's opinion, were considered placebo resistant," The author states that this resistance is "a factor worth considering because they were to be used as their own controls. " (p. 20). CES treatment consisted of electrodes being placed on the frontal and occipital cranium, with a rectangular DC current of 100 Hz and 0.1 to 0.3 mA passing from posterior to anterior. Subjects felt an electric sensation throughout the treatments. Subjects received five consecutive daily treatments of a half-hour each. Outcome measures were psychiatric interview ratings and objective self-reports given in a pre-post treatment design. For all subjects the dependent measures suggested a reduction of anxiety, depression, and insomnia immediately after the treatment phase and again after a six-month follow-up. At first glance, these findings appear rather impressive. However, the study did not have any no-treatment, normal treatment, or placebo control groups other than the author's assumed placebo resistance. Yet, on closer inspection of the results the statistical significance of the study may be viewed as an artifact because the more psychotic of the subjects improved greatly whereas less severely disordered subjects did not. These more psychotic subjects could have moved the whole study toward statistical significance. However, the fact that over 50% of these subjects who had not responded to previous treatments responded very well to CES justifies Flemenbaum's interest in further study of CES treatment for patients refractory to traditional treatments.

Smith and O'Niell (1975) used CES on 47 chronic alcoholics to investigate the affect on mood disturbance among this population. The subjects were administered CES using a

sinusoidal wave pattern pulsed at 100 Hz at an intensity of something less than 1.5 mA with electrodes placed on the mastoid process. This was a placebo controlled, normal treatment controlled, and single-blind study. It was the first experimental use of a technique to insure that the subjects were blind to whether they received active or sham CES treatments. This study used an active CES current level below the subject's tactile sensory threshold for electrical sensation. Unfortunately, the average current of treatment was not reported. A total of fifteen, 40 minute treatments were given to each subject for three weeks, excluding weekends. For both the active and the sham treatments the current was increased to a level of tactile sensation and then reduced to a point just below the subjects' tactile awareness. After this operation the current was turned off for the sham-placebo CES group, while the active CES group received active current. The investigators say that the intensity was never greater than 1.5 mA. Profile of Mood States subscales (i.e. anxiety, depression, anger, vigor, fatigue, and confusion) were used as dependent measures. Results showed that significantly more of the active CES group improved on more of the subscales than for the sham group. The results of this study must be questioned for several reasons. Unless the subjects were suffering alcohol induced neuropathy, they possibly could have felt a current of 0.5 mA or greater (Katims et al, 1986), and although the mean amplitude of current used in the experiment was not reported we do know that as much as 1.5 mA was used. Thus, it may be difficult to generalize improvement regarding anxiety and depression solely on the basis of subscales of the Profile of Mood States as is the case in this study. Further, close inspection of the data shows that the sham CES group improved as much as the active CES group on anxiety, depression and total mood disturbance. Originally, the assignment of

subjects was random using three groups of 12 subjects. However, because of subject attrition the groups were post hoc matched, eliminating the methodological benefits of randomization (Elmes et al., 1989). A more conservative interpretation of the data may have led the researches to a different conclusion.

Jemelka (1975) studied the effect of CES on anxiety among 28 psychiatric inmate prisoners. The subjects were randomly divided into active and sham CES groups. Thirteen successive thirty-minute treatments were given with a sinusoidal DC current of 100 Hz and an intensity of 0.6 mA. The electrodes were placed on the mastoid process. It appears this was the first use of multi-modal outcome measures in the CES literature (Snodgrass, 1977). A brief self-report anxiety inventory, the anxiety subscale of the MMPI, a psychiatric rating of anxiety, a behavioral anxiety scale, and systolic blood pressure were used as outcome measures in a pre-post experimental design. The author states that CES was significantly effective in the reduction of anxiety. Again, the results are in question. Close inspection of the data reveals that whereas the change scores for the self-report showed a significant reduction of anxiety within subjects, it only tended toward significance between groups. In addition, the behavioral indicators showed a trend toward significance but were not significant. The sole use of the Anxiety subscale from the MMPI is considered by some to be inappropriate as a measure for anxiety. It is generally accepted that using subscales as a dependent measure when reliability and validity of the assessment device are based on the test as a whole is a less than scientifically rigorous practice (Green, 1991). In addition, no normal or no-treatment controls were used. Given the above it seems premature for the author to state a positive finding based on the evidence presented.

In two studies Ryan and Souheaver (1976, 1977) used active and sham CES treatments in a double-blind study using a subject population of psychiatric in-patients suffering from what the author described as "significant anxiety." The subjects received five successive treatments of thirty-minute duration at 100 Hz of an unspecified "comfortable" intensity. Outcome measures consisted of a single, brief, self-report anxiety inventory. The authors concluded that CES was a viable anxiety intervention for this population. Again, the conclusions, are suspect because the active groups had a tactile sensation of treatment during the entire treatment process, while the sham groups did not. Additionally, there were no non-treatment controls. Both these factors allow the question of placebo effects to obviate the findings of Ryan and Souheaver (1976, 1977).

The results of the twelve studies used as exemplars for the positive efficacy of CES show the promise of CES as possible alternative and/or addendum to more traditional interventions for psychological disturbances. However, no conclusive position can be taken as to the positive efficacy of CES because of the methodological flaws demonstrated in the studies in this review. It is believed these twelve studies exemplify the methodological flaws plaguing the majority of studies showing the positive efficacy of CES. Thus, it is concluded that there is a need for the current study, because of this investigation's intent to improve on the methodological shortcomings evidenced in previous CES research.

CES Studies Considered Clinically Nonefficacious

The first American study reviewed that showed that CES was clinically nonefficacious was that of Woods, Tyce, and Bickford (1965). This was an uncontrolled, unblinded study

of sleep induction. Twenty-three subjects, whom we can only assume had no sleep disorders (no subject demographics were published), were given CES with electrodes placed on the frontal and occipital areas of the cranium. The current was of an unspecified frequency and intensity. Either one or two treatments ranging from 30 to 40 minutes were given to each subject. EEGs and clinical observations of sleep were used as a dependent variables to learn the subject's onset and depth of sleep. Clinical observation showed that six subjects appeared to attain sleep immediately after CES. However, the EEGs on 17 subjects confirmed only 4 subjects attained a state of physiological sleep given the EEG criterion for sleep induction. The authors concluded that CES was ineffective for the sleep induction. Methodologically this study leaves a great deal to be desired, in particular the fact that only one or two treatments were given, and they were given in an unspecified manner. Given the obvious problems with the above experiment's design and its operationalization, it is difficult to accept the researchers' findings as conclusive.

In 1965 Miller and Mathas (1965) investigated the use of CES for the remission of psychiatric symptoms. This is one of the first controlled studies of CES. Fifty-four subjects mostly psychotic, half who were symptom matched controls, were recruited for the study from a metropolitan psychiatric center. The CES device used was unspecified and the current parameters cannot be determined from the publication. Subjects were given from two to five CES treatments per week. Each session varied from thirty to fifty minutes. Apparently no systematic treatment protocol was used. "Two hundred and twenty-two treatments were given for a total of 160 hours." p 461. (Miller & Mathas, 1965). Results were determined by clinical assessment based on remission of presenting symptoms, adaptability, patient's

willingness to return to normal activities, and the mean weight of psychiatric medications given to each patient over the course of the study. There was no difference between the treatment and control groups on any of the above dependent criteria. Miller and Mathas (1965) concluded that CES was not a viable intervention for a heterogeneous psychotic population. Given the apparent unsystematic operational aspects of this study, the results should be viewed skeptically. However, these finding might support the conclusion of Rosenthal (1972) who states that CES would have the best effectiveness with neurotic patients. . . having good ego strength and without serious major personality disorders" (p. 105). This study is a good example of the difficulties faced by attempting a clinical trial in a clinic setting with its concomitant problems of treatment scheduling and consistency.

In 1973 Astrup (1974) performed a case history archival follow-up study of twenty-one psychotics and twenty-four neurotics treated in Norway with a combination of neuroleptics and CES treatments of unspecified parameters. CES was given for fifteen sessions with a duration of one hour per session. Of the twenty-one psychotics, seven patients died before the follow-study was completed. Of the surviving psychotics, thirteen showed initial improvement. However, using only his clinical judgement as an outcome measure, after perusing the patients' medical records he states, "With a 12 year observation period, I do not think patients did better than they would have done with drugs alone, and only seven could be classified as improved. . . of 24 neurotics I classified 15 as improved immediately after the series of electrosleep treatments, at follow-up, 12 of the 24 neurotics were classified as improved. . . . The long term improvement could be related to various factors, such as improved living conditions, psychotherapy with other physicians, and

behavior therapy with me." (p 116).

Because of the ambiguous nature of the independent variable, no control group baseline rates of traditional treatments, and the lack of reliable dependent measures, it is difficult to accept the conclusion of the study, based on what is published. Astrup concludes that their is no efficacy even though there seems immediate improvement, especially among the neurotic population, based on Astrup's criteria for efficacy. Even though Astrup methodology appears quite flawed, the results of immediate improvement seems to uphold Rosenthal's (1972) findings mentioned previously; that CES appears more effective with neurotics than psychotics, and clinicians should be careful to screen prospective CES candidates with this in mind.

Frankel, Buchbinder, and Snyder (1973) engaged 17 out-patients suffering from insomnia and administered fifteen daily (weekends excluded) CES treatments lasting 45 minutes. The CES was of sinusoid wave patterns at 100 Hz of an unspecified intensity that was described as comfortable to the subject. The CES electrodes were placed on the mastoid process. After the initial treatment the same subjects were given similar CES treatment for 15 days with the exception that the frequency was 15 Hz. The experimental design was unblinded and did not include no-treatment or placebo control groups. Sleep polygraph recordings, self-report mood and sleep inventories, and 17-hydroxycorticosteroid assays were used as dependent measures. The researchers reported no significant differences between pre and post treatment measures. Attempting to explain their negative findings in light of Rosenthal's (1972) study, the authors propose that the more chronic the insomnia the less likely CES would be an effective intervention. They point out that Rosenthal's subjects were

likely suffering from acute insomnia rather than a chronic condition, because none of the subjects suffered with insomnia for more than six months. In the Fenighner et al. (1973) study the subjects were insomnia sufferers of two years or less in duration, the findings were significant but with a smaller apparent effect size than that of the Rosenthal (1972) study. Rosenthal's results, however, were greater in effectiveness than those of Frankel et al. (1973) in which the subjects all suffered with insomnia for more than twenty years. The authors suggest the length of time the subject suffered with insomnia is negatively correlated to the effectiveness of CES. At first glance, some readers might rule out the inclusion of placebo effect as a moderating variable in CES treatment, because of the lack of improvement in change scores between the pretest and the posttest scores within the subject group. However, without a normal treatment control group to establish base rates of improvement, the lack of difference between outcome measures cannot be interpreted as conclusive proof that CES was not effective. Consider the possibility that a normal treatment group may have displayed a worsening of the insomnia over the length of study.

Hearst, Cloninger, Crews, and Cadoret (1974) administered both active and sham CES to 28 chronically mentally ill out-patients with predominant symptoms of anxiety and depression. The subjects were equally and randomly assigned to active and placebo treatment groups. The experiment was double blind and there was no no-treatment control group in the study. Frontal, occipital and mastoid process electrode placements were used with a DC and AC pulsed current of 100 Hz frequency and an intensity ranging between 0.3 and 1.1 mA. Five daily CES treatments lasting thirty minutes were given. All subjects received psychotropic medications throughout the study. This level of treatment might have produced

a tactile sensation perceivable to the active CES subjects (Katims et al.,1986). However, the investigators used tight headbands and electrode paste in an attempt to mask this sensation, trying to blind both the researchers and subjects as to which form of CES was being given to the subjects. Outcome measures were a self-report symptom scale, psychiatric mental status interviews, and global ratings of anxiety, depression and insomnia. No differences were found between groups although all subjects improved significantly. While this may be the first successful attempt at double blinding CES research, the conclusions are confounded by the subjective nature of the dependent measures. Further, close inspection of the data shows that there were more depressives in the active group and more hysterical patients in the placebo group. These facts could have confounded the results because the more suggestible nature of the hysterical patients. The placebo group was comprised of more hysterical patients according to the data, thus this group may have been more susceptible to the suggestion that the treatment was effective adding a confound to the placebo group and driving the results of this group artificially in a positive direction. Also, other studies have shown that CES might be contraindicated for depression (Fenighner et al., 1973; Rosenthal, 1972). If this conclusion is correct the presence of more depressives in the active CES group could have driven down remission of symptoms for the whole group. Also, the subjects were all chronically mentally ill. Rosenthal (1972) warns us that this population may be inappropriate for CES treatment. Given the above the composition of the various groups in the study one cannot rule out an unanticipated but nonetheless confounding variable, that of sampling error.

Moore, Mellor, Standage, and Strong (1975) recruited 17 nonpsychotic subjects suffering predominantly from anxiety, depression, and insomnia. The procedure was

double-blind and employed Rosenthal's (1972) method of sub-threshold tactile sensation for active CES treatment. A cross-over design was used in which all subjects received five active and five sham CES treatments. A sinusoidal wave device with a frequency of 100 Hz was used. The device had a range of intensity between 0.2 and 0.7 with a mean of 0.48 mA being employed for active CES treatments. Unfortunately, all other current parameters and electrode placements were unspecified. Subjects entering the study on medications were allowed to continue their use throughout the study. A variety of clinical ratings of anxiety, depression, and insomnia, self-report mood inventories, and the patient's subjective reports of anxiety and sleep disturbance were used as outcome measures. The only statistically significant difference reported between groups was the subjective report of improved sleep among the active CES subjects. Although the use of reliable and validated multi-modal outcome measures was a welcome advance for CES research, the double blind procedure in this study must be questioned on the basis of work done by Katims (1986). The mean sub-threshold sensation level in the Moore et al. study was 0.48 mA. Katims reports that as little as 0.2 mA has produce a tingling sensation among some subjects, depending on the placement of the electrodes. Because this study was a crossover design, electrode placement is not specified, and current intensity as high as 0.7 mA was used; it is questionable that these subjects who received both active and sham CES were all blind regarding the type of treatment they received.

Taylor et al. (1991) used 90 normal subjects to investigate stress reduction following a single CES treatment in a double-blind laboratory study. The subjects were randomly divided into five groups using various combinations of active CES, sham CES, and no-

treatment controls during the treatment and post treatment stress test. The groups were defined as follows: 1) active CES during treatment and active CES during stress; 2) active CES during treatment and sham CES during stress; 3) sham CES during treatment and sham CES during stress; 4) sham CES during treatment and active CES during stress; 5) no treatment. A sinusoidal wave pattern, with a current frequency of 100 Hz and intensity ranging from 0 to 10 mA was used in this study. The electrodes were placed on the anterior and posterior ear lobes. The single CES treatment lasted thirty minutes, immediately followed by three minutes of standardized mental stress in the form of mental arithmetic (subtracting serial 17s from 4300). To insure the subject blind, the authors devised a method to establish 0% threshold of tactile sensation. This method consisted of a series of five ascending and descending trails finding the highest level at which sensation of the stimulus was perceived 0% of the time. The outcome measures consisted of physiological measures of heart rate, systolic and diastolic blood pressure, and temperature, as well as a self-report of anxiety. A *t*-test showed a statistically significant difference between stress and increased systolic and diastolic blood pressure, and anxiety, but not temperature and pulse rate. No significant differences between active CES, sham CES, and no treatment groups were demonstrated for physiological and psychological outcome measures taken as a whole. However, anxiety was significantly reduced among the active CES groups as compared to the sham CES and no treatment group. No placebo CES effect was observed on any dependent measure. The authors concluded that CES would not be an effective intervention for stress management. This was a rather elegant study, with several innovations in the area of CES research, particularly in the insurance of the blinding procedure. However, the generalizability of the

conclusion might be questioned regarding *clinical* stress on several points. Is the stress created by a mental arithmetic problem comparable to the chronic stress suffered by many individuals seeking stress management? One might surmise that the stress caused by a mental arithmetic task is a rather weak independent manipulation for the stress suffered by clients seeking help from clinicians. On the other hand, one might question if a single CES session of only thirty minutes duration is enough to reduce psychological and/or physiological stress. Also, the post-tests were given only five minutes after CES. It is possible that any stress reduction available from CES may take more than five minutes to manifest. Further, from the perspective of clinical psychology, it could be argued that the reduction of anxiety should take precedence over the reduction of physiological markers of stress in the determination of whether the treatment is effective for the reduction of psychological stress. Further, the clinical value of an intervention should be judged on the efficacy of that treatment beyond five minutes after the treatment has been applied. Clinically, we are generally more concerned with the long-term amelioration of symptoms rather than the short term effects of a treatment. The question is compounded by the fact that the stressor failed to induce change in 40% of the physiological markers. Before the use of CES for stress management is abandoned these questions should be addressed by further investigation.

Studies Considered to Demonstrate Placebo Effect in CES Research

The first mention of placebo effects in connection with CES treatment can be found in the work of Achte, Kauko, and Seppala (1968). CES of frequencies ranging from 5 to 10 Hz and intensities ranging from 0.1 and 0.6 mA were applied to 24 mostly female patients.

The electrodes were placed above the eyebrows. All subjects suffered from chronic insomnia and nearly half were abusers of sedatives or narcotics. The number of treatments varied from between 6 to 29, with treatment duration ranging between thirty minutes and two hours. Dependent measures were subjective reports of symptom improvement from the subjects, nursing staff, and the attending physician. Eighty-three percent of the subjects were judged improved immediately after treatment. However, none of the subjects completely remitted from their symptoms, nor did they completely stop their dependence on medication. However, a two-month follow-up of these improved patients showed that 63% suffered a complete relapse. After the follow-up the authors concluded that the initial very positive result was the result of placebo effects based on suggestion and behavioral conditioning. One must question any firm conclusions or interpretation of results derived from this study due to the extremely loose and varied treatment protocol and the variation of current parameters of the treatments within the sample of subjects.

In 1970 Rosenthal and Wulfsohn (1970c) presented the position that the role of suggestion may play an integral part in CES treatment response, and needed to be investigated. In a replication of their previous study (1970b), Rosenthal and Wulfsohn administered CES to 12 neurotic outpatients with predominant symptoms of anxiety, depression, and insomnia. CES administration and outcome measures were identical to their previous study (Rosenthal & Wulfsohn, 1970b). Five to ten consecutive CES session were given to the subjects. Ten of the subjects substantially improved after only five sessions. Nine of the twelve subjects had nearly complete remission of their symptoms at the end of the CES trials. Eight of the subjects were completely asymptomatic for insomnia. While

impressed with their results the authors stated that the role of suggestion in CES should be further investigated. Six additional subjects with similar symptomatology were given five identical treatments, with the exception that the electrodes were not attached to the CES device. Using the same dependent measures four subjects showed partial symptom remission, while one subject showed the same level of remission displayed by the active CES treatment. This second finding led the authors to propose that suggestion played an inherent role in CES. At first this position seems to converge with the opinion of Shapiro (1968), who argued that persons with significant anxiety and depression respond favorably to all types of placebo treatments. However Krippner and Brown (1973) concluded that symptomatology does not account completely for placebo effects in CES. Their study showed that a single 20 minute exposure to sham CES identical to that used by Rosenthal and Wulfsohn (1970c) produced a significant placebo effect in normal subjects as well. This finding may give weight to Boblitt's (1969) proposal that CES may be nothing more than an "electronic placebo." However, there is a methodological problem with the Rosenthal and Wulfsohn (1970c) study. The sham CES subjects never had a tactile sensation of electrical current, while the active CES subjects did. Frankel (1974) proposed that subjects may have an expectation regarding feelings associated with the application of electrical treatments. Frankel (1974) suggests control subjects who feel nothing with CES used in the same experiment where active CES subjects can feel the treatment, may deduce they are not receiving the treatment, thus effecting the treatment outcome with regard to placebo effect based on expectation of improvement. The strength of this proposal is weakened by the Krippner and Brown (1973) finding that placebo effects are associated with CES where no subject felt an electrical

sensation. Based on the literature relating to the mediational effects of expectation regarding placebo effects (Wilkins, 1985) it seems Frankel's point warrants consideration when interpreting CES data.

Tomsovic and Edwards (1973) engaged 43 chronic alcoholics suffering from insomnia, anxiety, and somatic complaints in a treatment outcome study of CES. An AC current of sinusoidal wave patterns, with a frequency of 100 Hz was used. The CES device had an intensity range of 0.1 to 1.5 mA. The subjects were given five successive active or sham CES treatments lasting thirty minutes. All subjects had been in an alcohol rehabilitation program for three weeks when they were recruited for the study. The dependent measures were physician's reports derived from psychiatric interviews regarding symptom improvement for anxiety, insomnia, headaches, and stomach complaints. The results showed that 75% of both the active and the sham group improved with no statistically significant difference between groups. Tomovic and Edwards (1973) question the efficacy of CES and attribute any symptomatological improvement to be the result of a placebo effect. The authors' interpretation of the data is less than conclusive because they used a solely subjective percentage of improvement as a dependent measure and did not report whether both groups had the same sensation of treatment.

A year later Marshall and Izard (1974) published a CES treatment outcome study using 40 inpatients of heterogeneous diagnoses with predominant depression. The CES device was of DC rectangular wave pattern with a frequency of 100 Hz and a maximum frequency of 1.5 mA. Positive electrodes were placed on the frontal region of the cranium and negative electrodes were placed on the mastoid processes. The subjects were given five

consecutive active or sham treatments of thirty minute duration. Equal numbers of subjects were randomly assigned to either group. Sham subjects had the posterior electrodes disconnected from the CES device. Marshall and Izard (1974) state, "The sensation produced by placebo and cerebral electrotherapy were virtually non-distinguishable, since the amount of current each patient received was individually determined. . . ." (p. 19). Dependent measures consisted of patient and staff ratings on a Izard's (1972) depression inventory, given in a pre-post, one follow-up experimental design. The results showed that all the subjects had improved symptomatology, but there were no differences in improvement between the active and sham CES groups. Results after the follow-up were similar. The work of Katims and Ng (1985) and Katims, Long, and Ng (1986) can be used to dispute the authors' findings. According to these investigators, the passage of current between two sets of orbital electrodes (as was the case in this study) does not rule out the possibility of active CES effects. It follows it just as possible that both groups improved because they received active CES instead of relating the improvement to placebo effect.

Passini, Watson, and Herder (1976) recruited 60 neurotic and psychotic inpatients with heterogeneous diagnosis. All subjects were suffering from anxiety and depression as determined by staff observation. The subjects were randomly placed into either active or sham CES treatment groups. The subjects were given ten successive treatments over a two week period (weekend treatments were not administered). Treatments had a duration of 30 minutes. The electrodes were placed on the eyelids and mastoid process. A sinusoidal DC wave pattern pulsed at a frequency of 100 Hz was used. Subjects were administered a current of unspecified intensity, not exceeding 1.5 mA, just below the level of subjective discomfort.

For the sham group the current was never turned on. Most of the subjects were on psychotropic medications. No attempt was made to alter the subjects' medications or dosages during the CES treatments. Outcome measures consisted of subjective self-reports of depression and anxiety given in a pre-post treatment experimental design. The results showed significant improvements among both the active and sham CES groups with no significant difference between the two groups. In fact, close inspection of the data showed that the non-significant change scores favored the placebo group's improvement on most measures. The authors say that their findings contradict the view held at the time that CES was effective for anxiety and depression and they believe any benefit from CES was attributable to placebo effect. It is difficult to accept these results as conclusive for the following reasons: 1) the study lacks a no-treatment or normal treatment control group; 2) the totally subjective nature of the outcome measures; 3) the difference in the tactile sensation of the treatment between the active and the sham groups; 4) the solely subjective nature of the diagnostic criterion used for the determination of anxiety and depression among the subject population.

A Review of CES Studies in the Area of Chemical Dependence

Because the current study focuses on the use of CES for the relief of symptoms that are often concomitant with chemical dependence (CD) and/or withdrawal the author will present a summary of all the CES work done in the CD area. The data from these studies were analyzed via a meta-analysis (O'Connor, Bianco, & Nicholson, 1990), to be discussed later. A few of the studies mentioned in this section have been summarized previously, but they are included here for the sake of continuity and completeness.

The first published account of CES research involving chemical dependence (CD) was a study by Wen and Cheng (1973). Forty patients admitted to Kwong Wah Hospital in Hong Kong for a variety of ailments who were coincidentally addicted to opiates were engaged in a study using CES combined with acupuncture for withdrawal symptoms. The subjects had been addicted for periods ranging from 3 to 58 years. A CES device of unspecified wave pattern was used at a frequency which was gradually increased from 0 to 125 Hz at an unspecified intensity. The electrodes were attached to acupuncture needles placed in the concha of both ears. The length of treatment varied depending on individual patient needs, averaging 1.5 hours. The number of treatments also varied widely as seen from Wen and Cheng's (1973) comments, "In the first few days of treatment, we gave the patients two or three stimulation per day for two or three days, followed by one stimulations for the next four or five days" (p. 139). Outcome measures were the researchers' clinical observations of patient improvement and the patient's sense of well being. Neither normal treatment, no-treatment nor placebo controls were used in the study. The investigators claim that 39 of the 40 subjects were discharged to out-patient clinics non-addicted and withdrawal symptom free after the CES-acupuncture treatment. All 39 of the subjects suffered little or no withdrawal symptoms during the study. These findings were very interesting and received the attention of numerous clinicians and researchers throughout the world. However, these results are based on clinican observations using very subjective dependent measures. Thus, the results cannot be considered conclusive. Tomsovic and Edwards (1973) (see page 33 for a detailed description of the study) showed that 75% of both the active and the sham group improved with no statistically significant difference between groups. The authors question

the efficacy of CES and attribute any symptomatological improvement to be the result of suggestion based placebo effect. However, the interpretation of the results should be considered less than conclusive because the dependent measures were solely subjective percentage of improvement, and we do not know whether both groups had the same sensation of treatment, or the placement of the electrodes. Thus, according to Katims et al. (1986) if the electrodes were placed in an orbital arrangement, we can not be sure that both groups did not receive active treatments at the level of current intensity used in this study. Also, the fact that it is possible that there was a random differential tactile sensation of treatment within groups could confound the results.

Smith and O'Niell (1975) (for details of the study see p. 19) present a study that they believe demonstrates the efficacy of CES. One must question the positive results of this study for several reasons. Unless the subjects were suffering alcoholic neuropathy they possibly might feel a current of 0.5 mA or greater (Katims et al, 1986). Also, it is generally accepted that using subscales from an assessment whose reliability and validity have been determined from the test as a whole is a less than scientifically rigorous practice (Green, 1991). Thus, it would be difficult to generalize improvements from subscales on a single self-report assessment to clinical states of anxiety and depression. Further, close inspection of the data shows that the sham CES group improved as much as the active CES group on anxiety, depression and total mood disturbance. Originally, the assignment of subjects was random using three groups of 12 subjects. However, because of subject attrition the groups were post hoc matched, eliminating the methodological benefits of randomization (Elmes, Kantowitz, and Roediger, 1989). A more conservative interpretation of the data may have led the

researches to a different conclusion.

In a double-blind study of chronic alcoholics suffering from anxiety, depression, and insomnia McKenzie, Costello, and Buck (1976) recruited 36 subjects. Eight subjects were randomly assigned to each of four groups (i. e. sensation and active treatment, sensation and sham treatment, no sensation and active treatment, and no sensation and no treatment). The device used a rectangular DC wave pattern with a frequency of 25 Hz, intensity of 1.0 mA, with electrodes placed on the frontal cranium and mastoid processes. Five consecutive treatments lasting thirty minutes were give to all subjects. The outcome measures were unvalidated self-reports of depression, anxiety and insomnia created by the authors for this investigation given in a pre-post treatment design. The author recognized the limited number of subjects in each cell would limit the statistical power of their study and chose not to analysis their data statistically. Instead, they graphically represented their findings. "Group means were plotted and the slope of the pre-post line was visually analyzed for trends." (p. 190). From their analysis of the data, the authors conclude that CES could be beneficial for the reduction of anxiety and depression, but is not beneficial for sleep disturbance among the population studied. This study is designed well, in that it attempts to control for placebo effects. However, considering the small number of subjects in each group and the unvalidated nature of the outcome measures a definitive interpretation of the data is difficult at best. In the words of the authors, "In this type of study with small n, conclusions must be offered tentatively." (p 192). Close analysis of sham groups showed improvement on all the measures except one. Also, the normal treatment controls did not improve on any measure. Therefore, one could conclude that the improvement trends shown were the result of placebo effects just

as readily as the authors assume that CES is viable treatment for anxiety and depression. In short the conclusion drawn by the authors is far from conclusive.

Patterson (1976), a former colleague of Dr. Wen, did a follow-up study of 23 CD patients who used various drugs. The subjects were treated 1.5 to 21 months in a London inpatient CD rehabilitation center, before the study began. The CES treatments range in frequency from 5 to 2000 Hz, with an intensity ranging from 1 to 2 mA. Electrodes were place on the concha on the ears. The length of the treatments ranged from four to 35 successive days for 40 minutes. No blinds or control groups were used in this study. The dependent measures consisted of self-reports and clinical observations of degree of "character" improvement (no improvement, marginal improvement, fair improvement, moderate improvement, and marked improvement). However, no baseline of improvement using the traditional treatments were established. The subjects received no pre-treatment or post-treatment assessments. Eighty-three percent of the subjects showed some degree of improvement, based on the author's clinical judgement. Seventy percent of the subjects claimed they remained drug free for the duration of their individual follow-up periods. However, none of the subjects were drug tested to assure their claims of sobriety were true. The author concluded that CES treatment would be a valuable addendum to traditional drug treatment programs. Considering the poor methodology, variations in treatment protocols and current parameters, and the subjectivity and apparent unreliability of the dependent measures of this study one cannot give a great deal of weight to the conclusion of Dr. Patterson.

Smith and Day (1977) investigated CES as an intervention for cognitive deficits

among 227 chronic alcoholics treated in an inpatient rehabilitation center. Subjects were randomly placed into a normal treatment group or one of four treatment groups. The treatment groups were defined as follows: 1) electrodes placed on the frontal an occipital regions of the cranium with the subjects assisting in setting the current levels just below that of tactile sensation ranging from 0.21 to 0.71 mA; 2) electrode placed on the mastoid process with the subject assisting in setting the current level just below tactile sensation ranging from 0.10 to .44 mA; 3) electrodes placed on the mastoid processes with the current set at a fixed level of 0.10 mA; 4) electrodes placed on the mastoid processes with the current set at a fixed level of 0.20 mA. CES treatments were 40 minutes daily for 15 days excluding weekends. Both the normal treatment controls and CES treatment subjects remained in routine therapy throughout the study. The dependent measures of the study consisted of a validated non-verbal I.Q. and a visual retention test, given in a pre-post treatment design. Due to the differential attrition rate between the groups the authors felt it necessary to post hoc match the various groups. The results showed that 55% of the controls deteriorated on the outcome measures, while 84% of the CES treatment subjects had improved scores on the dependent measures. Close inspection of the data shows that the subject assisted current level setting group (group 1) fared the best, followed by the 0.10 mA fixed current setting group (group 3). The 0.20 mA group (group 4) showed little change between pre and post testing, while the normal treatment controls deteriorated in performance. Although, the results show some improvement on dependent variable scores among the CES treatment groups the difference in change scores were not statistically significant between groups. Also, close examination of the differential attrition shows that more severely impaired control subjects left the study

than was the case for severely impaired subjects in the CES treatment groups. Thus, among the controls there were less degrees of freedom for change toward improvement. Unfortunately, the study loses the methodological and statistical benefits of randomization (Elmes et al., 1989; Kachigan, 1986) due to the post hoc matching done after the completion of the study. No placebo controls were used in this experiment; therefore one cannot determine whether the cognitive improvements shown were due to some biopsychological component of CES, placebo effect, or a combination of both. For the above reasons, the study's results can be viewed as interesting and warranting further study of the use CES as an intervention for cognitive dysfunction, but nonconclusive for cognitive dysfunction in this population.

Snodgrass (1977) investigates the efficacy of CES in the reduction of anxiety, depression, and social and behavioral deficits among 36 chronically alcoholic inpatients. The CES device used a sinusoidal wave pattern of 100 Hz with an intensity ranging between 0.10 to 1.5 mA. The electrodes were place on the mastoid processes. Six consecutive daily, 40 minute treatments were given to active and sham CES treatment groups. The author employed no normal or no-treatment controls. All subjects received the usual extensive milieu of multi-modal treatment prescribed at the location where the study was done. CES treatments starting three weeks after the subjects entered a rehabilitation center. Outcome measures consisted of unvalidated self-report mood inventories of the author's own creation constructed primarily from the anxiety and depression subscales of the MMPI. Behavior inventories were also used and completed by staff health care providers. Both the self-report and behavior measures were given in a pre-post treatment design. No significant differences

were found between groups, while all subjects improved. The author concludes that any change toward improvement was due to benefits of the treatment program and long term detoxification and not to CES. Further, he says that CES is not a viable intervention for the affective difficulties faced by the chronic alcoholic. It is difficult to interpret the findings of this study as conclusive. The number of the subjects in each group was only thirteen, reducing the statistical power of the study (Cohen, 1977). The outcome measures were never validated and may have suffered from problems with internal and predictive validity problems (Cook & Campbell, 1979). Anxiety and depression subscales of the MMPI have a fair amount of symptomatic cross-over expressed in them (Green, 1991). The normal treatment regime was very extensive including drug, individual, group, and social skills therapies and the CES was started well after the other treatments began. Thus, any symptomatic improvement may have been in place before CES began.

Gomez and Mikhial (1978) studied the use of CES in methadone detoxification. The researchers engaged 28 subjects in a double-blind study to learn if CES would reduce the anxiety and sleep disturbance that is often concomitant to opiate use and withdrawal (Woody et al., 1986), as well as reduce methadone usage. Fourteen subjects were randomly placed in an active CES group, seven in a sham CES group, and seven in a normal treatment control group. The CES device used produced DC sinusoidal wave of 100 Hz frequency, and an intensity ranging between 0.4 and 1.3 mA. Electrodes were placed on the frontal region of the cranium and mastoid processes. The treatments were for ten consecutive days, excluding weekends with a duration of thirty minutes. Dependent measures were a validated self-report anxiety inventory, a validated structured clinical interview for anxiety, sleep disturbance, and

somatic complaints. Outcome measures also included behavioral observations by staff, and the amount of methadone ingested by the subjects during the study. The dependent measures were given in a pre-post treatment design. The results showed that the fourteen subjects receiving active CES had a statistically significantly greater reduction of anxiety, sleep disorders, somatic complaints and methadone ingestion than the sham CES or normal treatment control groups. Although the results of this study are quite impressive, the conclusions of the authors would carry more weight had there been more subjects in the various groups, particularly the sham and normal treatment groups. Also, the fact that the active CES group felt an electrical sensation during treatment while the controls did not may have flawed the blinding of the of the subjects.

In an uncontrolled, unblinded study, Smith, Guinee, and Reifsnider (1979) investigated the use of CES in a stress management intervention among chronic alcoholics undergoing detoxification and rehabilitation. Forty-three chronic alcoholic in-patients were recruited as subjects for the study ninety-six hours or less after admission to an alcoholic rehabilitation study. The CES device produced an AC sinusoidal wave pattern with a current frequency of 100 Hz and an intensity ranging from 0.10 to 1.5 mA. Electrodes were placed on the mastoid processes. The MMPI and hand tremor measurement were used as dependent measures, given in a pre-post treatment design. The treatment consisted of a single forty minute CES session given at an intensity level just below that of tactile sensation. Results showed that there was a curvilinear relationship between stress as measured by the MMPI and the reduction (group 1) or increase (group 2) in hand tremor following the CES treatment. There were no significant differences between the groups on pretest MMPI subscale scores. The

sham group whose tremor increased scored significantly higher on six of ten subscales on the post treatment MMPI than did the active group whose hand tremor decreased. The authors conclude that CES is an effective stress management intervention for the population under study. Smith et al.'s (1978) conclusion, should be accepted with reservation and deference to construct, internal, and predictive validity (Cook & Campbell, 1979). The authors assume that hand tremor is an indicator of psychological stress among this population ". . . the most common assumption is that internal stress causes the tremor. . . ." (Smith et al, 1978, p. 119). However, 20 to 25% of withdrawing alcoholics never experience any hand tremor (Edward & Gross, 1976; Mendelson & Le Duc, 1964;). Even if hand tremor were an accepted indicator of psychological stress among alcoholics, there is an assumption by the authors that the MMPI is a valid measure of stress. The MMPI is an assessment of pathological personality, and not an instrument validated for the measurement of psychophysiological stress. The use of the MMPI as a stress indicator may be unwarranted. Even if the use of the MMPI were appropriate in this study any conclusion based on the use of six out of ten subscales seems inappropriate given the opinion of Green (1991), as has been previously mentioned. Thus, any conclusions based on the data of Smith et al.'s (1979) study must be viewed with circumspection.

Smith (1982) hypothesized that CES would effectively improve cognitive dysfunction among chronic alcoholics. To test this hypothesis Smith recruited 100 chronic alcoholics from a rehabilitation center, and randomly assigned 50 subjects to an active CES treatment group and 50 subjects to a sham CES group. The CES device produced a sinusoidal wave of 100 Hz frequency with an intensity of 0.10 to 1.5 mA. The electrodes were placed just

below the ears at the maxillo-occipital juncture. CES treatment subjects were given stimulation for 40 minutes for 15 successive days excluding weekends. Outcome measures were the subscales of an I. Q. test given in a pre-post treatment design. Because twice as many subjects fell to attrition from the control group as opposed to the treatment group, the investigator post hoc matched the remaining subjects. The results showed that although the CES treatment group significantly improved more then the CES sham groups on two of the six scales, both groups were nearly identical on the total pre-test post-test change score mean. On the basis of this data, the author claims that CES is a viable intervention for cognitive dysfunction among chronic alcoholics. Again, caution must be taken with the interpretation of the data. The experimental design had no normal treatment or no-treatment controls and the total mean change scores for the placebo group and active group were identical. It seems that the active treatment was no more effective than any placebo effect that was engendered by the sham treatment. Yet one can't be sure if there was a placebo effect, because the study did not include a normal treatment control group (Ross et al., 1962). In addition, the validity of using significant differences in test subscales as a basis for concluding treatment efficacy when the mean total test change scores between groups were identical is questionable at best.

Patterson, Firth and Gardner (1984) used a tabular analysis of clinical improvement in 186 case studies of patients who had undergone inpatient and outpatient detoxification and rehabilitation from various substances in a longitudinal study of CES efficacy. The case studies varied greatly as to the current parameters used, with frequencies ranging between 1 to 2000 Hz and intensities between 1.5 to 3.0 mA. Some subjects received CES with a sinusoidal wave pattern and some received a rectangular wave pattern. Patients were treated

continually (24 hours) for the first six days and then for progressive shorter periods for treatment days seven through ten. There were no placebo or normal treatment controls involved in the study. Outcome measures were the patients' subjective reports of well-being, substance craving and anxiety. Also, some case studies used behavioral observations. A self-report questionnaire regarding freedom from drug use was mailed to the subjects after discharge and was used as a follow-up instrument. The authors state the data showed that 98.4% of the subjects showed a marked sense of well being, reduction of drug craving, and reduced anxiety. Further, of the 50% who responded to the follow-up 78.5% remained drug free for one to eight years. While these claims are interesting, the lack of methodological rigor, variation in the independent variables and the use of only subjective dependent measures make the authors' interpretation of the data less than conclusive.

Schmitt, Capo, Frazier, and Boren (1984) recruited 60 in-patient poly-substance users to participate in a study designed to investigate CES as treatment of cognitive brain dysfunction among the chemically dependent. The CES used was of a sinusoidal wave pattern, with a frequency of 100 Hz and an intensity ranging between 0.10 and 1.0 mA. Electrodes were placed at the maxillo-occipital juncture. Twenty subjects were randomly assigned to one of three groups: An active CES group, a sham CES group or a normal treatment group. For outcome measures three subscales (digit-span, digit symbol, and object assembly) of the WAIS (Wechsler, 1958) that are clinical indicators of organic brain syndrome (Schmitt et al., 1984) were used. Schmitt et al. (1984) state, "A final goal was to use a new device that permitted a completely double-blind study the first in the CES-chemical dependency field.". . . ."a double blind device was connected between the CES machine and

the patient, and four treatment settings. Three of the four settings passed current, and one did not. The current was delivered via ear electrodes placed just behind the ear lobe at the maxillo-occipital junction." (p. 61). The results showed that the active CES subjects significantly improved on the pre-post treatment measures of cognitive dysfunction, while none of the sham or normal treatment control subjects did. Unfortunately the number of subjects in the study is rather small with over 70% of the sham group falling prey to attrition, leaving only six subjects in the group at the end of the study. The attrition rate among the active CES group and the normal treatment controls was 13% and 15% respectively. It is not mentioned if this differential attrition was statistically adjusted in the final analysis of the data. Percentage of improvement was presented in tabular form without any description of statistical means and standard deviations. Therefore, this study does not lend itself to further investigation by meta-analysis or any other forms of statistical analysis based on means and/or standard deviations.

Schmitt, Capo, and Boyd (1986) studied the effectiveness of CES as an intervention for anxiety among chronic alcoholics and poly-substance users. Sixty patients from a CD rehabilitation volunteered as subjects for the study. Sixty percent were chronic alcoholics and 40% were multiple drug users. The CES device produces a sinusoidal wave pattern with a frequency of 100 Hz and an intensity of 0.1 of 1.0 mA. The electrodes were placed behind the ear at the maxillo-occipital junction. A blinding device identical to the one described in the above study (Schmitt et al., 1984) was used to insure the double blind integrity of the study. The dependent measures were various validated self-report anxiety inventories and semi-structured clinical interview scales for anxiety. Subjects were randomly assigned to

three uneven groups. Thirty subjects received active CES, ten received sham CES and twenty were placed in the normal treatment group. Using the Fisher *t* test, the mean pre-post treatment change scores of the active CES subjects were significantly improved on every anxiety measure used. The sham CES group improved only on two (Vigor and Tension) of the six scales of the Profile of Mood States, but on none of the other dependent measures. Normal treatment controls exhibited no significant change between pre-test and post-test scores. This is a well designed study with multi-modal dependent measures. Unfortunately, as in the above research by Schmitt et al. (1984), the percentile ranking graphic representation of the data without a description of means and standard deviation makes further analysis of this investigation's data difficult. Also, the authors do not specify if statistical adjustment were made to compensate for the small number of subjects in the sham CES group or for the dissimilarity of the groups sizes.

Meta-Analysis of CES Research on Chemical Dependence

Prior to beginning the clinical trails a meta-analysis (O'Connor, Bianco, & Nicholson, 1990) was performed to investigate the literature on the efficacy of cranial electrotherapy stimulation (CES) for the reduction of primary and secondary withdrawal symptoms among various chemically dependent populations. Meta-analysis was done to determine if further study in this area was warranted. Therefore, the meta-analysis was a precursor to the current study.

Previous evaluations of CES were narrative literature reviews. A perusal of these reviews suggests that the published studies have been methodologically flawed to the point

that conclusions regarding CES efficacy are unwarranted. The PI felt that a meta-analysis of the CES research would provide quantifiable evidence as to the viability of CES as an intervention in chemical dependency. Meta-analysis is not a new technique first appearing in the literature on medical outcome research in the 1950s. More recently it has been used to test the efficacy of psychotherapies and it is currently popular in some areas of epidemiology. Meta-analysis is a statistical technique used to can study the efficacy of a specific treatment by transforming the findings from individual studies into common metric and integrating the results across the studies. Unlike a traditional narrative review, meta-analysis yields an overall estimate of the magnitude of a treatment effect as well as a test of significance for the treatment effect. Analyses of these data points assess treatment efficacy across all the studies analyzed.

In a meta-analysis, every pertinent variable is coded at the study, treatment, and outcome level. These variables are then used to calculate the average effect size of the studies under investigation (see Appendix A). The effect size used Cohen's d (1977) defined as the difference between the experimental and control group means divided by the pooled SDs of the treatment and control groups. The effect size of each study is then averaged to find the mean effect size of the treatment across all studies. In addition, the relationships between study characteristics and effect size can be assessed empirically. The establishment of an effect size for a particular intervention permits the determination of the proper number of subjects for future clinical studies through power analysis. This, in turn, helps to reduce the possibility of a Type II statistical error.

Initially, we reviewed over 180 studies reporting on CES between the years of 1964

and 1987. Many of these studies were descriptive in nature, lacked control groups, and did not include the means and SDs of the outcome measures used. Despite an extensive literature search, we found only five studies relating to primary withdrawal symptoms and eight studies integrating the secondary withdrawal symptoms of distress and dysphoria found among most substance abusers and the cognitive dysfunction prevalent among post-withdrawal alcoholics and poly-substance abusers (Woody et. al, 1986). Of these thirteen studies only eight (Gomez & Mikhial, 1978; Smith & Day, 1977; Schmitt et al., 1984; Schmitt et al., 1986; Smith & O'Niell, 1975; Smith, 1982; Snodgrass, 1977; Tomovic & Edwards, 1973) could be included in the meta-analysis because the data in the other five studies (McKenzie & Buck, 1976; Patterson et al., 1976; Patterson et al., 1984; Smith et al., 1979; Wen & Cheng, 1973) were presented in a way that did not permit calculation of means and standard deviations. These eight studies included one of five studies of primary withdrawal symptoms and seven of eight of strictly mood disturbance and cognitive dysfunction. This paucity of information makes it quite difficult to determine the efficacy of any treatment. All of the viable studies employed the same device (Neuro Systems, Inc. Model 101, or its portable equivalent the RelaxPak), probably because it was one of the few CES devices available by prescription for the treatment of anxiety, depression, and insomnia. Table 1 presents background characteristics for the eight studies included in this review.(see Appendix A).

Table 1

The Background Characteristics Investigated in Meta-analysis

Characteristics	Mean	Range
No. of patients at pretreatment	78.2	28-22
No. of female patients	4.4	0-20
No. of male patients	73.9	28-227
Patients age (years)	40.5	40-45
Duration of symptoms (years)	12.2	5-26
Session duration (minutes)	36.3	30-40
No. of sessions of treatment	10.3	1-15
No. of weeks of treatment	2.8	1-5

The data as a whole show that some studies had quite large effect sizes in comparison to other studies. The largest effect sizes pertained to the primary withdrawal symptoms of drug use, drug craving, and anxiety specifically among methadone users, a most difficult opiate from which to withdraw. In addition the results showed effect sizes beyond that of a placebo effect in several studies relating to anxiety as a secondary withdrawal symptom. However, some studies that considered anxiety as a secondary withdrawal symptom were well below the placebo effect level. Among the studies relating to cognitive dysfunction in post-withdrawal alcoholics and poly-substance abusers the effect sizes were mixed and highly divergent. It should be noted that the results regarding the cognitive dysfunction groups related to the least severely impaired subjects. This is because there tended to be differential attrition among the most severely impaired alcoholics and poly-substance abusers in these studies. Those studies that had the smallest effect sizes were those that pertained to affective discomfort among post-withdrawal alcoholics and those concerned with the relief of depression as a secondary withdrawal symptom.

Table 2 presents the efficacy of CES as a treatment for primary and secondary

withdrawal symptoms among the between group studies. The analysis displayed an average effect size of 0.940 SD units when comparing CES plus a standard treatment to a CES sham plus a standard treatment, and an effect size of 1.68 when comparing CES plus a standard treatment to standard treatments alone. Individual study effect sizes between groups ranged from 0.11 to 3.50. The average between group effect sizes were not statistically significant. This was primarily due to the low statistical power created by the small number of studies (n=six) and the very large standard error of the mean apparent in this data set. The effect sizes derived from the meta-analysis could be important in determining the effectiveness of the clinical effects of CES as it is applied in the current research when compared to previous research.

Table 2

The Effects of CES Based on the Comparison of Different Control Groups

Types of Control	No. of Studies	Mean Effect Size	SD
Sham CES + Standard Treatment	6	.940	1.28
Standard Treatment Alone	2	1.681	2.99

Note. Effect sizes refer to standard deviation units. Neither effect differed reliably from zero.

The average effect sizes as presented in Table 3 for within groups studies were 0.534 SD units for CES treatments ($p < 0.10$), 0.391 SD units for CES sham treatments plus the standard treatment ($p < 0.05$) and, 0.171 SD units for the standard treatment alone. The range of effect sizes for the within group studies were between 0.25 and 0.83 SD units. The statistical significance of the within group analysis is quite impressive considering the low statistical power produced by having only four studies available to the within group analysis. However, it should be noted that the statistical significance of these findings is, to a large

degree, due to a very small standard error of the mean in relation to the small number of studies analyzed. The within groups analysis was used when there were no controls used in a study, but data were available to calculate means and SD of outcome measures. Within group analysis can also be used when the major outcome measure involved improvement over time.

Table 3

Change from Pretreatment to Posttreatment for Treatment and Control Groups in the Meta-Analysis

Group	No. of Studies	Mean Effect Size	SD
CES+ Standard Treatment	4	.534**	.294
Sham CES+ Standard Treatment	4	.391**	.286
Standard Treatment	2	.171	.242

Note Effect sizes are in standard deviation units. Asterisk indicates that the effect size differed from zero, **$p<.05$.

To put these figures presented above in perspective one should note that the two major meta-analysis research groups in the area of social science outcome research (Glass, McGaw, & Smith, 1980; and Shapiro & Shapiro 1982) state the average effect size of all psychotherapies are between 0.70 and 0.80 SD units when compared to no treatment. This means that roughly 75% of the patients who receive psychotherapy improve in their condition relative to controls who receive no therapy. The average effect size for non-specific factors or placebo effects among psychotherapies as compared to wait-list controls is about 0.40 SD units.

On the basis of eight studies employing questionable methodology, we cannot infer that CES is efficacious for primary and secondary withdrawal symptom. However, there was

some degree of statistical significance in the within group analysis and a trend toward statistical significance in the between group analysis. Possibly more important from a therapeutic perspective, there may be a cluster of CES efficacy regarding the primary withdrawal symptoms for opiate users and possibly secondary symptoms.

Almost half the available 13 studies in the literature were not incorporated into this meta-analysis for technical reasons, some of which are worthy of mention. Studies done by Drs. Patterson (1976; 1984) and Wen and Cheng (1973) and their colleagues constitute the initial work in the area of ELF electro-stimulation as treatment for substance withdrawal. These studies of Wen and Patterson displayed very impressive results based on anecdotal and clinical case study results of the patient's self-reported withdrawal discomfort and drug craving. In the initial study done by Dr. Wen, 39 out of 40 patients reported little or no withdrawal discomfort or drug craving following CES treatment. The two studies by Dr. Patterson relates a success rate for her treatment termed neuro-electric therapy (NET) of over 90% in the case studies she reports. Unfortunately, as is true with many initial clinical studies in new treatment areas, the methodology of the Wen and Cheng and Patterson's clinical studies leaves much to be desired. There were no control groups involved in the studies and the data used for the conclusion of efficacy came from only case studies. In these case studies, the outcome measures were either presented dichotomously (i. e. based on whether the patient improved or did not improve), or are in graded ranks of improvement. Thus, none of their studies were applicable to meta-analysis or any other type of quantification other than percent of patients improved based on self-report. This type of research is reminiscent of the initial cases studies done in psychotherapy research and pharmacology, both of which have

vastly improved over time from a methodological standpoint.

From the above studies, flawed though some may be, it can be seen that one potential therapeutic benefit of CES treatment is that it may be used to induce the addicted patient into therapy with the promise of minimal withdrawal discomfort. In addition, CES could eliminate the use of some substitute psychoactive drugs during the detoxification phase of therapy. From both a psychological and medical perspective CES deserves to be thoroughly investigated, particularly given the current crisis of drug abuse that many cultures in the world face today.

Given the effect sizes demonstrated by our analysis, particularly when we consider the low statistical power involved and the poor methodology, we feel that further investigation into the efficacy of CES as a means to relieve both primary and secondary withdrawal symptoms is warranted. Any further studies must provide tighter methodological controls. We also recommend that all studies in the future be double-blind and that the data be presented with the means and SD of treatment outcomes, as well as all pertinent demographics and inferential statistics. This information should be provided so that a comprehensive analysis of this area of research can be accomplished whether or not meta-analysis is chosen as the technique of review.

A summary of average effect sizes in standard deviation units for the various chemical dependency studies investigating the use CES as a treatment modality considered in this study is presented in Table 4.

Table 4

A Summation of Average Effect Sizes (ES) in SD Units of the Meta-Analysis

Within Groups (WIG)

Study	Effect Size
Smith (1982)	.25
* Schmitt et al. (1984)	.31
* Schmitt et al. (1986)	.74
Smith & O'Niell (1975)	.83

Note the mean ES for all WIG= .534; * the nonredundant WIG mean ES=.525

Between Groups (BTWG)

Study	Effect Size
Snodgrass (1977)	.11
Smith (1982)	.43
Smith & O'Niell (1975)	.43
Tomsovic & Edwards (1973)	.45
Smith & Day (1977)	.91
Gomez & Mikhial (1978)	3.50

Note the mean ES for the BTWG=.940

Possible Mechanisms of CES

The mechanism of the therapeutic action of CES has not been established conclusively, however, several researchers have presented proposals as to its action, a summary of which can be found in Taylor et al. (1991). It has been shown clearly that CES electrical current passes both transcranially and over the surface of the cranium (Taylor et al., 1991). It is possible that this current then affects various areas of the brain and peripheral nervous system. This electrical stimulus could affect various CNS and/or endocrine mechanisms. For example, it has been shown there are descending inhibitory pathways from

the medial brainstem to dorsal horn of the spinal cord which may involve opiate and non-opiate pathways (Mayer et al., 1971). It appears that these pathways can be activated by endorphins and apparently by electric stimulation of certain brain sites such as the periaqueductal gray matter (PAG) (Ng, et al., 1971; Pert et al., 1981; Wickramasekera, 1980). It is also possible that CES could produce effects through cortical inhibition by way of the gate control mechanism (Melzack, 1975). Finally, it is possible that CES may produce effects through parasympathetic autonomic nervous system dominance via stimulation of the vagus nerve (Taylor, 1991). Taylor et al. (1991) postulates that CES may affect the cranial nerves especially the trigeminal (5th) cranial nerve whose primary sensory fibers are in the midbrain, specifically in the trigeminal nucleus. Stimulation of this nucleus in animal studies has been shown to produce electrocortical activity (Fields et. al, 1975). Substance P and enkephalin have been found in neurons within the trigeminal nucleus which are implicated in the function of the limbic region of the midbrain regarding its role in emotions (Taylor, 1991). In addition, they are found in the PAG which is important in the control of pain and perhaps important in learned fear and anxiety (Thompson, 1985) . Therefore, stimulation of the trigeminal nerve and other cranial nerves via the placement of the CES electrodes at the maxillo-occipital junction could easily stimulate the trigeminal nerve or other cranial nerve pathways to the PAG and in turn through the trigeminal nucleus and/or the PAG, affecting the limbic system and/or PAG with their respective emotional and pain sensitivity functions. This mechanism might account for CES's affect on psychological mood during the early treatment phase of chemical dependency as shown in the current CES study.

Summary and Statement of the Problems Involved with Previous CES Research and Purpose and Hypotheses of This Study

The purpose of this study is to research the effects of cranial electrotherapy stimulation (CES) for the improvement of secondary (psychological) withdrawal symptoms among chemically dependents clients. The study was done from a field study perspective in an actual clinical setting. The study intended to offer improvements in methodology and design over previous studies done in this area and to provide results that are highly generalizable to community chemical dependence rehabilitation. The investigation uses patients served by the Oklahoma Department of Human Services in the clinical settings sanctioned by that organization.

The above CES literature review suggests that whereas Western researchers have attempted to improve the problematic methodology of the earlier Eastern Bloc research, it seems the majority of these studies have not lived up to this intention. This is particularly true regarding controlling for placebo effect. Many of the Eastern Bloc studies have been obscured by lack of normal treatment controls (Fenighner, Brown, & Oliver, 1973), almost exclusive use of subjective dependent measures of questionable reliability or validity (Jemelka, 1975), questionable double blind procedures and poor placebo controls and sham treatment procedures. Further, these appear to be extreme variations in electrical current parameters, treatment protocols and CES devices (Brown, 1975).

For future clinical research of CES to be both valid and replicable the methodological structure of investigation should include the following: 1) standardized devices using the same wave pattern, frequency, intensity of current electrode placement and polarity 2) standard

treatment protocols regarding number and duration of treatment sessions; 3) multi-modal outcome measures of acceptable reliability and validity; 4) valid blinding procedures; 5) no-treatment and/or normal treatment controls to help establish baselines of treatment and placebo effects; 6) identical active and sham treatment procedures; 7) and equivalent sensation of treatment stimuli between active and sham groups.

With the exception of insomnia research (Reite et al., in print) most of the Western researchers gave up on CES research around the mid 1980s. Presumably, this was due to the questionable efficacy of CES stemming from mixed results of the research as is exemplified in the foregoing literature review. It is this study's contention that the mixed result of the CES research is not necessarily due to the inadequacy of CES treatments. Instead it might stem from the poor design and methodology of the previous studies and/or the strong additive moderating variable of placebo effect operating within CES treatments.

The hypotheses that will be tested in the current study are as follows:

1. H_0: There will be no statistically significant difference between the groups of active CES treatment groups, the CES shame group, and the control group regarding the amelioration of anxiety and depression in a chemically dependent population going through the detoxification and initial phases of substance abuse treatment and recovery.
2. H_1: There will be a statistically significant difference between the active CES treatment group and the CES shame group, as well as the CES treatment group and the control group. This difference will demonstrate that CES is a viable treatment for anxiety and depression in a chemically dependent population going through the

detoxification and initial phases of substance abuse treatment and recovery.

3. H_2: There will be a statistically significant difference between the sham CES group and the control group. The CES sham group will score statistically significantly lower on outcome measures than the no-CES treatment control group, but greater than the CES treatment group. Thus, the CES sham group will show a greater improvement of anxiety and depression over the CES treatment period than that of the control group. This difference will demonstrate that CES has a considerable incidental placebo effect as a concomitant to the active CES treatment effect for anxiety and depression in a chemically dependent population going through the detoxification and initial phases of substance abuse treatment and recovery.

Because of possibility that placebo effect may be a significant force in CES treatments, it seems appropriate to briefly review the literature relating to placebos. The following chapter will review the definitions, methodological procedures, and possible mechanisms involved in manifestation of placebo effects.

CHAPTER III

PLACEBO AND PLACEBO EFFECT

Introduction

The term placebo is derived from the Latin meaning "I shall please." One of the major contributors to placebo research in recent years Shapiro (1960) states that the term placebo has been widely used to refer to the clinical effects of procedures which offer solely reassurance or the expectation of help to the patient. These procedures which are considered biomedically inert by Western medical science may, lead to improvement in the patient's clinical condition. There is currently no universal definition that is generally accepted within the placebo research and clinical communities. Further, the boundaries of the various definition for placebo phenomena are not well established. The terms placebo and placebo effect vary greatly in meaning depending upon the discipline using the term (i. e., psychologists, anthropologists, pharmacologists, physicians, etc.), and upon the definition that each researcher or practitioner chooses to use. Lacking a rigorous and accepted definition of placebo and placebo effect, the description and interpretation of the placebo phenomena remains ambiguous.

This chapter identifies the major issues relating to placebo phenomena, and reviews some of the literature regarding definitions, research methodology, and theory pertaining to this subject. As the literature is extensive, this chapter's exposition of the subject will cover only the essentials.

Historical Perspective

Current as well as ancient health care providers have confronted the problem of explaining why one patient responds positively or negatively to a treatment while another may not respond in any fashion.

An early explanation of what is today called placebo effect dates back to classic Greeks and the healing rites of priests at the oracle of Delphi who thought of human imagination as a source of healing power (Lain Entralago, 1970). The Greeks might have argued that the although the health care provider may make a difference in disease outcome, the real source of curative power lies in the positive emotions prompted by the power of the mind within the afflicted individual and not necessarily within the active elements of the intervention.

Another explanation of placebo discussed by Neuberger (1932), dates from the late Renaissance and is based on the idea of *vis medicatrix naturae* or the healing power of nature. This explanation calls into account the organism's innate and powerful recuperative powers, and the organism's ability to restore homeostasis in the face of illness. From this perspective it might be argued whether any cure is due to the intervention of a health care provider.

Late in the 18th century Franklin, in his study of animal magnetism, utilized an elegant single-blind control design to show that the emotional state of the patient and not the physiological effect of the treatment caused the observed behavioral and physiological changes in the patient (Brody, 1980).

Most medical dictionary definitions around the late 18th century emphasized the

tension between the imaginative as opposed to the *vis medicatrix naturae* explanation of placebo effect (Brody, 1980). By the early to the middle 19th century theoretical emphasis began to favor the *vis medicatrix naturae*, defining placebo as a remedy given to please the patient without any belief that it possessed any curative power (Shapiro, 1968)

By the middle 20th century, reinforced by evidence from psychosomatic research, the theoretical pendulum began to swing back to imagination as the central mechanism of placebo effect. Finally with the beginning of a demand for double blind studies, particularly in the area of pharmacology, the modern era of placebo research began. Given the proposed effects of endorphins, catecholamines, and particularly neuropeptides there are some possible explanations of psychosomatic phenomena offered by the relationship between emotional states and physiological reactions (Brody, 1980).

Definition

In what may be seen to be a fairly radical statement, Shapiro and Morris (1978) say that for the most part history of medical care has been the history of placebo. They state that "the placebo effect flourished as the norm of medical treatment" (Shapiro & Morris, 1978, p. 371) even after the advent of contemporary medicine.

Nevertheless, the very use of the term placebo has been called into question by some authors (Berg, 1983; Cahill et al., 1978; O'Leary & Borkoveck, 1978;). Thus, it behooves one to look at some questions relating to the definition of the term placebo.

A major problem with defining placebo and placebo effect lay in which discipline generate the definitions. Often these disciplines approach the subject of healing from

divergent perspectives, and their definitions tend to reflect this disciplinary bias. The attempts to develop and accurate definition of placebo generally fall into one or more of three basic approaches: 1) an attempt at a universal or generic definition; 2) pharmacological and/or medical definitions; 3) psychologically based definitions.

Shapiro and Morris (1978) define placebo in multiphasic fashion as follows:

"A placebo is defined as any therapy or component of therapy that is deliberately used for its nonspecific, psychological or psychophysiological effect, or that is used for its presumed specific effect, but is without specific activity for the condition being treated. . . . A placebo when used as a control in experimental studies, is defined as a substance or procedure that is without specific activity for the condition being evaluated. . . . A placebo effect is defined as the psychological or psychophysiological effect produced by placebo." (p. 371)

Pertaining specifically to the fields of psychology and psychiatry, Jerome Frank (1973) proposes that of the many psychotherapeutic techniques developed over the last century whose efficacy exceeds that to be expected from spontaneous remission owe their ameliorative effects not to distinctive treatment factors, but rather to nonspecific factors common to all functional psychotherapies. In Frank's definition of placebo the term nonspecific becomes almost synonymous with the notion of placebo. He states that a particular psychotherapeutic theory will define what is a considered active or effective procedures. Further, he theorizes for whatever the specific interventions that are defined as active by a particular theory that all these "effective" interventions have in common the ability to engender the patient's *hope* that they will get better and mobilize the patient's *sense of efficacy* to combat the sense of demoralization that is common to all disorders. These qualities of hope engenderment and efficacy to combat demoralization are the nexus of what he calls nonspecific effects. Frank goes on to say that we should not decry such

"placebogenic" gains in psychotherapy until there is something more effective in the treatment of psychological disorders. Critelli and Neumann (1984) attempt to improve on Shapiro and Morris's (1978) and Frank's (1973) exposition of placebo effects as stated above. They concluded that the *"common factor criterion"* was the basis for understanding the efficacy of psychotherapies. Critelli and Neumann believe this to be the most effective definition of placebo at least in a psychological context. They contend that the notion of a generic placebo presented by Shapiro (1968) suffers from a futile attempt to include both a psychological and pharmacological definition of placebo. This *"common factor criterion"* is a very controversial position that has met with more than a little criticism by researchers in the areas of psychotherapy and placebo research because it tends to minimizes the relevance of psychotherapy to the level of lay advice giving, or worse, shamanistic trickery (Parloff, 1984). Further, Critelli and Neumann are criticized for failing to distinguish between incidental and intentional effects of a particular theoretical approach to psychotherapy (Grunbaum, 1985; Parloff, 1984).

Berg (1983) suggests that although the placebo remains a useful idea in the investigation of medicine, psychology and the behavioral sciences, the term has become ambiguous. For the most part, it covers too many dissimilar situations. Grunbaum (1985) agrees with Berg saying that the standard technical vocabulary of Shapiro and Morris (1978) tends to be misleading, obfuscating, and begs for clarification. To help alleviate this problem, Grunbaum (1985) suggests that the definition of placebo be reformulated. By making it theoretically relevant to the framework of the speaker. He goes on to say the term nonspecific causes so often associated with the definition of placebo be replaced with the term

"incidental causes." Grunbaum believes that we should differentiate between the intentional placebo and inadvertent placebo. It follows that when placebo effects are examined from a given theoretical framework they are due to unidentified causes. However, while these unidentified causes are elusive because of ill defined theory and methodologies, they are discernible through the proper scientific study (Grunbaum, 1985).

Brody's (1985) definition of placebo demands a grounding in a historical and clinical context. While agreeing with a need for a theory driven definition of placebo, he is at odds with Grunbaum's more generic definition of placebo. Brody encourages a more particular definition that emphasizes symbolic processes, imagination, and emotions which have long been recognized as aspects of the healing process. He believes that authors such as Grunbaum and Shapiro and Morris (1978) err by excluding such considerations in their respective definitions of placebo. In Brody's definition the central concept is *"the disciplinary matrix."* In this idea the characteristics of the discipline interested in the idea of placebo determines whether symbolic processes are to be recognized as significant or spurious factors in disease. Thus, the disciplines of anthropology, ethnomedicine, holistic medicine, psychology and psychiatry might consider these symbolic processes to be included in the development and amelioration of disease. Brody believes that a systems approach (which are is discussed at length later in this chapter) may provide a more comprehensive disciplinary matrix for contemporary health care providers.

Pharmacology, allopathic medicine, microbiology, and biophysiology may not consider these symbolic processes relevant to the formation or alleviation of the disease process. They may consider them as confounding variables to be removed methodologically from

consideration, if possible. The exclusion of placebo effects in contemporary mainstream medicine stems from the dominance of a biological reductionist matrix that necessarily dismiss symbolic processes (Brody, 1980).

Among several authors, exemplified by Borkovec (1985), there exists a goodly amount of pessimism regarding the establishment of an "agreed-upon" definition for placebo effect in the future, given the divergence of opinion prevalent in the field of placebo study. He agrees with Grunbaum (1985) that theory should be used to determine placebo definitions and procedures. Elements that are predicted by a specific theory to be active in a treatment of a disorder should be incorporated in research while those that are not predicted to be relevant or active should be left unmentioned. Borkovec believes that this open system is more amenable for the specification of what is considered placebo verses active treatment. He also agrees with Grunbaum (1981) in the belief that the term nonspecific should be abandoned. He believes replacing nonspecific with Grunbaum's (1985) term *"incidental"* focuses the scientist on procedural elements and theory-relevant characteristics as opposed to simple unspecified effects. This focus on procedural elements and their engendered behavioral effects would then help to find mechanisms through which these specified effects occur.

Borkovec agrees with Brody (1980) in affirming that the patient's imagination and emotional state are the most salient aspects regarding the concept of placebo. Borkovec believes that the term imagination itself could be a substitute explication of placebo effects. Further, he believes that the concept of imagination lends itself to scientific investigation better than does the current divergent conceptualizations of placebo effects. For the most

part, Borkovec believes that definitions of placebo that emphasize patient centrality and the meaning attached to specific treatments by a patient, as with Brody's definitions, are too phenomenal and symbolic. He fears that this places placebo effects into a category of symbolic effects, forcing the investigator's attention away from specific mechanisms of the placebo effect that ultimately can be used to explain the behavioral consequences that the placebo might engender.

It seems the task of developing a adequate definition of placebo and placebo effects remains to be accomplished. "The task of defining what we do not know will always fail." (p. 62 Borkovec, 1985). In the absence of an integrated placebo theory incorporating the salient features extracted from various disciplines, trying to fully understand placebo phenomena and developing a specific definition seems futile. For the purposes of this research, the principal investigator (PI) offers the following as a operational definition for placebo. A placebo can be defined only in theoretically relative terms, and as such is a theory specific inert treatment for a specific disorder. A possible definition for placebo effect may be the effect of a procedure or procedures which have a ability to mobilize an organism's self-recuperative powers, whose mechanism is not fully understood by theoretical perspective, or the active interventions defined by the theoretical perspective held by the investigator or practitioner observing such effects at the time of the said observation.

Methodology Relating to Placebo Effects

If one accepts Shapiro's (1960) proposition that the history of medicine is inextricably linked to the placebo effect, then it follows logically that in a treatment outcome design one

should build in some means to control for placebo effect. Traditionally, the best answer to this problem has been the drug verses placebo blinded study (Ross et al. 1962). Ross et al. (1962) believe that to differentiate the true potency of the active medication or *"drug response"* from the additive effect of the intervention plus placebo effect or *"drug effect"* one must incorporate into the design methodology a *"no treatment"* control and a *"disguised drug"* group. They argue that there is a false dichotomy between drug and placebo groups, inherent in the two-group comparison methodology, because the effects obtained by the drug group may be partially due to psychological effects. Further, they argue that uncritical endorsement of the two-group methodology by most pharmacological and chemotherapy investigators has led to a neglect of the empirical examination of psychological influences on biological processes. Ross and Bacilli (1985) using the four-group design say that they have shown drug effects are altered by instructions. They state further, that when the intervention is given surreptitiously typical clinical effects are modified. Thus, there is a significant interaction between the context and instruction variables and treatment effects that occur in a clinical setting. Although the idea of "disguised treatments" and triple-blind designs are theoretically appealing, there are ethical and practical problems with such designs. Only withdrawal studies in which subjects have previously received pharmacological treatments whose effects are to be measured by removing such chemical interventions would be appropriate for such designs (Paul, 1986).

Ross and Bacilli (1983) reviewed the literature and found that variables were sensitive to placebo manipulation in several domains: In the area of physiological variables, blood pressure, heart rate, and pain were affected. In psychomotor capability, grip strength,

reaction time, and to a lesser degree finger tapping. From a cognitive perspective, short term memory was affected while more complex functions remained unaltered. On an affective level, general mood level appeared less reliably affected, while self-perception of relaxation and activation was amenable to placebo manipulation. These conclusions must be judged in light of the fact that they lack replication in similar studies. According to Ross and Bacilli (1985) this nonreplication is primarily due to the fact that the list of variables they considered is comprised of dependent variables, while the listings of most other studies that do not agree with their findings are composed of categorical disorders.

It is important to assess the effects of placebo procedures for different types of research. Wilkins (1985) looks at placebo methodology in the domains of chemotherapy, placebo, and psychotherapy research. He proposes that within each of these domains placebo designs have been developed for exhibiting the efficacy of therapy. Wilkins believes this helps to determine whether an intervention causes improvement, as well as isolating the mechanisms through which placebo procedures affect outcomes. This distinction requires a shift in emphasis from a search for a credible correlation between independent and dependent measures, to a search for intervening variables that are inferred from scrutinized phenomena. In chemotherapy research psychological factors are often simply considered a confounding variable contributing to a placebo effect. There mediational effects are to be eliminated the from the outcome measures after they have been noted. On the other hand, psychological factors are the main concern of psychotherapy research. Psychotherapy research that emulates chemotherapy research becomes flawed to the extent that it relies on inactive control mechanisms and a concomitant hasty focus on psychological mechanisms (Wilkins, 1983).

1983). Placebo procedures using "inactive" treatment component are often inappropriate for psychotherapy research. This is because the placebo treatment and the active treatments are often not equivalent in credibility, and in their ability to create confidence in treatment and the expectancy of healing (Borkovec & Nau, 1972; Lick & Bootzin, 1975). Further they do not control as well for observer bias as they do in chemotherapy research (Wilkins, 1985).

In research where there is the possibility of a placebo effect acting as moderating and/or confounding variables it is imperative that the investigators use an experimental design sensitive to detecting placebo effects. These designs should differentiate various placebo occurrences from the salient psychological and/or medical interventions involved in the treatment under study. To this end Paul (1969) suggests that all researchers investigating treatments in which placebo effects may be present keep in mind the following *"ultimate question"*. "What treatment, by whom, is most effective for this individual with that specific problem under which set of circumstances and how does it come about?" (Paul, 1969, p.44). To answer the *"ultimate question"* Paul (1967) circumscribed particular domains of variables that should be considered in any therapy outcome research. The domains to be considered are: client, staff, and time variables. Paul (1986) elucidates these domains and the classes of variables that may be considered within each as follows:

The Client Domain:

1. Problem behaviors---those aspects of the client's physical, psychological, motor, affective, or cognitive. functioning that are disturbing to the client or others, due to excesses, deficits, or inappropriateness, at which the treatment is directed.

2. Stable personal-social characteristics---those attributes other than problem behaviors that may differentiate clients, define role behavior and/or interact

with responsiveness to treatment. These may be demographic, diagnostic categorization, personality traits, historical, physical status, motivational, or expectancies related to the treatment under consideration.

3. Physical-social ecology---settings and events external to the interventions that provide life experiences determining the timing or place of manifestation for problem behaviors, or that interact with the outcome of the intervention. These may be economic or social resources, personal associations, work or educational situations.

The Staff Domain:

1. Therapeutic techniques---The specific interventions that may contribute to the improvement of the client's problem behaviors. These may be somatic treatments, and intended or unintended psychosocial procedures defined by the nature, frequency, content, and timing of verbal and nonverbal acts delivered by staff to clients.

2. Stable personal-social characteristics---those attributes other than those specific to treatment being investigated which may differ between staff members that can interact with the effectiveness of specific intervention techniques under study. The attributes should be specified for given classes of clients, problem behaviors, and environments such as demographics, personality traits, physical status, experience, prestige, theoretical orientation, confidence, attitudes, and opinions regarding conditions of the intervention under study.

3. Physical-social treatment ecology--those characteristics of the setting where the treatment under study takes place that may interact with other classes variables. These variables may include: size, age, location, reputation, sumptuous versus shabbiness of the setting, staffing levels, public versus private, and institutional versus *in vivo*.

Timing Domain:

1. Timing circumstances--those variables that specify the set of circumstances for assessing other classes of variables and that determine the focus and nature of measurement needed within and between treatment periods. These include both the moment in time or *"window of time"* at which information should be obtained.

Experimental designs should incorporate as many criteria mentioned above as is

possible within the constraints of practicality. This would greatly benefit the development of theories leading to the understanding of the mechanisms behind the placebo effect. Further, Paul's research perspective which explores a multidimensional "ultimate question" goes a long way to meet what some have suggested is the primary objective in the healing professions, the individualization of treatment (Pelligrino & Thomasma, 1981). Studies should be designed to answer specific aspects of the "ultimate question." In field research each condition should be constructed to control for specific aspects of treatment and presenting problems to whatever degree is possible in the "real world" of clinical trails.

Due to the complex and interactive nature of variables inherent in many psychosocial and pharmacological studies, the only designs capable of displaying cause and effect attributable to a specific treatment or treatment aspect are factorial or partial factorial group designs (Paul, 1986). These designs should include a treatment group, no treatment and nonspecific treatment controls (Rosenthal, 1985). The nonspecific control group could be a placebo, attention placebo, pseudo-treatment, component treatment, or alternative treatment. They might also be a combination of the preceding, depending on the nature of the problems being investigated, the treatment being evaluated and the logistics of the study (Paul, 1986). Paul (1969) strongly suggests the use of a no treatment group when placebo effects are suspected to play a role in experimentation. This is necessary because most estimates of placebo effects are derived from retrospective analysis of drug versus placebo comparison. This retrograde procedure is wholly inadequate because it lacks controls for spontaneous change in the natural course of a disorder which may be autonomous to the manipulation of the placebo. At times in clinical studies it is not possible to use a no treatment control due

to ethical or practical considerations. When this is the case a normal treatment control group could be used to determine a baseline against which placebo effects for a new intervention can be compared. Without a no treatment and/or normal treatment control groups, the true extent of the placebo effect may be overestimated or underestimated, because of the unique qualities of various placebo manipulations. This is true because the experimental manipulation of the placebo may not cause the changes observed. Also, estimates of the placebo effect can be biased for various reasons (Paul, 1986). Under these circumstances the researcher would be unable to isolate confounds related to outcomes without the baseline provided by the no treatment and/or normal treatment control.

Theories About Placebo Mechanisms

Theoretical mechanisms relating to placebo phenomena cross disciplinary lines with respect to understanding placebos and placebo effects. This section will present examples from various disciplines. In general, these disciplines explain placebo phenomena from four psychological theoretical positions, with some crossover evident. These general theoretical positions are: psychosocial/symbolic, cognitive/behavioral, classical conditioning, and psychobiological. Finally, a synthesized model (White et al., 1985) will be presented integrating the above categories. This final approach, seems to be a comprehensive and functional approach to the understanding of placebo phenomena.

Hahn (1985) and Kleinman (1980) are examples of an "ethnomedical" perspective using a psychosocial/anthropological paradigm to understand placebos and placebo effect. This psychosocial paradigm of healing incorporates biomedical knowledge into a broader,

interdisciplinary concept giving credence to the sociocultural variables involved in disease formation and mediation, including the role of placebo phenomenon (Hahn, 1985). Ethnomedicne includes ideas about systems of symbols, beliefs, sentiments, and rules and standards for interpersonal interaction relating to health conditions. Included in these rules of societal and interpersonal interaction are the effects of the "sick role" (Parsons, 1951) and "illness behavior" (Mechanic, 1968). There are also values that affect collateral relationships and relationships between the health care providers and the sick individual. The effects of moral and ethical ideas related to these systems, as well as theories about the production and mediation of disease and healing also are included in the ethnomedical perspective (Hahn 1985).

Hahn (1985) and Hahn and Kleinman (1983) emphasize the role of culturally generated belief systems and the effects of these beliefs on illness, health and healing processes. Perhaps, the most spectacular illustration of his position is the phenomenon of the "nocebo" Hahn (1985) cites (Kissel & Barrrucandor, 1964) which is the physically harmful effect of a negative belief as exemplified by voodoo inspired death. The first scientifically observed and documented case of voodoo death was in Australian aboriginal society during the 1950s (Godwin, 1976). Several highly respected researchers among them Cannon (1942), Cohen (1988), Engle (1968), and Hahn and Kleinman (1983), have approached this subject attempting to understand the mechanisms involved. These researchers come to the same conclusion: individuals react to culturally generated negative symbolic events and certain societal defined personal interactions. If dictated by societal values, these symbolic events and interactions will cause the targeted individual to suffer grave illness and even death. Less

dramatic, but more important then the phenomenon of voodoo death are the finding of researchers regarding the social matrix as an aspect of pathogenesis and healing. Meyer and Haggerty (1962) found that streptococcal and other respiratory aliments were four times more likely to occur in individuals having experienced some acute family and/or interpersonal stress, than among individuals not experiencing such stressors.

As stated, culturally generated belief systems, expectations, and states of thought can be pathogenic (i.e. nocebos), but they can also be curative (i.e. placebos). In a treatise about miracle cures and spontaneous remissions, Cousins (1983) suggests that the hope engendered by belief systems generated from supportive, interpersonal networks, institutions, or the general culture, may explain such spectacular and inexplicable cures. Beecher (1955) says that placebo effects account for approximately 30% of all healing attributed to allopathic medical interventions. To explain the efficacy of these ethnomedical aspects of healing, several investigators (Brody, 1973; Chopra, 1990; 1991; Cousins, 1989; Engle, 1960; Hahn 1985; Hahn and Kleinman, 1983) have turned away from the Cartesian perspective of mind-body dualism and moved toward the idea of mind-body unity. This principle suggests that psychological states having a cultural or societal basis directly cause biological manifestations of both a positive and negative nature, due to the interaction of psychological and physiological mechanisms within the organism.

An explanation of placebo effects, medication efficacy and medical compliance from a psychosocial/symbolic/psychodynamic paradigm is seen in the work of Hauser (1986). Using Winnicott's (1951) psychodynamic model of object-relations theory, Hauser (1986) explains the effects of medication and medical compliance. The author suggests that

medications are a transitional object in Winnicott's scheme of things. Placebos or active treatments become a self-object interface giving them the potential to mimic the original nutrient dyad of the child and primary care giver. The efficaciousness of placebos, active treatments, and compliance with prescriptions are examined by Hauser (1986). It is proposed that the health care provider assumes a transference role relating to the original care giver. This transference is examined in respect to the benefits and vicissitudes of the original internalized object-relations. Dunn (1986) agrees with Hauser (1986) that the Winnicott model is interesting and bears further research. This is especially true in that medical noncompliance and the placebo effect may be understood as being related to a "split" in idealized transference by the client toward the health care provider.

From a psychosocial/symbolic/psychodynamic paradigm with an anthropological twist, Adler and Hammet (1973) hypothesize that placebo effect is derived from the universal need of humans for a group membership. They say this need leads to symbolic projection of a system of healing that helps the dysfunctional member of the group to become a more functional group member. Alder and Hammet say there are common or nonspecific factors relevant to any therapeutic process that follows a sequence of crisis, conversion to the implied dictates of a group, and possibly leading to the formation of cults. These cults may believe in healing practices that are placebo based. This could account for the prodigious number of faith healings among certain cult groups.

Bootzin (1985) approaches comprehension of placebo and placebo effect using a cognitive/behavioral psychological paradigm that emphasizes expectations regarding the treatment received. Bootzin sees that cognitive psychological theory offers placebo

theoreticians the ability to explain symbolically processed information and nonconsciously or covertly learned material. The classic conditioning theoretical placebo models that will be discussed later do not fully offer an explanation of these symbolic and nonconsciously learned phenomena regarding placebo effects (Bootzin, 1985; Reiss, 1980). The role that modelling, imagery, language and information processing play in mediating behavior leads to the hypothesis that emotional reactions and defensive behavior are primarily or at least partially cognitive mediated. This contrasts with the classical conditioning model in which images, symbols, language are covert stimuli that have acquired their capacity to invoke an affective response through previous exposure to conditioning via external stimuli (Wolpe, 1978). To exemplify the contrast in viewpoint Bootzin and Max (1980) say that people can verbally induce arousal, depression and elation. Further, if symbolic stimuli (CSs) are considered covertly conditioned stimuli then repetitive practice of such stimuli without a presentation of the associated unconditioned response should lead to the extinction of these affective reactions. Bootzin and Max (1980) say this is not the case, supporting the idea that emotional response is at least in part cognitively mediated rather than purely a classically conditioned response.

Reiss (1980) suggests that expectancy is derived at least in part from a cognitive model. He states that cognitive learning and modelling can alter expectancies. He identifies two types of expectancies: anxiety and danger. Danger is the expectancy of physical damage or social rejection, while anxiety is in essence anticipatory fear. Applying this model to various phobias, would require interventions using different types of expectancies. Placebo effects regarding various treatments for anxiety phobia should be considered in Reiss's

opinion.

Bandura (1977) identifies two basic types of expectation: 1) outcome expectancies, or the belief that a given behavior leads to specific outcomes; 2) efficacy expectancies, or the belief that an individual can be successful in the execution of behaviors required for some desired result. Although there are similarities between Bandura's efficacy expectancy and Riess's anxiety expectancy, Bandura offers a theory applicable to a wider range of disorder, than does Riess's theory. Reiss's theory is limited to interventions for phobias.

Kirsch et al. (1983) found that a highly credible expectancy attention placebo manipulation was equal to systematic desensitization in efficacy in the treatment of phobias. In a similar vain, Bernstein and Kleinknecht (1982) found that among dental surgery patients a highly credible attention placebo was as effective as five other treatment manipulations. They concluded that all treatments that show therapeutic improvement share two expectancy elements. These are the expectancy of decreased anticipatory anxiety and decreased stress reactivity. Gyrll and Kathan (1978) had similar results among dental patients. They found enthusiastic messages about the effectiveness of a placebo regarding pain reduction produced a statistically significant reduction fear related to pain and much lower ratings of pain by the subject. Bandura's (1977) efficacy expectations are derived from four types of information: 1) feedback from prior experiences; 2) vicarious experiences; 3) verbal persuasion; 4) feedback from the individual's autonomic responses. Among information sources, prior experience is the most salient. Thus, from this theoretical perspective, placebos would have the most powerful effect when they increase the expectancy resulting from successful experiences (Bootzin, 1985).

As mentioned above expectancies are some of the major variables that have been investigated in relation to placebo mechanisms and effects. It appears that most of these expectancies are variations of the expected predictability and control. Thompson (1981) says unpredictable and uncontrollable experiences are very stressful, producing higher levels of cortisol and other stress hormones, than other aversive events over which an individual has more control. "Controllability, " in particular, has shown salience relevant to psychological and physical health (Hull et al, 1987).

An interesting clinical phenomenon is discussed by Evans (1985). In a review of fifteen double-blind studies of the efficacy of placebo analgesia for individuals suffering from a variety of traumatic and postoperative pain, Beecher (1959) showed that 35% of the subjects studied demonstrated significant pain reduction from placebo administration. In a similar review of eleven studies Evans (1974) displayed a 36% reduction of pain in a similar population of subjects with placebo administration. Evans recognized that the above results needed to be reinterpreted because of the fact that analgesic drugs that are normally compared to the placebos do not completely eliminate pain. According to Evans a standard dose of morphine usually will reduce subjective pain only by 50% in 75% percent of individuals tested. He says that a better way to find the analgesic effect of a drug is to use a ratio comparing the pain reduction of an unknown drug to the analgesic effect of a known drug. Generally the drug or placebo of interest is compared to the 50% pain reduction from a standard dose of morphine. From six studies that compare placebos to a standard dose of morphine, Evans (1985) tells us that placebo analgesia is as effective as morphine in 56% of the subjects studied. It is interesting that Thorstiensson et al. (1978) have empirically shown

similar findings, albeit a lower rate of placebo analgesia with CES, using active and sham CES groups. Self-reported analgesia was found in 32% of the sham CES group as compared to a 48% reduction of pain among the active CES group.

Evans tells us that this 56% effectiveness of placebo treatment is not limited to analgesia:

> It is also found in double blind studies of nonpharmacological insomnia treatment techniques (58% from 13 studies) and psychotropic drugs for the treatment of depression such as tricyclics (59% from 93 studies reviewed by Morris & Beck, 1974) and lithium (62% from 13 studies reviewed in Marini, Sheard, Bridges, & Wagner, 1976). Thus, it appears that placebo is about 55-60% as effective as active medications, irrespective of the potency of these medications." (Evans, 1985, p. 223).

Evans (1985) hypothesizes that there are four possible mechanisms responsible for placebo response: 1) suggestion; 2) expectancy; 3) anxiety; and 4) endorphin mediation. As expectation and suggestion have been discussed at some length as mechanisms in placebo effect, the PI will move to a discussion concerning endorphin mediation and anxiety.

Along with Evans, Frank (1982) hypothesizes that miracle cures as well as placebo may have as their mechanism the release of endogenous endorphins. Several researchers have discussed the role of endogenous endorphins in placebo analgesia (Grevert et al. 1983; Levine et al.,1978; Levine et al., 1979). Unfortunately there have been some methodological problems with these studies (Grevert & Golstien, 1985). Also, several of the findings in relation to the endogenous endorphin mechanism of placebo effect were not replicated (Evans, 1985).

Because anxiety is incorporated into this CES study as a secondary aspect of withdrawal, we look with some interest at what has been found concerning the interaction of

placebo effect and anxiety. Placebo effects are often linked to anxiety reduction (Evans, 1974; 1985: 1990). Various other researchers have shown that placebos figure prominently in the reduction of anxiety. For example, Counts et al. (1978) say they found a placebo manipulation and biofeedback treatment to be equally effective in the reduction of anxiety. They go on to say the self-reports of anxiety are subject to placebo effects. Gyrll and Kathan (1978) say that suggestions have a placebo effect that mediate anxiety which in turn reduces pain. Relaxation used as a placebo manipulation was shown to be as effective in reducing anxiety as biofeedback (Grynol & Jamieson, 1975). Grynol and Jamieson (1975) state that because both accurate and inaccurate feedback causes a reduction of anxiety on psychological self-reports, electromyography (EMG) may act as a placebo affecting self-report of anxiety. As mention earlier Passini et al. (1976) believed that active CES was no more effective than a sham CES placebo manipulation, but both were significantly effective in reducing self-reports of anxiety.

From the above it is obvious that researchers using only self-reports of anxiety in experiments incorporating placebo manipulations must guard against incorrect findings related to anxiety reduction. This reduction of self-reported anxiety may be stemming from placebo effects and not from any intended treatment manipulation. The best way to control for such an error would be to use a methodology incorporating active placebo, and no treatment groups into the experimental design. Also, the use of observational and/or clinical measures of anxiety in addition to self-reports, as outcome measures, incorporated into the experimental design would help to attain more accurate findings in such research.

In a response to Evan's proposition that placebos are as effective as active analgesics

in 56% of the population, Tursky (1985) says that this ratio may be an artifact. It is an artifact that stems from the unidimensional way in which pain is usually measured. Also he says pain self-reports are normally categorical, and should be continuous and multidimensional. Tursky (1976) showed that pain responses consist of three major dimensions. These dimensions are: intensity, reactivity, and sensation. Gracely et al. (1976) has replicated the existence of these three dimensions of pain response.

Chopra (1990, 1991) Frank (1973; 1977) and Plotkin (1985) see faith as the central mechanism regulating placebo phenomena. For these investigators faith engenders hope which sets in motion self-healing. The basic assumption of their position is that a placebo will work in reducing pain if an individual has faith in the potency of the cure.

These authors do, however, differ in their beliefs about how faith is developed. Frank (1973) takes a cognitive position seeing faith as the product of the persuasive ability and credibility of the health care provider. Faith acts a mediator to overcome the demoralization and gives the patient a belief in self-mastery which aids in the patient's ability to self-heal. This happens through nonspecific healing forces common to all therapies that are effective. Chopra (1990) takes a biopsychosocial position believing that faith and hope lead to a psychological state of mind that releases a kind of genetic recoding. This process initiates the release of innate recuperative powers locked within an organism's DNA which during the illness process have become dormant. With the proper psychological condition a recoding of the genetic healing mechanisms take place which initiates healing process. The assumptions of Chopra and Frank are to some degree reminiscent of the classical Greek position that the healing power of the organism was released by positive emotion.

Plotkin's (1985) position is one that stands between a phenomenal and a conditioning explanation of placebo effect. For him, the source a personal faith stems from an individual's conditioning history in relation to the meaning the individual attaches to the health care providers, hospitals, and outcomes of previous actions taken when ill. For Plotkin placebos work because faith, based on this history, impels the person to behave in ways that counteract the health problem. This healing behavior becomes the catalyst for "self-healing." For Plotkin the placebo effect is completely psychological as opposed to biological or even biopsychosocial. He emphasizes that there is nothing phenomenal or "unreal" or "insubstantial" about his position. To prove this point he points out that biofeedback, learning and conditioning as psychological occurrences are not unreal or artifactual.

However, Plotkin reasserts the Cartesian position of mind-body dualism, this leaves his "strictly psychological" concept of healing solely in the realm of phenomenology with no connection to the physical realm (Turkkan & Brady, 1985). Plotkin takes the source of control for the development of faith out of the environment and places it within the individual. He describes the placebo effect as *"strictly psychological" and "self-regulating,"* although he explains the source of faith as based in the conditioning history of the patient. This concept seems somewhat circular and contradictory. Personal histories are by nature a product of the sociocultural environment or physical world. Therefore, Plotkin's idea seems a rather untenable philosophical position. Plotkin's concept of healing being is *strictly* psychological or derived solely from attributions derived internally from the conditioning history without any consideration of organicity, seems to be a solely metaphysical explanation of something that has at its end very often a physical result (Turkkan & Brady, 1985). Would this notion

true even if the object of the healing was behavioral given what is known regarding psychophysiology? It is difficult to think of the organic functioning of these conditions without some concomitant physiological or biochemical changes within the organism (autonomic response, neurotransmitter, neuropeptide, hormonal changes etc.). To do so one would have to become very metaphysical about the processes. Frank (1982) also suffers from a problem of philosophical contradiction in terms when he proposes a solely psychological explanation regarding the healing power placebo effects, faith healing, and meditation. He states that to explain the functioning of such phenomena the mystical and scientific "faces" of psychotherapy must begin to parallel one another. It appears, however, that this idea is an oxymoron, for the mystical and scientific natures of any phenomenon are divergent and not parallel by definition. Chopra (1991) overcomes this philosophical problem in his notion of healing by adhering strictly to the philosophical position of mind-body unity and total interaction. For Chopra faith develops a proper healing state of mind which sets into motion genetically coded mechanism's which in turn engender self-healing biochemical processes.

Among the many placebo researchers using a behavioral model to explain the mechanisms that control placebo effect (Cammeron et al, 1976; Jodogne, 1990; Turkkan, 1989; Wickramasekera, 1980), Wickramasekera (1980) presents the most comprehensive theoretical treatise. Wickramasekera makes seventeen predictions regarding placebo effect, using a strict classical conditioning model. He describes the nocebo in classical conditioning terms, saying that conditioned stimuli (CS) can acquire harmful effects relating to health. This is a reasonable explanation for such nocebo effects as sorcery and hex induced illness, and voodoo death. Conditioned nocebo effects have been demonstrated empirically in animal

studies (Harlow et al., 1971; Siegel, 1985). Wickramasekera notes that in a psychobiological study the use of any active ingredients as an unconditioned stimuli (UCS) creates potential for those active ingredients to become agent of classical conditioning which may cause a placebo effect. With these classical conditioning models, the effect of placebo leaves the realm of the phenomenal. These models place placebo in a more systematic empirical light, in which placebo effects can be measured and identified. This allows for placebo effects to become a more evident, expected, and quantifiable component of some intervention under study or in clinical use. Therefore, placebo effects can be more readily adjusted, or used according to accepted precepts and standards.

Wickramasekera (1980) objects to the term nonspecifics regarding the understanding of placebo. He believes that a large body of precise and empirically validated principles from the classical conditioning model should be integrated into the phenomenal aspects of understanding related to the nebulous field of placebo.

> It is perhaps time that we settle down to the tedious business of making these "nonspecific" effects specific by isolating, explicating, and specifying the type of subject, the type of therapist, and the situational and procedural conditions under which these effects can be negated, attenuated or potentiated (Wickramasekera, 1980, p.18).

The observation of the phenomenon that Siegel(1985) called "drug-mirroring" in which at times conditioned responses (CRs) move in a direction opposite to that of their associated unconditioned responses (UCRs) seems to run counter to the traditional paradigm or classical conditioning (Turkland & Brady, 1985) . This drug-mirroring is considered an important phenomenon for the understanding of the development of drug tolerance (Siegel, 1985) as discussed below. Form an orthodox perspective, drug-mirroring may appear to be

an incongruence within a strict classical conditioning model. However this controversy seems to be resolved by theorists such as Turklan (1989). He believes that the traditional perspective regarding classical conditioning is giving way to a more expansive concept of conditioned response which incorporates and synthesizes ideas and precepts formerly considered the exclusive domain of classical or operant conditioning, learning theory, and cognitive psychology. With this expanded perspective, he believes such phenomena as drug-mirroring and drug-mimicking can be understood within the realm of classic conditioning paradigm.

Relating specifically to the development of drug tolerance and withdrawal symptomatology, Siegel (1985) proposes a behaviorist model of placebo mechanism. In this Pavlovian model the active drugs act as the UCSs producing a specific physiological UCRs. Exposing an experimental subject to a specific drug UCS associated with consistent ecological cues CS has produced a condition in which the ecological cues alone cause a CR similar response to the drug UCR (Jodogne, 1990; Siegel et al., 1982) as well as opposite responses (Livine, et al., 1979; Siegel, 1985). When the CRs engendered are opposite to the drug responses UCRs they diminish the drugs UCSs effect size and mediate tolerance. This is termed by Siegel as "drug mirroring." Siegel (1985) points out that laboratory animals have shown this opposite drug CR to a variety of drugs. This is an important awareness to be incorporated in the methodology of analogue and natural setting experiments in which some drug or drug like intervention (e.g. CES) is being manipulated as an independent variable. The researcher must be aware of ecological stimuli that become associated with the onset of the intervention's action can create a "compensatory" CR. This compensatory CR may be one

explanation for the modulation of substance tolerance. More prevalent in occurrence and relevant to placebo effect is what are termed "drug mimicking" CRs (Ader, 1985; Siegel, 1980). In drug mimicking CSs obtain properties associated with the drug or drug like intervention UCSs and enhances their effect.

In relation to conditioned immunosuppression Ader (1985) also discusses drug mimicking. He proposes replacing active drugs to an extent with placebos by using partial conditioning. This idea is also broached by Evans (1974) and Wickramasekera (1980) regarding pain management. Using partial conditioning in a clinical setting the active drug (UCS) is sometimes omitted during the conditioning period and placebo (CS) is presented alone. Ader (1985) reminds the reader that generally continuous pairings of an UCS and CS produce a more robust acquisition of the CR, while partial reinforcement weakens the extinction of the CR. He also notes that: "To the extent that such drug action is not mediated by the CNS, they do not constitute UCSs and will not directly induce CRs" (p. 319 Ader, 1985).

The propositions regarding partial reinforcement proposed by Wickramasekera (1980) and Evans (1985) with respect to analgesics and Ader (1985) regarding the immunosuppression have some interesting clinical implications. It has been shown that certain drugs including some analgesics suppress the immune system (Ader, 1985; Donahoe & Falek, 1988). Also, there is often a question of tolerance and withdrawal in the use of medication (Thompson, 1985). A conditioned placebo effect CR may be acquired by associating the placebo with an active drug UCS using a continuous reinforcement schedule. With a potentially immunosuppressive or addictive active drug this constant pairing would continue

until the intervention became optimally effective. Then the amelioration of the presenting problem could be maintained on a partial schedule of administration of UCS alternatively administering the CR, thus reducing the inherent risk of addiction and/or immunosuppression.

A strictly behavioral orientation can be criticized on various grounds. It does not give credence to the position that CRs may arise from internalized expectations associated with UCS. It gives no weight to symbolic expectations or any nonconscious processes not being mediated by CSs. There may be times when expectations arise to manifest a CR without the mediation of external stimuli. For example the anticipatory fear felt by an agoraphobic CR can manifest itself simply by the expectation of leaving the phobic's house, before the CS of being outside the house is a reality. This idea falls within the framework of the cognitive information processing paradigm, and suggests the notion of an internalized processing algorithm (Lewicki & Hill, 1987). This notion hypothesizes that symbolic information (in this case expectations) can set into motion nonconsciously mediated behavior without the intercession of external stimuli. As is obvious from the above exposition, the psychodynamic and psychosocial paradigms also allow for symbolic mediation of placebo phenomenon without a need for external stimuli. From this perspective it is not necessary for the CRs to arise from the external CSs after former pairing with the drug or drug-like UCS.

Concluding Remarks on Placebo and Placebo Effect

White et al. (1985) state that placebo effects are a subset of biopsychosocial interactions. They have proposed a systems theory perspective that provides for the integration of disparate data and competing theories. This integrative perspective would

hopefully lead to the development and testing of an interdependent hypothesis regarding the nature of placebo and placebo effects.

Schwartz (1983) has proposed three criteria for an integrative "macro-level" theory:

1. Integration of diverse and/or competing theories relating to placebo phenomena.

2. Stimulation of new predictions and discoveries which are unattainable from theories that are more micro-level in nature.

3. The macro-level theories should be friendly to more micro-level theories by showing how micro-level theories are subsumed by macro-level theory, and represent specific levels of the encompassing theory.

The development of a more comprehensive model of treatment interventions would facilitate clinical efficacy as well as critical research. Development of this integrative model is needed because there is neither a single placebo effect nor level of efficacy in relation to placebo phenomena. Neither is there a single mechanism driving such effects. There are, in fact, a complex multiplicity of effects, mechanisms, and levels of efficacy. Table 5 (White et al, 1985) presents a vast array of variables that are placebogenic.

Table 5

Placebogenic Variables

I.	Cultural context
	A. Belief systems
	B. Faith
II.	Environmental
III.	Instruction
IV.	Suggestion
V.	Self-control
VI.	Expectancy
VII.	Outcome expectancy
VIII.	Efficacy expectations
IX.	Operant behavior
X.	Symbolic processes
XI.	Preparation characteristics

XII. Doctor-patient relationship
XIII. Patients' expectations and needs
XIV. Patient's personality
XV. Psychological state
XVI. Symptom severity
XVII. Anxiety/stress
- A Cognitive processes
- B Cognitive schema
  1. Self-schema
  2. Imagination
  3. Covert rehearsal
  4. Emotions

XVIII.. CNS influences on physiology
- A. immune system
- B. psychophysiological response to stress mechanisms
- C. endogenous endorphins

XIX. Classical conditioning
XX. Spontaneous remissions

The large number of variables derived from cultural and psychodynamic influences, psychosocial, cognitive, behaviorist, and psychophysiological theories present one of the major difficulties in developing an integrative placebo theory. To make understanding of this topic even more complex, one must consider the interaction of process levels linking biological, psychological and social systems. This biopsychosocial rubric was first proposed by Engle (1960) to more fully understand the human condition in relation to all health matters, physical and psychological. It makes sense that this integrative theoretical model should be used to try to understand a phenomenon such as placebo that has been investigated by the same major disciplines (i.e. biomedical, psychology, and sociocultural). These biopsychosocial levels are connected via interactive loops which need to be considered in any approach to defining, researching, and the development of theory regarding placebo phenomena. These system interactive levels linking biological, psychological, and social systems into biopsychosocial matrix are tabularized by Schwartz (1983). Placebo Table 6 is constructed so that each process as defined by Schwartz (1983) as marked by a lower

numbered is subsumed progressively by higher numbered processes.

Table 6

Processes Affecting Placebo and Placebo Effect

1. Homeostatic cybernetic self regulation
2. Classical conditioning
3. Operant conditioning
4. Motor skills learning
5. Discrimination training
6. Cognitive/behavioral/emotional/ecological self-control
7. Education and insight
8. Motivation and belief
9. Social interaction

This hierarchial systems perspective holds that effects which occur at one level have consequences upon other levels. This system is composed of interrelated subsystems via a complex matrix of totally interactive loops. The interaction of subsystem components may produce unpredicted new effects. A new integrative theoretical model which examines interaction effects between placebo variable and subsystems level may discover new combination that cannot be discovered when such components are studied in isolation.

It seems evident that all of the diverse definitions, methodological and theoretical positions described in this chapter contain valuable information for understanding the placebo phenomena. Only an integrative process such as the one described above allows for knowledge to be gleaned from such diversity and ambiguity. This diversity and ambiguity is typified by the legacy of placebo study. Such an integrative system would go a long way to resolve the innate disputes that develop when different theoretical positions vie for supremacy in the explication of any phenomenon. If we hope to understand such an important and complex topic as placebo phenomena, then an integrative theory allowing for various orientations to be incorporated into its structure seems necessary. As mentioned, placebo

phenomena have been observed since man first began to scrutinize the healing process. Placebo effects are so pervasive that any study of the treatment interventions for the multitude of human afflictions must consider the placebo effect. Yet our understanding of the placebo remains obscure and disjointed. Possibly, this ambiguity and obtrusiveness can best be dispelled by approaching the study of placebo from this biopsychosocial integrated systems model. Because of the pervasiveness of the placebo effect, any investigation of a treatment outcome should consider the influence of placebo phenomena not simply as a nuisance variable to be controlled but, also as a moderating variable that may be a component to the efficacy of treatment. Further, the methodologies of treatment outcome studies should attempt to isolate placebo phenomena, and determine the mechanisms driving their occurrence. In this way over time, nonspecifics may turn into specifics and some placebo effects will begin to transform into treatment effects.

CHAPTER IV

METHODS

Subjects

The subjects in this study consisted of 20 males and 9 females who were clients of the Oklahoma Department of Human Services (ODHS). They were referred to one of two chemical dependence treatment facilities (Tulsa Regional Medical Center or 12 and 12 Residential Treatment Center) sanctioned by ODHS. The subjects suffered from chemical dependence and consistently used two or more substances before admittance to the treatment facilities. All patients were admitted to at least a medical detoxification program. However, some subjects qualified for extended treatment, and these extended treatment patients were the focus of this study. The decision to give extended treatment was determined by the CDU staff after the patients were initially admitted to detoxification. Thus, when the subjects were recruited, the experimenters did not know if the subject was going to be a patient for detoxification only or an extended treatment patient. All subjects were screened for psychosis before being admitted into the chemical dependence program via their medical history and a psychiatric nurse who performed the initial intake interview for all admissions. The experimenters approached potential subjects for participation in this study within 24 hours of admission into the facilities.

All subjects were unpaid volunteers and met the following criteria before being

approached regarding entrance into this study:

1. The subject had no current or past history of psychosis.
2. The subject had never been treated previously by CES or any other similar treatment.
3. The subject was at least 18 years of age.
4. The subject had volunteered a history of regularly using more than one intoxicating substance and did not consider himself or herself addicted to just one drug.
5. The subject was not pregnant.
6. The subject did not have a demand type cardiac pacemaker.
7. The subject did not refuse the regular course of treatment provided by the chemical dependence unit.
8. The subject had not been admitted into the program more than 24 hours before he or she was recruited into this study.

Frequency Information Relating to the Subjects of the Experiment

There were nine subjects in Group 1, representing 31% of the total subjects. Group 1 were all non-CES controls who received the regular treatment protocols at the CDUs. There were nine subjects representing 31% of the total subject population in Group 2. They received simulated CES treatment in addition to the normal CDU protocol. Group 3, consisting of eleven subjects or 38% of the subject pool, received active CES treatment in addition to the normal CDU protocol.

The age of the subjects ranged from 20 to 49 with a mean of 31.3 years and a standard deviation of 7.7. Table 7 presents the in each of the experimental groups as well as the subject pool as a whole.

Table 7

Mean and SD of the Age of the Subjects

	Mean	SD
no CES	34.3	7.3
sham CES	31.9	9.4
active CES	29.6	6.5
All Grps	31.8	7.7

Thirteen (45%) of the subjects were single, 11 (38%) of the subjects were married and 5 (17%) of the subjects, in the experiment were divorced. Twenty (69%) of the subjects were males and 9 (31%) of the subjects were females. Twenty-two (76%) of the subjects were Caucasian and 7 (24%) of the subjects in the experiment were Afro-American.

As stated, the location of the experiment varied. Twelve of the subjects were seen at TRMC and 17 were seen at 12-and-12 Treatment Center.

Tables 8 presents the number of subjects involved in the experiment who showed positive and negative for the various drugs present in their urine beyond the various thresholds considered by the CDU's to represent substance dependence. The subjects were all polysubtance abusers and all subjects registered positive on two or more drugs. These considerations should be noted when perusing the following data. The respective threshold for each drug represented are as follows: ETOH <10.0 mg/dl negative; AMPHETAMINE

<300 ng/ml negative; BARBITURATE <200 ng/ml negative; BENZODIAZEPINE <200 ng/ml negative; CANNABINOID <25 ng/ml negative; COCAINE <300 ng/ml negative; OPIATE <200 ng/ml negative; PROPOXYPHENE <300 ng/ml negative; PCP <300 ng/ml negative. Table 8 presents the number of subjects in the CES experiment that showed a positive UA for beyond the threshold level for substance content in their urine as tested by the CDU's staff upon admission for treatment as observed in each experimental group.

Table 8

Positive UA Frequencies

	Positive no CES	n	Positive sham	n	Positive CES	n
Alcohol	4	9	3	9	2	11
Amphetamine	2	9	0	9	1	11
Barbiturates	1	9	0	9	1	11
Benzodiazepines	4	9	5	9	4	11
Cannabinoid	8	9	7	9	5	11
Cocaine	3	9	5	9	6	11
Opiates	1	9	0	9	1	11
Propoxyphene	1	9	2	9	0	11
PCP	0	9	0	9	0	11

Both of the CDUs often placed their clients including the CES subject into one or more of five medication protocols (see Appendix B) during the initial days of the detoxification phase of treatment. The prescriptions (Rx) consisted of either multiple vitamins, Librium, Ativan, Dilantin, or other medications, such as mild analgesic (aspirin or Tylenol), and/or mild gastrointestinal remedies, such as laxatives or antiacid medications.

Table 9 provides a breakdown of the medical protocols used in the various experimental groups and by the subject pool as a whole. It should be remembered that a patient may have been on several medication protocols at one time. However, in general, patients suffering from benzodiazepine and/or alcohol dependence received Librium, while cocaine addicted patients received Ativan. Patients testing positive for benzodiazepines or patients with a history of seizures during withdrawal were given Dilantin.

Table 9

Frequencies of Medical Protocols Used in Detoxification

	Vitamins		Librium		Ativan		Dilantin		Others	
	Rx	n	Rx	n	Rx	n	Rx	n	Rx	n
no CES	5	9	6	9	1	9	1	9	3	9
sham	6	9	5	9	1	9	1	9	2	9
CES	5	11	5	11	4	11	0	11	3	11
All Grps	16	29	16	29	6	29	2	29	8	29

The relevant information regarding the number of previous admittances to CD treatment facilities by the subjects is presented in Table 10.

Table 10

Subject's Previous Admission to Treatment Centers

	Means	SD
no CES	1.33	1.94
sham	1.78	1.99
CES	0.91	1.14
All Grps	1.31	1.67

Protection of Human Rights and Informed Consent

This study passed a Human Studies Review Board at The University of Tulsa, The Tulsa Regional Medical Center, and 12 and 12 Residential Treatment Center. All the subjects were fully informed of the experimental procedure both verbally and in writing. The subjects were asked to read carefully and then sign an informed consent form (see Appendix C) indicating that their involvement in the study was entirely voluntary.

Apparatus

CES Device

The instrument used to generate the cranial electrotherapy stimulation was the LB 2000, a product of Life Balance International Inc. of South Jordan, Utah. Fourteen portable units with self-charging batteries were provided to the experimenters by Life Balance International Inc. The device generates a gated sinusoidal wave burst of current with no direct current bias. The LB 2000 has a burst rate of 100 hertz per second with a pulse width of 2 milliseconds. The amplitude of current is adjustable from 0 to 1.50 milliamperes. The subjects received the CES current via two electrodes attached to a headset. The electrodes were coated with conductive gel and placed behind the subject's ear lobes at the maxillo-occipital junction of the mastoid process. Before treatment the electrode contact areas around the mastoid process of the cranium were lightly swabbed with alcohol to remove body oils and assure good electrical conductivity.

Additional Equipment

A charging device was provided to the experimenter by Life Balance International. All CES devices were recharged immediately after use, and charged for at least 12 hours to ensure that self-charging batteries were at full electrical potential. Fourteen blinding devices were also provided by Life Balance International. The blinding devices were connected in a circuit between the CES device and the electrodes, to either complete or break the circuit between the CES unit and the electrodes. Whether the circuit was open or closed depended on which of five settings were used on the blinding device. These settings were numbered (0-4) on the surface of the blinding device. Any setting was selected via a rotating dial upon which was an engraved arrow that pointed toward the selected setting. The zero setting provided a complete circuit and was used exclusively for setting the treatment amplitude just below tactile sensation threshold for electrical stimulation during the tactile sensation calibration phase of the treatment. This operation was done at the beginning of each CES treatment. This procedure was done for both the simulated and the active treatment subjects to ensure an equivalent treatment sensation for both groups. Settings 1-4 were either open or closed circuit settings, with two settings being open circuits to the electrodes and two settings being closed circuits. Whether a particular numerical setting was open or closed on a specific blinding device was known only to the manufacturer of the blinding device.

The setting on the blinding device for a particular subject was determined randomly using a random list of numbers taken from a statistics text (Kachigan, 1986) so that each setting of each device was presented once. A specific blinding device was assigned to a subject based on his order of entrance into the study with every third subject admitted being

placed into a control subject pool. A numbered "fanny pack" was used to contain a CES device and specific blinding device during treatment. The number on the pack corresponded to the number on the blinding box and only one blinding device was stored in or used with a specific pack. A lock was attached to the "fanny pack" to assure that neither the subject nor staff could tamper with the CES unit or blinding device during or after treatment.

Experimenters

An off-site research director supervised the development of the experimental protocol. The director determined the blinding box settings, the nature of subject group assignment, the scheduling of subject treatment assignment, and when to end the study. The two on-site experimenters were both doctoral students at The University of Tulsa. Both collected data, performed pretest and posttest assessments, administered CES treatments, and administered follow-up treatments. The experimenters were thoroughly trained in the administration and scoring of all the assessments in addition to being thoroughly familiar with the research protocol used in this study before the study began.

Outcome Measures

The Revised Beck Anxiety Inventory (BAI; Beck, Epstein, Brown & Steer, 1988)

The BAI is a commonly used self-report measure of anxiety. It was designed to assess symptoms of anxiety that are minimally shared with those of depression (Beck & Steer 1990a). It consists of 21 descriptive statements of anxiety symptoms rated on a four-point system as follows: (0 points)="Not at All"; (1 point)="Mildly; it didn't bother me much"; (2

points)="Moderately; it was very unpleasant, but I could stand it"; (3 points)="Severely; I could barely stand it." There have been extensive reliability and validity studies done on the BAI (Beck & Steer, 1990a) that show this instrument to be reliable and valid measures of anxiety among various clinical and nonclinical population and that they are sensitive to clinical change.

The Revised Beck Depression Inventory (BDI; Beck et al., 1979)

The BDI is one of the most widely accepted instruments in mental health for measuring depression in psychiatric patients (Beck & Steer, 1990b; Piotrowski et al., 1985) and detecting depression in normal populations (Beck & Steer, 1990b; Steer et al.,1985). It consists of 21 descriptive statements of depression designed to assess the severity of depression. It is also rated on a four-point system as follows: (0 points)="Not at All"; (1 point)="Mildly; it didn't bother me much"; (2 points)="Moderately; it was very unpleasant, but I could stand it"; (3 points)="Severely; I could barely stand it." There have been extensive reliability and validity studies done on the BDI (Beck & Steer, 1990b) show this instrument to be reliable and valid measures of anxiety among various clinical and nonclinical population and that they are sensitive to clinical change.

Structured Interview Guide for the Hamilton Anxiety Scale. (SIGH-A; Williams, 1988a)

The SIGH-A is a structured interview format of the Hamilton Anxiety Rating Scale (HARS, Hamilton, 1959) as revised by Guy (1976). It consists of the original 14 symptoms of anxiety as put forth in the revised HARS which are placed within a structured interview

format. Each symptom is accompanied by a symptom description and a five-point rating scale (0-4) with the following correspondence: (0 points)=symptoms not present; (1 point)=mild symptomatology; (2 points)=moderate symptomatology; (3 points)=severe symptomatology; (4 points)=very severe symptomatology. The first question relating to each of the original symptoms is always to be asked exactly as written on the form. Often this question will elicit enough information concerning the severity and frequency of a symptom to be rated with confidence. However, follow-up questions are provided to be used when it is necessary to gain further clarification of the symptom. The test is designed to cover the emotional state of the patient over the previous week. However, it can be used for any time period by simply verbally changing the period to as short as a three days period (Williams, 1988a). The HARS is the most widely used clinical rating scales for the measurement of anxiety and many studies of reliability and validity have been done on this scale (Riskind et al., 1987). The SIGH-A provides a standardized interviewing format that lends itself nicely to the demands of scientific methodology. It gives consistency to both the interview itself, particularly if more than one researcher is involved in a study, and also between the interviewer and all the subjects in a study. In his original article Hamilton (1959) shows the correlation of interrater reliability for the HARS as .89. This is an important characteristic of the assessment as at times the pretest and posttest of a given subject in this study were administered by different experimenters. To this author's knowledge there is no other reliability or validity study published regarding the SIGH-A. However, such a study being directed by Janet B. W. Williams is near completion. It is being conducted at Biometrics Research Institute, New York State Psychiatric Institute, 722 West 168th Street, New York,

New York.

Structured Interview Guide for the Hamilton Depression Scale (SIGH-D; Williams, 1988b)

The SIGH-D is a structured interview format of the Hamilton Depression Rating Scale (HARS, Hamilton, 1960). It consists of the original 21 symptoms of depression as put forth in the original HDRS placed in a structured interview. The SIGH-D is to be scored, as is the original HDRS, on only the first 17 of the 21 symptoms, although many investigators have used all 21 items (Hedlund & Vieweg, 1779). Each symptom is accompanied by a symptom description and an anchor-point descriptive scoring scale that increases in intensity; clinicians are to consider both the intensity and the frequency of a symptom when assigning it a rating value. Administration and scoring parameters are standardized, as each rating point of intensity and frequency has a specific qualifier attached to it. The first question relating to each of the original symptoms should always be asked exactly and written on the form. Often this question will elicit enough information concerning the severity and frequency of a symptom, allowing the investigator to rate it with confidence. However, follow-up questions are provided for use when it is necessary to gain further clarification of the symptom. The test is designed to cover the psychological state of the patient over the previous week. However, it can be used for any time period greater than three days (Williams, 1988b) by simply verbally changing the time frame when necessary as the interviewer administers the test. The HDRS is the most widely used clinical rating scale for the measurement of depression and many studies of reliability and validity have been done on this scale (Riskind et al., 1987). The SIGH-D provides a standardized interviewing format that lends itself nicely

to the demands of scientific methodology. It gives consistency to both the interview itself, particularly if more than one investigator is involved in a study, as well as interaction between the interviewer and all the subjects in a study. In his original article Hamilton (1959) shows the correlation interrater reliability for the HARS as .91. The test-retest reliability of the SIGH-D has been demonstrated to be .89 (Williams, 1988b).

Symptom Checklist (SCL) adapted self-report version of the Himmelsbach Scale

The SCL is a modified, self-report version of the Himmelsbach Scale (Kolb & Himmelsbach, 1938) and is one of the primary self-report measures used by the Midwest Research Institute in Kansas City, MO for studies pertaining to chemical dependence withdrawal. It was provided to the research team by Charles Graham (personal communication, Fall 1991). The SCL consists of 47 descriptive symptoms relevant to chemical dependence withdrawal and rated on a four-point system as follows: (0 points)=Not at All "never"; (1 point)=Mildly; "a little"; (2 points)=Moderately; "quite a bit"; (3 points)=Severely; "extremely." It is generic in nature and developed to be used with any substance withdrawal syndrome and was not specific to any particular substance or substance group.

Some psychometric properties of the assessments used for this study

The Cronbach coefficient alpha of internal consistency reliability (Kachigan, 1986) for the assessment used in this study as outcome measures are as follows:

1. HARS=.78 (Riskind et al., 1987)

2. HDRS=.73 (Riskind et al., 1987)

3. BAI=.93 (Beck & Steer, 1990b)

4. BDI=.86 (Beck & Steer, 1990b)

According to Beck and Steer (1990b) the correlation coefficient for concurrent validity between the revived BDI and the HDRS for six normative samples of depressed subjects is as follows:

1. Mixed Diagnostic=.66

2. Major Depression=.40

3. Major Depression (recurrent episodes)=.56

4. Dysthymic Disorder=.56

5. Alcoholic=.87

6. Heroin addicted=.69

The above six correlations are all significant at the $p < .001$ level

Beck et al. (1988) state that among anxious subjects the correlation coefficient between the BAI and the HARS is .51 at the $p < .001$ level.

Interrater reliability for the SIGH-A and SIGH-D in this study

To establish interrater reliability among the experimenters participating in this research, the HARS and the HDRS were given to eight subjects who were not included in the study. These subjects fit the study criteria and were admitted to TRMC for medical detoxification, but were tested more than 24 hours after admission. Both experimenters scored the interrater reliability subject simultaneously, while the principle investigator (PI)

administered the interview. The assessments were tallied after all eight subjects had been tested. A Pearson r product moment correlation (Kachigan, 1986) was used to determine the interrater reliability between the experimenters which was .99 for both the HARS and the HDRS.

Attention placebo control interview

An attention placebo device was used called the BPRS-CD Interview (see Appendix D) by the PI. It is a structured interview which was developed from the Brief Psychiatric Rating Scale (Overall & Gorham, 1962), in such a way that it was relevant to chemical dependence. This interview was given to all the subjects including controls to standardize experimenter/ subject interaction and control for attention placebo effects. Subjects were asked to comment on whether they had any problems with drug cravings, cognition, anger, interpersonal relationships or strange thoughts, and how they were feeling emotionally and physically. The interview lasted 10 to 15 minutes and was always politely stopped before 15 minutes elapsed. The interviewer simply asked the standardized questions in a polite manner offering no therapeutic support, restatement, clarification or general advice of any kind. The experimenter simply attentively listened after asking a semi-structured question containing the same salient content daily and noted the subject's responses. Although it was scored as part of the attention placebo procedure, the scores generated by BPRS-CD were not analyzed or presented in this study. BPRS-CD scores were not used because, although the interview format was in place before the experiment began, the scoring format was not used until the fourteenth subject was recruited into the study. The purpose of the BPRS-CD was to act as

a simple control for the effect of the interaction between the experimenters and subjects, including controls, and to assure that the CES treatment subjects and control had as similar an experience in the research protocol as possible with the exception of receiving CES treatment. Thus, any improvement in symptomatology shown by the outcome measures of the simulated CES subjects over the control subjects can be considered the result of placebo effects related to the CES treatment procedure. Further, the BPRS-CD interview or any aspect of its components has never undergone any reliability or validity studies and it was considered improper to use it as a quantifying instrument.

Setting

The study originated on August 23, 1992, at The Tulsa Regional Medical Center which was contracted by the ODHS to provide medical detoxification and chemical dependency treatment for its Tulsa clients. The subjects were all patients at the Chemical Dependency Unit (CDU). The CDU was a lock-in unit and there was a 24-hour staff present, consisting of registered nurses and substance abuse technicians. Between the hours of 7:30 a.m. and 5:00 p.m., the staff included licensed substance abuse counsellors, a clerical staff, and a clinical director besides the aforementioned staff positions. A physician was on call as needed and made regular rounds twice per week. The patient's quarters consisted of semi-private rooms with two patients per room. The treatment milieu was intensive, including medical detoxification, chemical dependence educational groups, process groups, relapse prevention groups, 12 step groups, and Alcoholics Anonymous meetings.

On December 1, 1992, the ODHS transferred their Tulsa substance abuse contract to

12-and-12 Treatment Center (12-and-12) in Tulsa Oklahoma. Thus, after December 1 only clients of 12-and-12 were approached for recruitment into this study.

The specifics of the 12-and-12 and TRMC settings were identical with two exceptions. The patients were housed four to a room and the CDU was housed in a considerably older building than the CDU at TRMC. Also, the 12-and-12 CDU was not located in a hospital as was the CDU at TRMC, but part of a larger substance abuse and transitional housing treatment center. Except for these differences the treatment milieu and the staff configuration were identical at the two locations.

Length of the Study

The study initially began in October of 1989 with the initiation of a pilot study consisting of the meta-analysis of CES in relation to substance abuse treatment (O'Connor, Bianco, and Nicholson, 1990). In July of 1992 the research team received final approval from the human subjects research committee at TRMC to proceed with the study. The first subject was interviewed on August 3, 1992 and last follow-up treatment was completed on July 7, 1993.

Procedures

Subject Recruitment

All new admissions who met the recruitment criteria were individually approached by the experimenters within 24 hours of admission to the CDU and asked to decide if they were interested in participating in the CES study. The experimenters verbally explained the nature

and the terms of the study and the informed consent. If the potential subject was cognitively unable to read and comprehend the informed consent due to detoxification symptoms, it was read to the individual. All patients who consented to enter the study did so voluntarily and were offered no remuneration for their involvement in the study. They were offered active CES treatment and stress management therapy after their involvement in the study was completed. If the subject agreed to be in the study, the subject signed the informed consent as did the experimenter. It was then dated and witnessed, and the pretreatment assessment and initial treatment (if the subject was not a control) immediately commenced. Originally, 65 subjects agreed to enter the study. However, 36 subjects (18 CES treatment subjects, 13 control subjects, and 5 CES simulation subjects) left the CDUs or became absent without medical approval (AMA). These subjects terminated treatment before posttesting for this study could be done. No subject dropped out of the CES study and remained at the treatment center.

Experimental Group Membership and Double Blinding Procedures

Immediately after the informed consent was signed, the subject was assigned a subject I. D. number. The I. D. number, the subject's demographics, as well as their personal and medical history relevant to the study, were entered on a CES study data collection sheet (see Appendix E). The subjects were introduced to the treatment room where all assessments, interviews, and CES treatment induction took place. The room in each facility consisted of an experimenter desk and chair and a comfortable chair for the subject. It also contained a storage cabinet that contained all CES study assessment materials, records, and apparatus.

The cabinet was locked unless needed by the experimenters. The subject was then given a pretreatment assessment battery with the previously mentioned outcome measures by one of the experimenters. Immediately after the pretesting, the subjects were assigned a blinding device code number. This code corresponded to the serial number of the blinding device and a specific blinding device setting. As stated above, the blinding device setting was chosen randomly. Thus, based on this blinding device setting the subjects were assigned randomly to one of three groups: a) Group 1--a no CES treatment control. These subjects received only the attention placebo and the normal CDU treatment. Every third admission to the treatment program entered the control subject pool. ; b) Group 2--a simulated CES treatment group plus attention placebo and the normal CDU treatment. In this group the subject received no active CES treatment other than the thirty seconds needed to calibrate the tactile sensation threshold. The subject received no CES if the blinding device setting represented a broken circuit. The circuit was broken as soon as the experimenter placed the dial on the prescribed setting after he was informed that the subject no longer felt the current during the sensation calibration phase ; c) Group 3--an active CES treatment plus attention placebo group and the normal CDU treatment. All subjects received the normal treatment facility's treatment regimen consisting of medical detoxification as prescribed by their physician and the normal treatment milieu which all patients at the facilities experienced (see Appendix F).

To insure that knowledge of CES treatment or control status would not affect the subject's pretest performance, the subject was not made aware of this status until the pretreatment assessments were completed. Whether a subject received active or simulated CES treatment was unknown to the subject, experimenter, treatment facility staff or any other

person involved with this research. Only the manufacturer of the blinding devices had the codes to determine which device provided active or simulated treatments. The research director was provided with these codes only one month prior to the termination of data collection. Only she had knowledge of whether a subject was in the active or simulated CES group until all data were ready for analysis . If a subject was in a CES treatment group that subject was *always* treated using the specific blinding device and setting that corresponded to his/her subject number. Further, a particular subject was treated using only one blinding device setting (1,2,3, or 4) that also corresponded to the subject's number. A specific blinding device associated with a particular setting was used until a subject completed the treatment protocol. In other words, if a subject dropped out of the facilities' treatment program leaving AMA, as happened with 36 subjects, that blinding device with the same setting was used again with the next subject in the treatment pool. This was done until a complete CES treatment sequence was completed using a specific blinding device with a particular setting.

Length of CES Treatments

Each subject in the CES treatment pool was given either active or simulated treatment for 45 minutes daily. If the CDU deemed a subject needed only detoxification, that subject was given CES treatments for only six days. The CES treatment sequence lasted 14 consecutive days if it was decided after detoxification that the patient was to be admitted to the facilities' extended programs. The extended programs lasted at least 28 days. Three subjects were given treatments for 15 days (working days excluding weekends over a 19 day period) due to scheduling problems between experimenters. When the patient entered the

CDU, neither the experimenters nor the subjects knew whether they would be in the facility for only detoxification or the extended program.

Subject Experimenter Interaction

To control for placebo effects based on experimenter/subject interaction confounds the subjects were assessed and treated randomly by the experimenters. For example, a subject might be pretested by one experimenter and posttested by another. Also, the number of CES treatments administered to and time spent in contact with each experimenter by each subject varied randomly. This was done for several reasons. Primarily, the schedules of the experimenters caused their availability for research to vary considerably. Thus, it was would have been very difficult, if not impossible, for the same experimenter to be present when a specific subject was due for their scheduled CES treatment between the subject's CDU activities that comprised their normal treatment program. Also, the attention placebo given all the subjects consisted of asking the subjects to report on various psychological aspects of their lives since last seen by an experimenter. It was decided that the information gleaned by the experimenter, particularly regarding the subject's affective states, might influence his expectation regarding treatment outcome. Therefore, it was decided that random contact might reduce this potential.

Administration of treatment

Introduction of the patient to treatment protocol

Immediately after pretreatment assessment the initial CES treatment commenced for

those subjects in the treatment groups. As per the experimenter protocol instructions, the experimenter said to the subject, "As you know, we are interested in studying the effectiveness of CES treatment as an addition to your other treatment at this facility. We use it to help you deal with the stress of chemical withdrawal symptoms. The CES device puts out a small pulse of electrical energy that you will only feel for about 30 seconds during your CES treatment. To make you comfortable with it I want you to try it on your finger to see what it feels like."

The experimenter plugs the electrodes attached to a head set into the blinding device. The electrodes are sterilized with an alcohol swab and the blinding device is set on zero. The experimenter hands the subject the head set, turns on the CES unit and says to the subject, "I want you to press the electrodes gently against each side of your finger, holding the headset with your other hand. Now I'm going to slowly turn the control knob until you say you feel a mild tingling in your finger." The subject states when he/she feels a mild sensation and the experimenter says, "now I'm going to slowly reduce the stimulation and right at the time you don't feel the tingling anymore, you let me know." When the subject first says he/she feels no sensation, the experimenter says, "that's exactly what you will feel during the treatments. The only difference is that you will be wearing the headset so that these cups (electrodes) are behind your ears and you'll be wearing this "fanny" pack with this number on it (subject number is attached to the pack). It will hold the CES unit and the blue box (blinding device). The unit will turn itself off automatically; when it does you'll hear a beeping sound. You can go anywhere on the unit you normally go and do anything you want while wearing the device except for taking a shower or exercising, because we don't want you to get the electrodes

wet. When you hear the beep come find me or a staff member and we will take it off for you. Please don't take it off your self, OK? If you're ready we will start your first treatment now." The experimenter waited for an affirmative response and the initial CES treatment protocol begins.

CES treatment protocol

The "fanny" pack is sized to the patient and placed behind the subject and out of reach. The experimenter sterilizes the head set with an alcohol swap and places a small amount of conductive gel on the electrodes. Next, he sterilizes the electrode placement areas around the maxillo-occipital region of the mastoid process and puts the electrodes in place. The blinding device is set on zero and the experimenter stands behind the patient and says,"OK I'm going to turn the unit on now. Please let me know when you first feel any tingling sensation where the cups touch your head." The experimenter slowly turns on the unit and increases the current flow until the subject first indicates that he/she feels a sensation in the area of the electrodes. The experimenter says, "OK, tell me when you first feel the tingle go away." When the subject says they feel nothing the experimenter immediately double checks the blinding device setting marked on the data collection sheet and reaffirms the match for that particular subject. He, then, sets the dial on the blinding device to that specific setting. Then the experimenter immediately writes the frequency setting for both the sensation threshold and the treatment, date, and time on the subjects data collection sheet. The CES unit and blinding device and excess cords are placed in the pack and locked. The experimenter reminds the subject not to remove the head set and to come back to the

experimenter when they hear the beep. The experimenter adds, "if I don't see you in 60 minutes I'll come and find you, OK?"

The experimenter arranges a daily time for successive treatments. Unbeknownst to the subject, this is always at a time when the subject is in a group, or at meals or at another time when the subject is under observation by staff. This minimizes the chance that the subject will remove the electrodes or tamper with the device in any way. All staff members are briefed concerning the experiment and instructed in the maintenance of experimental integrity. All new members to the staff are also briefed before they start their first shift on the CDU (without exception all staff members seemed eager to help in the project and were quite helpful during this study).

If 60 minutes expire and the subject has not come to the experimenter's office, the subject is located and the head set and the pack removed. After the treatment is over the CES unit is removed from the pack and immediately placed on charge. The experimenter sets the blinding device to zero, and the blinding device is stored in the locked pack and placed in the locked storage compartment. The experimenter completes any notes on the data collection sheet and also places it in the locked storage compartment. This protocol was repeated exactly every day until the subject ended the study, with one exception. On the second day and thereafter, all subjects including the controls were engaged in the attention placebo control procedure. This was done to insure that the subjects received the same amount of attention in a standardized manner from whichever experimenter administered the treatment on a particular day. With the treatment subjects this was done after CES treatment induction.

Posttreatment Assessment

After six treatments for the detoxification subjects and 14 or 15 days for the extended treatment subjects, the subjects were posttested with the same assessment battery as used in the pretreatment assessment. As stated, this was the revised Beck Anxiety Inventory (BAI; Beck et al., 1988); the revised Beck Depression Inventory (BDI; Beck & Steer, 1990b); The Structured Interview Guide for the Hamilton Anxiety Scale, (SIGH-A; Williams, 1988a); The Structured Interview Guide for the Hamilton Depression Scale (SIGH-A; Williams, 1988b), and Symptom Check List (SCL; Graham (personal communication, Fall, 1991). The posttesting session was done one hour after the last CES treatment. This was done so that any immediate short term characteristic, incidental, or placebo effects of treatment that might influence the outcome related to the timing of posttesting would be held constant. For the treatment subjects it was noted whether the subject and the experimenter who did the posttesting thought the subject received active or simulated CES treatment. All posttested subjects were debriefed regarding their participation in the study. The subjects were asked if they wanted the active CES therapy treatment for 14 days as promised during study recruitment. No subject declined this offer, and the day after posttesting the follow-up active CES treatment began and lasted for fourteen days or until the subject left the CDU. In addition, as promised during the recruitment, all follow-up subjects were administered stress management treatments consisting of progressive relaxation, autogenic relaxation, deep breathing exercises, and guided visualization.

CHAPTER V

RESULTS

Statistical Procedures

All statistical operations were calculated using the statistical soft ware "Statistica." "Statistica" is a product of Statsoft, Incorporated located in Tulsa, Oklahoma.

The original experimental design based on a power analysis of the meta-analysis study (O'Connor et al., 1992) presented in Chapter II called for a power level at .80 (beta =.20), with n from 60 (20 per group) with the effect size of .40, and an alpha of .05. It was not possible to achieve the n so another power analysis was performed. In order to achieve a power of .8 (beta =.2), alpha was set at .05, effect size was set at .60, and n was set at 30 (10 per group).

Analysis of variance (ANOVA) was used to determine the significance of the results. The main effects were initially tested using a simple one-way ANOVA. The independent variable was the treatment and dependent variable was the posttest measure. Additionally, an analysis of covariance (ANCOVA) (Campbell & Stanley, 1963)using the group pretest scores as covariant was calculated, because of an anomaly in the pretest data of the sham CES group which was not statistically different from the other groups, but was of interest. ANCOVA results indicated no additional significant effects beyond those indicated using the simple one-way ANOVA (the ANCOVA results are listed in Appendix G)

Scheffe tests were performed in order to determine the significant differences between the means each of the three groups. The groups were examined in the following manner: 1. CES treatment vs. no CES control; 2. CES sham vs. no CES control; 3. CES treatment vs. CES sham.

Effect sizes were calculated in order to determine the magnitude of the differences between groups. The groups were examined in the following manner:
1. CES treatment vs. no CES control; 2. CES sham vs. no CES control; 3. CES treatment vs. CES sham. The effect size was calculated using the error variance numbers obtained from a one-way ANOVA. The effect sizes were also calculated using the error variance numbers obtained using ANCOVA (see Appendix G). Using the following formula used to determine the effect size for the difference between posttest experimental groups as presented by Cohen (1980):

$$ES=X1-X2/S$$

X1=mean one posttest(i.e., treatment)

X2=mean two posttest (i.e., control)

S=pooled standard deviation (derived from mean square error variance of posttest)

Main Effects.

Hamilton Depression

Analysis of Variance

The ANOVA of the SIGH-D posttest scores indicated a significant effect, $F(2,26) = 9.17$, $p<.05$. The mean score for the CES treatment group was 5.45, the mean score for the CES sham group was 14.56 and the mean score for the control group was 17.44.

Scheffe Test

The Scheffe test indicates a significant difference between the CES treatment and the no CES control group, $p<.05$. Additionally, a significant difference were indicated between the CES treatment and the CES sham, $p<.05$. No significant differences were indicated between the CES sham and the no CES control, $p>.05$.

Effect Size

The effect size between the CES treatment and the no CES control was 1.80. The effect size between the CES treatment and the CES sham was 1.38. And, the effect size between the CES sham and the no CES control was .42

Figure 1

Plot of Means for the SIGH-D

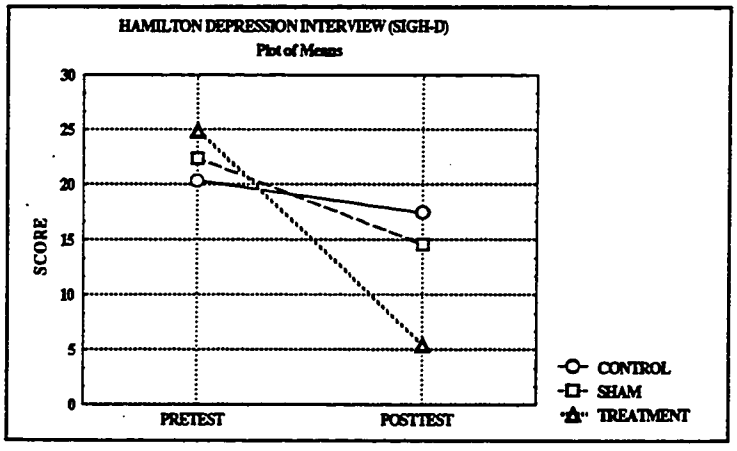

Figure 1. The SIGH-D for the pretest and posttest scores for the no CES controls, sham CES, and active CES . The means for groups.

Hamilton Anxiety

Analysis of Variance

The ANOVA of the SIGH-A posttest scores indicate a significant effect, $F(2,26) = 6.01$, $p<.05$. The mean score for the CES treatment group was 5.09, the mean score for the CES sham group was 15.67 and the means score for the no CES control group was 16.89.

Scheffe Test

The Scheffe test indicates a significant difference between the CES treatment and the control group, $p<.05$. Additionally, a significant difference were indicated between the CES treatment and the CES sham, $p<.05$. No significant differences were indicated between the CES sham and the no CES control, $p>.05$.

Effect Size

The effect size between the CES treatment and the no CES control was 1.42. The effect size between the CES treatment and the CES sham was 1.25. And, the effect size between the CES sham and the no CES control was .17.

Figure 2

Plot of Means for the SIGH-A

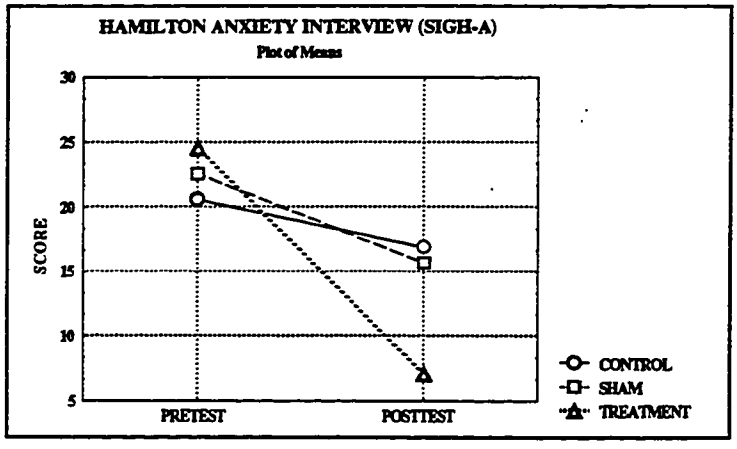

Figure 2. The means for the SIGH-A for the pretest and posttest scores for the no CES controls, sham CES, and active CES groups.

Beck Depression

Analysis of Variance

The ANOVA for BID posttest scores did not indicate a significant effect, $F(2,26) = 1.37$, $p>.05$. The mean score for the CES treatment group was 6.91, the mean score for the CES sham group was 11.22 and the mean score for the no CES control group was 12.11.

Effect Size

The effect size between the CES treatment and the no CES control was .68. The effect size between the CES treatment and the CES sham was .57. And, the effect size between the CES sham and the no CES control was .12.

Figure 3

Plot of Means for the BDI

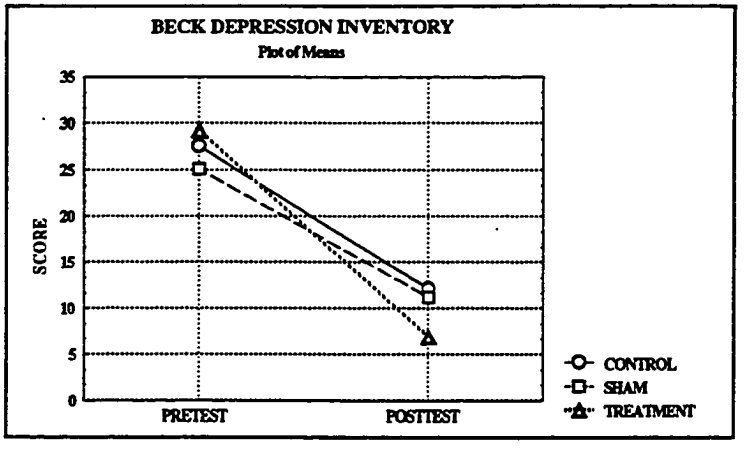

Figure 3. The means for BDI the for the pretest and posttest scores for the no CES controls, sham CES, and active CES groups.

Beck Anxiety

Analysis of Variance

The ANOVA of posttest BAI scores did not indicate a significant effect, $F(2,26) = .83$, $p>.05$. The mean score for the CES treatment group was 5.27, the mean score for the CES sham group was 9.33 and the mean score for the no CES control group was 9.78.

Effect Size.

The effect size between the CES treatment and the no CES control was .52. The effect size between the CES treatment and the CES sham was .46. And, the effect size between the CES sham and the no CES control was .06.

Figure 4

Plot of Means for the BAI

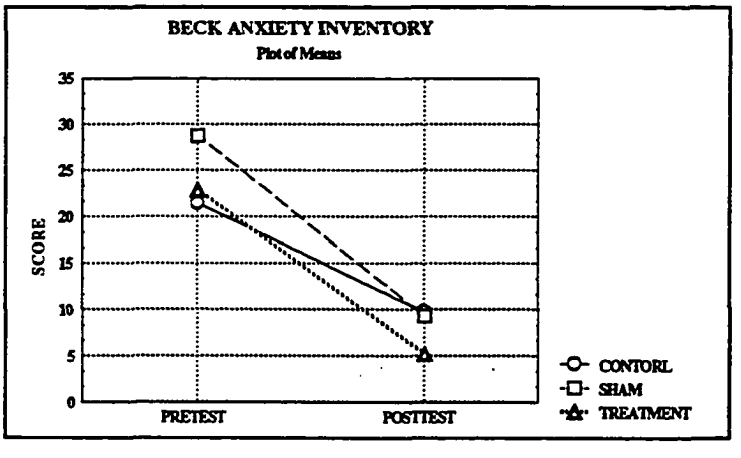

Figure 4. The means for the BAI for the pretest and posttest scores for the no CES controls, sham CES, and active CES groups.

Symptom Checklist

Analysis of Variance

The ANOVA for the SCL posttest scores did not indicated a significant effect, $F(2,26) = .46$, $p>.05$. The mean score for the CES treatment group was 18.00, the mean score for

the CES sham group was 24.11 and the mean score for the no CES control group was 29.22.

Effect Size

The effect size between the CES treatment and the no CES control was .56. The effect size between the CES treatment and the CES sham was .31. And, the effect size between the CES sham and the no CES control was .26.

Figure 5

Plot of Means for the SCL

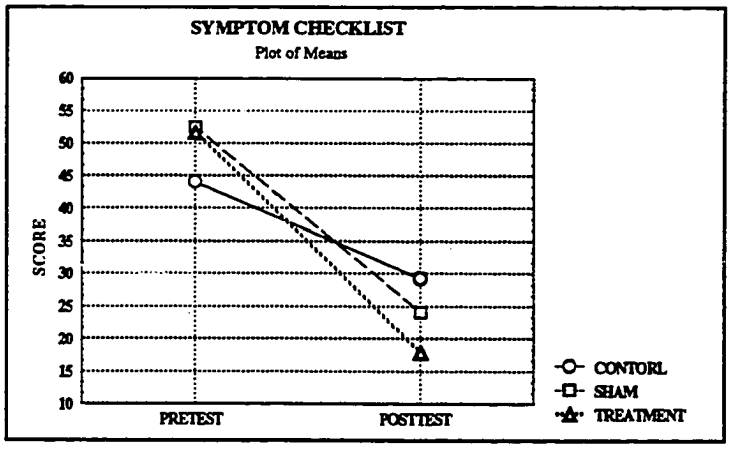

Figure 5. The means for the SCL for the pretest and posttest scores for the no CES controls, sham CES, and active CES groups.

Summary of ANOVA Results

Posttreatment outcome measures as analyzed by ANOVA are summarized and presented in Table 11.

Table 11

Summary of ANOVA results

Assessments	SS Effect	df Effect	MS Effect	SS Error	df Error	MS Error	F	p
SCL	630.38	2	315.19	10290.40	26	395.79	.79638	.46166
BAI	126.13	2	63.062	1967.74	26	75.68	.83325	.44591
BDI	158.10	2	79.047	1497.35	26	57.59	1.3725	.27122
SIGH-A	582.10	2	291.48	1261.80	26	48.53	6.0061	.00717*
SIGH-D	796.83	2	398.41	1129.17	26	43.41	9.1738	.00097*

Marked effects are significant at $p < .05$

Summary of Scheffe Tests

Posttreatment outcome measures as analyzed by Scheffe Tests are summarized and presented in Table 12.

Table 12

Summary of Scheffe Tests

Assessments	CES treatment vs. Control	CES treatment vs. CES sham	Control vs. CES sham
SCL	.46552	.79337	.86271
BAI	.52344	.58959	.99415
BDI	.32829	.46031	.96963
SIGH-A	.01568*	.03706*	.93326
SIGH-D	.00174*	.01783*	.65355

Marked effects are significant at $p < .05000$

Descriptive Statistics

Variable List

For the convenience of the reader, a list of all the variable numbers, abbreviations, and variables used in the analysis of the data of the experiment appears in Appendix H.

Interrater Reliability for the SIGH-A & SIGH-D

The interrater reliability correlation was performed to determine the reliability of the SIGH-A and SIGH-D between the two experimenters. Eight subjects recruited were from the Tulsa Regional Medical Center subject pool. These subjects were not included in the CES study. They were administered both the SIGH-A and SIGH-D by the PI and research associate. Both the PI (EXP-FB) and his research associate (EXP-DM) scored these subjects on the SIGH-A and SIGH-D. Table 14 shows the resulting scores as determined by the PI (EXP-FB) and his research associate (EXP-DM) Descriptive statistics Table 15 is the results of the over all interrater reliability correlation analysis. As the interrater reliability correlation was r =.99 for both the SIGH-A and the SIGH-D, the data for both measures is combined and presented in one table for brevity.

Table 13

SIGH- A & D Scores Used for Interrater Reliability

Test	EXP-FB	EXP-DM
SIGH-A 1	21.00	20.00
SIGH-A 2	25.00	24.00
SIGH-A 3	32.00	31.00
SIGH-A 4	27.00	27.00
SIGH-A 5	16.00	16.00
SIGH-A 6	20.00	21.00
SIGH-A 7	25.00	25.00
SIGH-A 8	30.00	29.00

SIGH-D 1	17.00	17.00
SIGH-D 2	23.00	24.00
SIGH-D 3	27.00	29.00
SIGH-D 4	32.00	32.00
SIGH-D 5	14.00	15.00
SIGH-D 6	23.00	23.00
SIGH-D 7	24.00	24.00
SIGH-D 8	31.00	31.00

Table 14

Interrater Reliability

N=16

	EXP-FB	EXP-DM
EXP-FB	1.00	.99
EXP-DM	.99	1.00

correlations are significant at $p < .05000$

Summation of All Descriptive Statistics

The descriptive statistics presented in Table 15 represent all the continuous variables used in the analysis of data. Categorical variables such as gender, marital status, race, and UA testing above the positive threshold for substances in the urine, were included in the frequency tables in the subjects subsection of the Methods section (see Chapter IV)

Table 15

Summation of All Descriptive Statistics

Variable	Valid N	Mean	Minimum	Maximum	SD	Std. Error
DAYSNUM	29	14.07	10.00	15.00	.88362	.165
PRESCL	29	49.55	12.00	100.00	24.16548	4.49
PREBAI	29	24.28	1.00	52.00	12.12405	2.25
PREBDI	29	27.45	6.00	49.00	10.06616	1.87
PRESIGH-A	29	22.69	7.0	40.00	8.51513	1.58
PRESIGH-D1	29	22.69	9.00	38.00	7.84266	1.46
PRESIGH-D2	29	26.24	9.00	46.00	9.26844	1.72
PT1SCL	17	34.53	3.00	75.00	23.01119	5.58
PT1BAI	17	16.59	0.00	39.00	13.13421	3.19
PT1BDI	17	14.53	1.00	29.00	8.42702	2.04
PT1SIGH-A	17	14.53	1.00	35.00	8.21673	1.99
PT1SIGH-D1	17	13.94	5.00	29.00	7.34397	1.78
PT1SIGH-D2	17	16.18	5.00	35.00	8.55304	2.07
PT2SCL	29	23.38	2.00	79.00	19.74917	3.67
PT2BAI	29	7.93	1.00	40.00	8.64759	1.61
PT2BDI	29	9.86	0.00	37.00	7.68916	1.43
PT2SIGH-A	29	12.79	3.00	36.00	8.11691	1.51
PT2SIGH-D1	29	12.00	1.00	31.00	8.29372	1.54
PT2SIGH-D2	29	13.62	1.00	37.00	9.95853	1.85
AGE	29	31.79	20.00	49.00	7.72925	1.44
ADMDATE	29	92.62	92.23	93.16	.40350	.075
MED1VIT	29	.55	0.00	1.00	.50612	.094
MED2LIB	29	.55	0.00	1.00	.50612	.094
MED3ADA	29	.21	0.00	1.00	.41225	.077
MED4DIL	29	.07	0.00	1.00	.25788	.048
MED5OTR	29	.28	0.00	1.00	.45486	.085

MAMP.TX0	11	.23	0.10	.330	0.68109	.021
LOCATION	29	1.59	1.00	2.00	.50123	.093
PREVADMT	29	1.31	0.00	6.00	1.67126	.310
SUBJOPIN	29	3.34	0.00	9.00	3.87553	.720
RESOPIN	29	3.21	0.00	9.00	3.97653	.738
UA1ETHOL	29	.31	0.00	1.00	.47082	.087
UA2AMPH	29	.10	0.00	1.00	.30993	.058
UA3BARB	29	.07	0.00	1.00	.25788	.048
UA4BENZ	29	.41	0.00	1.00	.50123	.093
UA5CANN	29	.69	0.00	1.00	.47082	.087
UA6COKE	29	.41	0.00	1.00	.50123	.093
UA7OPIA	29	.07	0.00	1.00	.25788	.048
UA8PCP	29	0.00	0.00	0.00	0.0000	0.00
UA9PROP	29	.10	0.00	1.00	.30993	.058

Means and SD for Pretreatment and Posttreatment Assessments

The means and SD for the pretreatment and posttreatment assessments are presented for the experimental groups and the subject pool as a whole in Table 16 and Table 17 respectively.

Table 16

Means and SD of Pretest Outcome Measure Scores

	SCL		BAI		BDI		SIGH-A		SIGH-D	
	Means	SD	Means	SD	Means	SD	Means	SD	Means	SD
no CES	44.00	22.82	21.44	9.89	27.56	9.74	20.56	6.21	20.33	6.56
sham	52.44	30.05	28.78	15.21	25.11	10.86	22.56	9.95	22.33	10.14
CES	51.73	21.35	22.91	10.99	29.27	10.25	24.55	9.22	24.91	6.67
All Grps	49.55	24.17	24.28	12.12	27.45	10.07	22.67	8.52	22.70	7.84

Table 17

Means and SD of Outcome Measure Posttest Scores

	SCL		BAI		BDI		SIGH-A		SIGH-D	
	Means	SD	Means	SD	Means	SD	Means	SD	Means	SD
no CES	29.22	20.06	9.78	12.17	12.11	6.15	16.89	9.06	17.44	6.97
sham	24.11	19.46	9.33	7.97	11.22	11.16	15.67	7.92	14.56	9.08
CES	18.00	20.11	5.27	5.23	6.91	4.46	7.09	3.21	5.46	2.91
All Grps	23.38	19.75	7.93	8.65	9.86	7.69	12.79	8.12	12.00	8.29

Interactions Between Variables and Differences Between Variables at the Pretest

There were no significant interactions between variables ($p<.05$) affecting posttest results neither were there any significant differences between variables ($p<.05$) at the pretest level . To determine if there were interactions between the treatment and other variables and/or significant statistical difference between variables at the pretreatment a two-way ANOVA was performed using the treatment as the first predictor variable in combination with all the other pertinent variables in the data set as the second predictor variable. The second predictor variables investigated were age, gender, marital status, location of treatment (CDU), medical protocol, UA, and previous admissions to CD facilities. The posttest measures were used as the criterion variable for investigation of interactions and the pretest measures were used as the criterion measure for investigating any statistically significant difference at the pretest.

CHAPTER VI

DISCUSSION

Summary of Findings

The main thrust of this study was to examine the effects of CES on the secondary withdrawal symptoms of anxiety and depression that are often concomitant of substance withdrawal during the initial stages of substance abuse treatment. The data show that the null hypothesis proposing no significant differences among the three experimental groups can be rejected. The active CES treatment procedure, when combined with the normal treatment regimen given at the treatment facilities was more effective in reducing anxiety and depression in a clinical population of chemically dependent polysubstance abusers than the normal treatment regimen alone and the sham CES plus normal treatment regimen. The assessed impact of the sham treatment plus the normal treatment regimen was not statistically different from the no CES control treatment. Thus, the anticipated results regarding CES was supported, while the anticipated results regarding placebo effect was not supported. Among the observer-rated outcome measures there was a statistically significant difference between the improvement shown by the active CES group and the sham CES group, with the active CES group showing the greater improvement. Although there were no statistically significant differences shown among the subjective self-report measures, there was a trend toward significance. This study supports the findings of authors who show CES to be an efficacious treatment for the treatment of anxiety and/or depression among the chemically dependent

individuals (Gomez & Mikhial, 1978; McKinzie, Castello & Buck; 1976; Patterson, 1976; Patterson et al., 1984; Schmitt et al., 1986; Smith & O'Niell, 1975). This investigation's findings do not agree with the finding that stated CES treated patients improved no more than controls (Snodgrass, 1977; Tomsovic & Edwards, 1973). Of all the studies done on chemically dependent subjects only, the Schmitt et al. (1986) study dealt with a similar population as the current study, namely a polysubstance abuse population. However, the Schmitt et al. (1986) study had pure alcoholics mixed with the polysubstance users. Therefore, the current study represents the only investigation of the effectiveness of CES with a solely polysubstance abuse population.

Only two studies of a CES type of treatment approximate the methodological rigor of this study. Taylor (1992) used a TENS unit (a cranial electrotherapy stimulator producing a different wave form than the LB-2000) and studied its effect on psychophysiological response to mental stressors. Schmitt et al (1986) had a very well designed study of CES affects on anxiety and depression among chronic alcoholics. Unfortunately, their results were presented without means and standard deviations but in the form of percentile ranking graphs.

The PI also believes that the findings of the current study are more generalizable to indigent, agency-referred CD clinical populations than previous CES studies. This investigation better defined the typical indigent or so called "hard core" CD client than previous studies. The typical indigent, agency managed CD client of today is a multiple substance user with one or two drugs of choice, excepting the pure alcoholic or occasional single substance user, many of which (50%) exhibit severe psychological symptomatology (Struening et al. 1991). Most previous CES drug studies described the subject by a single

particular drug of choice. Probably, the subjects used more than one drug regularly, because the addict typically under-reports substance use and the variety of drugs used (Craig, 1993; Tomas & Kozel, 1991). Because the typical substance abuser is an unreliable witness for self-report (Craig, 1993; Hopkins & Frank, 1991; Tomas & Kozel, 1991) the CES studies that identify a subject's inclusion in a study based on self-reported drug use may have suffered from sampling errors. Only in recent years has urine analysis (UA) been used to define drug usage as opposed to self-report. This investigation is the only CES study of which the PI is aware that categorized its sample via UA. This is likely because the previous CES studies were done, for the most part, before the wide spread use of UA.

Limitation of the Study and Proposals to Reduce Extraneous Variables

It is important to recognize certain improvements could be made in the methodological format of the current study to control for extraneous variables in future studies, if feasible. In any research it is important not only to report successful results of treatment, but also to look at possible extraneous variables and alternate explanation for these results. This is particularly true with clinically based research in a natural setting because of the increased possibility for the intrusion of extraneous variable into these types of studies (Barlow, 1981).

A power analysis done for this study based on the meta-analysis for CES studies (O'Connor et al., 1990) showed the number of subjects needed to maintain sufficient statistical power (Cohen, 1977) in the experiment was 20 subjects per group or 60 subjects in the case of the current study. Unfortunately, due to the unforeseen difficulty in collecting

data with this population, subject attrition, and scheduling conflicts with prior commitments, the collection of data ceased after data on only 29 subjects were collected. This was true even though the data was collected eight hours per day, seven days a week for nearly eleven months. Regarding attrition, it is worth noting that the attrition rate of this study was nearly identical to that of the general attrition of both CDUs, and did not effect one experimental group more than another. To compensate for the smaller number of subjects the effect size level was raised from .40 to a more conservative figure of .60, while alpha and beta were held constant at .05 and .20 respectively. Thus the study remained within acceptable limits to guard against Type I and Type II errors. However, if this study is replicated it is recommended that 20 subjects per group be used to insure full randomization of the experimental design.

Placebo effects must be considered in any clinical treatment outcome study based in a natural setting (Elmes et al., 1989). Placebo effect was of particular interest in this study. The nature of CES treatment, in general, and perhaps more so among the CD population, lends itself to the creation of placebo effects. From a biopsychosocial integrative perspective as discussed in Chapter III, CES treatment has several potentials for powerful placebo effects acting as moderating for research in this area. From a cultural perspective, in our Western scientific and technologically based society, individuals expect technical "high tech" interventions to have more potential curative potency then more traditional "low tech." treatments. This increases the mediational aspects of expectancy from such "high tech" intervention. The fact that the sensation of an electrical charge is present during the CES treatment also increases the expectancy in the patient/subject that something is going to

happen with this treatment. The informed consent necessary in this or any research must state that CES has been used for many years without any harmful effects and may help them with their problem. This also increases the expectancy that something "positive" may happen. The fact that the nature of CES treatment in this and most studies requires daily contact with a "care giver" increases the potential for attention placebo effects. The CD population is used to and, in fact, seeks out alterations to the CNS and their concomitant psychotropic effects. Thus, it was reckoned that anything that has the potential to effect the brain might have a particular potency to alter perception of potency and effectiveness with this population. Also, as mentioned in Chapter II, several authors said they believed CES had a placebo effect when considering the improvement of anxiety and/or depression. With these potentials, it seemed that it might be likely, even with controls for its effects, that placebo effects might play a strong role in any positive results seen with this treatment.

To the surprise of the PI no such placebo effects were found and the current study does not support those findings that showed CES had solely, or in large part placebo effects on anxiety and depression. The sham CES group showed no statistically significant improvement over the control group on any of the outcome measures. The observer-rating outcome measures (SIGH-A and SIGH-D) showed the active CES group to have a statistically greater decrease in affective distress than the CES sham group. This leads to the conclusion that the CES treatment shown to be effective in this study was not affected significantly by placebo effects. The PI believes no significant magnitude of placebo effects was shown because they were managed methodologically through the use of attention placebo control devices, as well as expectation placebo controls via the sham control protocol, the

double blind design, and the randomization of treatment and assessment administration. The attention placebo.controls consisted of daily and weekly semi-structured interviews that were part of the procedural protocol for all CES study subjects including the no CES treatment controls. It was thought that perhaps the change of ecology from a hospital setting (TRMC) to a treatment center (12-and -12) may have been a moderating variable. However, testing the group differences against the various treatment ecologies using one-way ANOVAs, produced no statistical differences. There were no interactions between the UA groups and group outcome differences. For that matter, there were no interactions found between any of the variables. Unfortunately, only UA screens were used with threshold cut-offs to determine drug use by the subject. It is possible that the level of addiction, which should be measured by a continuous scale UA, may have affected the results. Thus, the inclusion of this type of UA would be preferable in any further studies of CES among CD subjects. However, this is an expensive procedure and funds were not available for its use in this study. It is recommended that the use of a fully quantified UA screen be included in further CES studies to investigate the possibility of severity of addiction as an moderating or extraneous variable. Due to the expense of this instrument, a possible alternative might be an extensive structured clinical interview to evaluate substance abuse in addicts (Craig, 1988) The Addiction Severity Index is another possibility (ASI; McLellan, Luborsky, Woody, & O'Brien, 1980). This is an objective test which, as with all self-reports used with the CD population is susceptible to distortion and relies heavily on the openness and honesty of the patient. This dependence on honest self-report can be problematic, concerning symptomatological veracity in this population, according to the National Institute on Drug Abuse (NIDA) (Craig, 1993).

Self-report versus Observer-ratings in CD research.

The question of self-report versus clinical observational rating seems a central issue in interpreting the results of this study. As mentioned , the SIGH-A and the SIGH-D showed a greater statistically significant effectiveness with CES treatment plus the normal CDU treatment regimen than the sham CES plus normal treatment and the normal treatment milieu alone, while the self-reports (BAI, BDI, and SCL) did not. The debate regarding the relative reliability and validity of self-report versus clinical observation and interview is long standing and controversial, with each side presenting reasonable arguments regarding their perspectives (Anastasi, 1982; Catell, 1986; Friedman, 1989; Wetzler, 1989). As psychological assessment is not the main thrust of this investigation, it seems inappropriate to present the whole of each side's argument here.

Very briefly, the points of each side's advantages and limitations can be summarized as follows:

A) Self-reports are based on introspection and self-evaluation and are thus accurate reports of the way a person evaluates him/her self at the time of report. They are economical, easy to administer, and standardized. Self-reports have been proven reliable and valid for temperament or general personality traits sensitive to clinical change. However, this is true only when the person reporting is reliable, honest and cognitively able to be introspective (Catell, 1986; Wetzler, 1989). The limitations of self-reports are such that they may not be reliable measures of abilities, dynamic traits and interests. Also, they require the full cooperation of the individual and they possess what Catell (1986) calls *"subjective floating*

norms." These norms imply that whatever is considered the sample mean of what is measured is peculiar to the reporter. Further, self-reports are fixed to the situational and response modes (i.e., tests versus other modes of assessment) and there is no way to calculate the homogeneity of the observations derived from cumulative self-reposts (Catell, 1986).

B) Structured observational-ratings are based on ratings and recorded events and seem to be truly objective, but they must be filtered through the perceptions of the observer (Catell, 1986). They can be sensitive to clinical change if the observer is trained in the signs and symptoms of the disorder in question and if the observer maintains objectivity (Freidman, 1989). Observer-ratings can be reliable and valid for abilities, interests, dynamic features, as well as temperament and personality traits (Catell, 1986). Observer-ratings do not require full cooperation of the interviewee. They do not have fixed situation and response modes (Catell, 1986). Their limitations are they demand a high degree of training of the clinician, therefore their use can be costly. Observer-ratings can be subject to observer bias and/or observer/client interactions (Catell, 1986). As with the self-report, observer-ratings are subject to the *"floating norms"* Catell (1986) discusses.

Given what has been said, if observer-ratings are to be used in scientific investigation, it is necessary to insure that the observations are as close to objective as possible. These instruments demand multiple observers who have displayed a high degree of interrater reliability. This was the case in this investigation with the SIGH-A and SIGH-D being given in random fashion by two observers with an interrater reliability correlation of .99. The PI believes that this high degree of interrater reliability was due to two factors. First, the experimenters underwent the rigorous standardized training in the use of the SIGH-A and the

SIGH-D. This was provided through precise instructions via video tapes by the author of these scales. Not only were detailed instructions provided, but practice interviews with actual patients having varying degree of mood disturbance were used as interview examples along with interview modeling provided by skilled researchers. Second, the nature of the SIGH-A and SIGH-D is such that they provide specific parameters for the interview questioning and experimenter/subject dialogue, and also observer-rated scoring.

This is relevant because a central issue for this study is the discrepancy in results as represented by the self-reports and the clinical observer-rated interviews regarding clinical change. Why did the self-reports not also show the statistical difference between the various experimental groups and to what may we attribute differences in magnitude of treatment effect shown by the two different formats of outcome measures? The answer to these questions can be found in part by a proponent of self-report Scott Wetzler (Wetzler & Katz, 1989) when he states:

> The question of how sensitive self-reports are to clinical improvement is a relative one. Are self-report tests earlier or later indicators of clinical change in comparison to other vantage points? Some authors suggest that the self-report is more sensitive to clinical change that an observer' vantage (Raskin & Cook, 1976), whereas others conclude that clinicians are more sensitive to clinical changes (Lambert, Hatch, Kingston, & Edwards, 1986). It is difficult to make a sweeping generalization about this topic since types of illness differ, severity of illness may vary widely, and self-report tests differ with regard to specificity of the changes measured. Nonetheless, it seems fair to conclude that if a patient makes a significant improvement, that both self-report tests and observers' ratings will be sensitive to change. Which vantage is the earliest indicator of change seems to depend on the particular disorder or component of psychopathology being assessed.
>
> To take an example using patients with severe depression described by Katz (1987), observers were more sensitive than patients to changes in certain components of the illness (e.g., depressed mood), but less sensitive than the patients to other components (e.g., hostility). By the time the patients were fully recovered, all vantages corroborated each other. (pp. 111-112)

Katz's (1987) position is supported by Watkins et al. (1993) specifically regarding the HRSD and the BDI. They showed in a comparative treatment study between pharmacotherapy and various psychotherapies that the HRSD, was sensitive to a clinical change at statistical significance at eight weeks while the BDI was not sensitive to such a change until 12 weeks of treatment. Other studies of pharmacotherapy and psychotherapy as they relate to the relief of depression (Edwards et al., 1984; Greenberg et al., 1992: Huges et al., 1982; Lambert et al., 1986; Moran & Lambert, 1983) demonstrated a similar differential sensitivity to clinical change between the BDI and the HRSD. These authors attempt to explain the difference with various arguments on both sides of the controversy. Lambert et al. (1986) speculate the difference may be due either to halo effects by self-reports or to the fact that observers may manifest response sets in the direction of therapeutic change. Carroll et al. (1973) believe that self-reporters may be minimizing, while Huges el al. (1982) speculates that they may be exaggerating their depression.

Sayer et al. (1993) point out that the median correlation of pretest scores between the BDI and the HDRS from 12 studies investigated was only .58. Various researchers have suggested the modest correlation between the HRSD and the BDI is due to differences in the symptomatology sampled by each assessment. (Lambert et al., 1986; Sayer et al., 1993). The HDRS emphasizes behavioral and somatic symptoms of depression while the BDI places the greatest weight on the subjective experience of depression. This is intuitively obvious given the nature of each assessment. In this case, the argument might shift to a question of whether the observable symptoms of depression improve before the subjective dissipation of

depression. (Huges et al, 1982; Lambert et al, 1986) . Finally, there is the cynical, but relevant question of there being a halo effect due to the researcher's investment in obtaining a successful treatment effect (Edwards et al., 1984; Greenberg et al., 1986; Lambert et al. 1986). This proposition is easily allayed by having a properly designed double-blind experimental study with a placebo or sham group (Greenberg et al., 1986) as in the current CES investigation.

Sayer et al. (1993) present an explanation of why observer-ratings may be a better measure of clinical change when measuring depression. Using factor analysis in an attempt to determine why the HDRS and the BDI have demonstrated differential findings in the study of depression these researchers conclude the HDRS may be a better instrument for measuring clinical change in depression. The researchers used effect size to measure clinical change among clinically depressed patients, treated with ECT. Their factor analysis showed a group of depressed patients that catastrophize their symptoms and over-report depression on the BDI posttests. This marked increase of perceived depression by the subjects in this sub-group on the BDI posttests did not influence the observer-raters using the HRSD. Further, the researchers said some individual self-reports were low on the BDI pretest while observers rate them substantially higher on the HDRS. Thus, on the BDI there is little room for clinical improvement as compared to their situation with the HRSD. According to Sayre et al. (1993), the disparity in this group of subjects was not due to the differential samples of symptoms manifested by the scales; rather, factor loadings for both scales of a factor analysis showed depressed mood to be the first factor, with the second factor being somatic symptoms. Finally Sayer et al. (1993) point out that a substantial number of patients had

low baseline scores on the BDI leaving little room for improvement. The effect size for improvement was approximately 50% greater with the HRSD in comparison to the BDI and this is explained by the three subgroups of patients as assessed by the BDI: 1. Those with a low baseline score on the BDI; 2. those with a low baseline on the BDI and a high baseline on the HRSD; 3. and those who reported a marked increase of symptoms with the BDI. As with other researchers, Sayre et al. note that the correlation between the HRSD and BDI at baseline is modest with the HRSD and the BDI sharing only about 25% of common variance. After the course of the treatment the outcome measures were repeated and this relationship of commonly held variance increased to nearly 60%. The effect size for change was substantially higher with the HRSD (1.54 SD units) than with the BDI (.78 SD units).

With respect to the differentiation of anxiety and depression Fledman's (1993) factor analysis of these mood disorders with clinical and nonclinical populations states that self-reports including the BDI and the BAI do not distinguish between these affective constructs. Similar results have manifested regarding the non-discrimination between these affective states with the use of observer-ratings. However, when observer-ratings were used for diagnostic purposes, Fledman (1993) states:

> A differentiation has been found in correlation studies (e.g. Beck et al., 1988; Riskind, Beck, Brown & Steer, 1987) only when clinician's ratings are made in the context of the diagnostic process, with an inclination toward differential diagnosis between anxiety and depression (L. A. Clark, 1989). (p. 636)

This quotation is important to this study because it was precisely the intent of the experimenters to differentiate diagnostically between anxiety and depression when using the SIGH-A and SIGH-D. Further, the very nature of the structured interview and the symptom

description aspects of these instruments was to help differentiate between affective disorders and provide an objective criteria for evaluation.

With regard to the use of self-report assessments specific to substance use, there is little disagreement among those that have studied the issue (H. Catell, 1986 H. Cohen, 1986; Craig, 1993; Kolb & Himmelsbach, 1938; O'Leary & Donavan, 1974; Schuckit, 1979; Ross & Glaser, 1989; Tomas & Kozel, 1991). Singular reliance on the self-report among substance abusers is unreliable as a source of symptomatology and/or substance use. The use of only self-reports to glean information from the substance abuser has been described as at best problematic (Craig, 1993) and at worst totally unreliable (Tomas & Kozel, 1991). Speculation as to why this is so runs from explanations relating to organic cognitive dysfunction to functional explanations such as the high comorbidity of personality disorders with drug abuse (Craig, 1993). Whatever the explanation, experts in the field of substance abuse seem to agree with Craig (1993) who believes that the clinical interview provides the best tool for symptomatic diagnosis among the chemically dependent. He goes on to say that self-report instruments are more susceptible to distortion and denial than observation-rating among the CD patient. Craig (1993) says that temperament may be reliably assessed by self-report during treatment, however one should wait until after the detoxification, because the self-report can be exaggerated due to the effects of withdrawal. O'Leary and Donavan (1974) point out that substance abusers exhibit distorted self-perception of psychological impairment with self-report. Others state the same distortions of psychological symptoms especially symptoms of withdrawal that mimic anxiety and depression (H. Catell, 1986; Ross & Glaser, 1989; Schuckit, 1979).

If one accepts that self-reports are more susceptible to exaggeration, distorted self-perception, and unreliable veracity than are observer-ratings, due to the unreliable testimony of the substance abuse population, this may explain the higher rating of mood disturbance on the posttests by the BDI, BAI, and SCL than the SIGH-A and SIGH-D in this experiment. Also, if Sayre et al. (1993) are correct and a subgroup of catastrophizing and under-reporting subjects can cause differential results between the Beck and Hamilton scales, this may in part explain the differential results shown between the self-report and observer-rating scales in the current study. Further, as stated, the clients in each CDU were judged by the staff as to whether they would continue in residence for the full 28 days of treatment after two weeks. Unfortunately, for the internal validity of this experiment this judgement was usually based on the gravity of the clients withdrawal and recovery symptoms both physical and psychological and the motivation of the client to stay in treatment. The clients were aware of the decision criteria. Thus, it may have been that secondary gain may have precipitated a faking bad or a cry for help leading some subjects to over-report on the self-reports, even though they were told these reports were confidential. There may have been the motivation and subsequent secondary gain to stay in resident treatment, because many of the subjects were indigent and homeless or lost their housing while in treatment. The preceding is another possible explanation regarding the differential result seen between the self-report and the observer-ratings in this study. In future CES studies it may behoove the experimenters to have the staff reassure the subjects that in no way would the outcome of their experimental assessment be used to make treatment judgements. As stated above several authors have shown that the HRSD, for whatever postulated reasons, often shows clinical change before

the BDI. Later, after the treatment is complete these two assessment instruments have a higher correlation as to clinical change. Had the study been able to posttest the subjects after the recommended 28 day period of treatment the scores between the self-reports and the observer-ratings might have equilibrated.

The results of this study bring into question the use of self-reports with this population. The use of self-reports in future CES substance abuse research attempting to replicate this study's findings might investigate this question by changing the methodology of the study. This change would include more appropriate outcome measures or an extension of the treatment in order to take advantage of the aforementioned convergence of findings that occur between the Beck and Hamilton Scales over time. The description of this phenomenon in relation to the HRSD and the BDI by so many researchers spanning so many years is an argument for a greater sensitivity to certain symptomatic changes by observer scale, at least for depression. If one accepts Feldman's (1993) position that anxiety and depression are non-distinct psychologically one could extrapolate this to anxiety as well.

Another explanation of the differential results between self-report and observer-rating in the CES study may be related to experimenter bias as Greenberg et al. (1986) suggest. The experimenters remained blind throughout the study. Because of the nature of the attention placebo devices they may have been aware of affective improvement in the patients. Having noted the subjects that were getting better they may have guessed who was being treated with active CES and who was receiving sham CES. This possibility, in turn, might compromise their objectivity on the observer-rating posttest and create a halo effect toward positive treatment effects. In fact, the experimenters were correct in predicting group membership

91% of the time for the CES treatment group and 78%of the time for the sham group. However, the subjects predicted group membership 91% of the time for the CES treatment group and only 33% of the time for the CES sham group. The PI believes this was due to the amelioration or non amelioration of the symptoms which may have been more evident because of the daily placebo attention control which inquired as to the subject's emotional state among other things. Therefore, if there was improvement, it may have been more evident to the experimenters. However, it should be remembered that the PI hypothesized that a placebo effect would occur. In the future, to assure total objectivity, the assessment, treatments, and attention placebo should be given by separate experimental collaborators. Unfortunately, both financial and temporal logistics did not allow for this methodological safe-guard of internal validity. However, it should be remembered that the pretests, posttest, and treatments were given in completely random fashion in an attempt to control for this possible extraneous variable. The PI believes that although not ideal this randomization of experimenter roles was sufficient to maintain experimental integrity.

Implication for This Study

The support of the effectiveness of CES in a CD population during substance abuse treatment has several implications. The chemically dependent individual often avoids detoxification and treatment because of the fear of withdrawal syndrome symptoms, not the least of which are anxiety and depression (Allen & Frances, 1986; Ross & Glaser, 1989). Several previous CES studies have demonstrated the amelioration of some physical symptoms of withdrawal especially among the opiate addicted subgroup of the CD population

(Gomez and Mikhial, 1978; Patterson, 1976; Patterson et al., 1984; Wen and Cheng, 1973). The availability of a treatment such as CES that helps to alleviate withdrawal syndrome with its concomitant primary psychophysiological and secondary affective components of withdrawal might prove to be a powerful inducement to entice the addict into treatment once the CD population became aware of its existence. It should be emphasized that the PI does not see CES as a recovery treatment for chemical dependency itself. Only a proactive comprehensive therapeutic intervention into the biopsychosocial aspects of addiction can approach this need. Rather, CES is seen as an addendum to the detoxification and treatment stages of chemical dependence recovery.

Any inducement into treatment for the type of population studied in this research seems valuable. The trend in substance abuse is moving away from recreational use to more of the "hard core" addicted population of severely compromised addicts. This trend poses an increase in concomitant social and legal problems for our culture and others throughout the world. CES treatments for the chemically dependent have been investigated for some time, with for the most part mixed results. The results of the current study are not totally conclusive with respect to the improvement of anxiety and depression because of differential results between self-report and observer-ratings. However, this study has contributed something from a methodological and theoretical perspective to help explain some findings of previous studies and to benefit future research. Should the proper use of CES prove to be beneficial for other populations suffering from affective disorders it might be considered as an addendum to other psychotherapy techniques that have proven efficacious to the relief of affective disorders such as behavioral, cognitive, and interpersonal interventions. The

addition of CES to the aforementioned interventions might accelerate the ameliorative process of these therapies.

Suggestions for Future Research

Based on the current research and possible explanations of the results, the following suggestions are offered for any future research in this area:

1. In the past, CES treatments have been given for varying durations, intervals, and electrical frequencies with mixed results. Future research might investigate the use of CES at frequencies other than 100 Hz and for longer periods than the 45 minutes used in this experiment. In addition, it might be informative to investigate the efficacy of CES at a set amplitude of electrical current, because in this experiment the amplitude varied as part of the blinding procedure. For future research, Taylor (1991) offers a method for calibrating the threshold of tactile sensation that may allow for a constant amplitude of current at least among individual subjects. However, this method limits the maximum range of amplitude available for research and is idiosyncratic to each subject. Another, perhaps better, alternative would be to develop a device that would mimic the "tingling " sensation felt during CES treatments above the sensation threshold level. This would allow for the use of an active placebo among the sham group subjects and provide consistent treatment sensation within the active CES group and between the sham and active groups. This procedure would allow the frequency to be set at whatever level of amplitude the researcher wished to study.

2. Because of the nature of the population studied, the effects that substances of abuse had on the results of the study are difficult to determine. It is possible to find CD

subjects that use only one substance, although in the PI's research and clinical experience it is difficult to isolate such a subgroup in today's treatment population. This observation is supported by recent epidemiological studies of substance use. It may be possible, however, to isolate and maintain the integrity of such a sample from a potential subject pool by using UA. There may be an easily identifiable group that is singular in their drug use that would use alternative drugs of the same class. Specifically, this group would be composed of opiate addicts on methadone maintenance. Although clinically this group is prone to relapse into illicit drug use, when this occurs it is typically with other opiates. To guard against those in this subclass that might use other drugs periodic UA screens would be necessary. The limitation of either of these methods discussed above (1 & 2) would be the expenditure of time and financial resource because of the logistics involved. Therefore, it is suggested that any such research be well funded.

3. It was impossible to determine fully the effects that severity of addiction had on this study because of the use of UA screens as opposed to fully quantified UA. It is suggested that any further research include a fully quantified UA taken at the time of subject recruitment. If this proposal proves to be to difficult logistically or financially for the future investigation, the use of the ASI (McLellan et al., 1980) is suggested. This inventory has both observer-ratings and self-report as parts of its information base. Thus, if one is concerned about the veracity of the subject's report one can look for a convergence correlation between the two aspects of the scale. Further, several reliability and validity studies are available showing the viability of the inventory. Unfortunately for the researchers with budget constraints, the training needed to use ASI properly is rather lengthy and expensive.

4. The differential results of this study based on the use of self-report and observer-rating outcome measures poses several research opportunities in the area of assessment among the CD population. As mentioned, several authors have discussed the phenomenon that the Hamilton based scales such as those used in this study seem to show clinical change sooner in the course of treatment than do the Beck scales. However, it has also been shown that toward the end of treatment the scales converge. It is recommended for future CES treatment research that the treatment or at least the period between pretest and final posttest be extended to one month if both these scales are used as outcome measures in the same experiment. It is generally recognized in clinical circles that at least one month of treatment is necessary to complete a course of substance abuse intervention. It may be that at the end of this period the outcome measures would converge. If this were to occur it would give credence to the proposal that the Hamilton scales are more sensitive to certain aspects of clinical change, and/or that there is a latency period between the clinician's sense of clinical change regarding mood disorder and the patient's sense of emotional well-being. If there is no convergence between the scales, this finding may give strength to the arguments that in some way the observer-rater is influenced toward a halo effect, or that self-report truly is inappropriate with the CD population due to a particular lack of veracity regarding self-report.

5. Because of financial and logistic consideration, it was necessary for the researchers themselves to provide treatment, daily attention placebo controls, and administer the outcome measure assessments. It may have been that the observation of the clinical progress of the patients on a daily basis skewed the objectivity of the observer-rating.

Although an attempt to control for this possibility was done with the random treatment and assessment assignment between experimenters. To insure the above situation does not present a research confound and that the experimenter/subject interaction potentials are reduced, completely different individuals should give the assessments, treatments, and attention placebo controls.

Concluding Remarks

While the results of the current study are interesting, as with many previous CES studies, the results are mixed. A review of the literature shows that during the last few decades CES studies became rather faddish. One gets the impression that many of the researchers were engrossed with an enthusiasm driven by the idea that a real panacea may had been discovered. Through the years there have always been claims by clinicians, patients, and researchers of CES's efficacy especially in the area of substance abuse. This may have lead to rather loose methodological procedures in the study of its effects, which became a basis for criticism of the true utility of this treatment. After an attempt to study the effects of this treatment, the PI can better appreciate the difficulty faced by his predecessors, especially in the area of CES's application to a substance abuse population. This is a difficult population to study for the reasons pointed out above. In particular, use of self-report with this population seems questionable. This opinion comes from the thoughts of clinicians in the field, and many of the authors who have discussed the situation, as well as from the small contribution of this study. The main reasons given for this conclusion include organic dysfunction, the characterological compromise of much of this population, and the confused

cognitive state of the individual going through withdrawals. Whatever the reasons, the results of this study appear to confirm this opinion. As to the effectiveness of CES to ameliorate some affective distress with this population, the PI believes the results have demonstrated good reason to believe it can be helpful. This method of treatment could make substance abuse treatment a more attractive alternative to the addict who is concerned about the discomfort and distress of withdrawal. This appears to be one of the major issues that keeps the addict out of treatment. If CES can successfully be used in this regard, it may present a non-chemical alternative to the negative effects anxiety and depression play in the recovery process.

CES could be used not only as an intervention in the initial treatment of substance abuse recovery, but also as a tool in the aftercare process to help prevent substance abuse relapse in the chemically dependent population. Once the addict has gone through treatment and is well into recovery CES treatments may provide an intervention to alleviate the anxiety and depression that often follow the initial adjustment to an abstinent life-style. Often anxiety and depression play a sizable role in relapse into substance use for the recovering addict. The addict's method of coping with anxiety and/or depression generally has been the use of chemicals to alter his/her affective state. Until the recovering addict has learned alternative coping skills such as stress management, stress inoculation and the identification of relapse triggers including various manifestations of anxiety and depression, CES might offer a non-chemical alternative to ameliorate these negative emotional states.

More research in the area of CES treatment should be carried out to carefully study the most effective amplitude dose, duration of treatment, and optimum number of treatments

for the various disorders to which CES might be applied. Such studies should be well staffed and funded, particularly in the if the research is to be done in the area of chemical dependency for reasons as stated above relating to the maintenance of experimental integrity and the use of instruments to determine the severity of addiction and its role in CES treatment. Further, more research might be considered to determine the best measurements for evaluating this treatment among the specific populations of interest to which CES might be used as an intervention. Hopefully, in some small way, this study has added some knowledge to this area of research and poses questions that will be useful in future research. In particular, this study provides evidence that CES treatment deserves further serious consideration since its effects cannot be accounted for as mere placebo effects. The current study supports the investigations that have shown CES to be an effective treatment in the area of chemically dependency, albeit it at times these studies have exhibited mixed results and less than ideal methodologies. Thus, the finding of this study warrants further serious investigation of this treatment modality, especially regarding the possible mechanisms involved in CES's ability to affect clinical change in substance withdrawal symptomatology.

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APPENDIX A

Appendix A

Meta-analysis Variables

Level I

___ Study number
___ Blind study
1. single
2. double
3. none

___ Screening for psychotics

___ Total males entering therapy
___ Total males completing therapy

___ Total females in therapy
___ Total females completing therapy

___ Total N in therapy
___ Total N completing therapy

___ Mean age

___ Patient status
1. inpatient
2. outpatient

___ Diagnosis
1. alcoholism
2. drug abuse
3. polysubstance abuse

___ Substance abused (type of problem)
1. alcohol
2. opiate
3. stimulant
4. sedative
5. tranquilizer/hypnotic
6. mixed (any two or more)

___ Symptom duration--mean years.

___ Focus of study
1. withdrawal symptom reduction (somatic/sleep)
2. anxiety
3. depression
4. cognitive dysfunction

5. combined (any two or more)

___ Assignment to groups
1. random
2. failed randomization
3. matched and random assignment
4. post hoc matched
5. nonrandom
6. not applicable
7. missing

Level II

___ Study number

___ Type of comparison
1. treatment vs. control
2. within group

___ Type of CES Tax
1. CES alone
2. CES + meads.
3. CES + psychotherapy
4. CES + meads. + psychotherapy

___ Type of control
1. Sham
2. Wait-list
3. Usual therapy minus CES
4. No control

___ Per Tax GN
___ Post Tax GN
___ Per CN
___ Post CN

___ Sensation of electrical impulse Y/N

___ Sensation of electrical impulse
1. Among active treatment
2. Among sham

___ Subject set Amp level by sensation Y/N

___ Amps. Range
1. 0 mA
2. 0-1.5 mA
3. 1.5-2.5 mA
4. 2.5+ mA

___ Frequency
1. 100 Hz
2. 125 Hz
3. 125+ Hz

___ Pulse duration

___ Peak pulse

___ Electrode placement
1. mastoid process stethoscope
2. frontal/occipital headband
3. Combined
4. Acupuncture needles with electrode

___ Length of session mins.

___ Number of sessions

___ Number of sessions-days

___ Number of sessions-weeks

___ Attrition

Level III

___ Study number
___ Effect number

___Therapist blind to Tax

___ Point of assessment
1. post Tax
2. follow-up

___ Time of assessment--weeks

___ Content of outcome
1. Anxiety questionnaire
2. Depression questionnaire
3. Self-report of somatic withdrawal symptoms
4. Symptom inventories withdrawal symptoms by therapist
5. Sleeplessness
6. Cognitive function (I.Q.)
7. Personality
8. Global improvement
9. Reduction of substance intake
10. Psychomotor

11. Miscellaneous

___ Source of outcome measure
1. Client self-report
2. Therapist rating
3. Independent observer rating
4. Physiological measure
5. Cognitive function measure
6. Behavioral measure
7. Laboratory task
8. Amount of drug intake reduced

___ Source of effect size
1. Means and standard deviations
2. t-test; oneway ANOVA; df(num)-1
3. One way ANOVA; df(num)>1
4. Repeated measure ANOVA
5. p level
6. No control - per Tax SD
7. No control - post Tax SD
8. difference reported as nonsignificant

___ Degrees of freedom for error term

___ Effect size--SD units

___ Subscale used Y/N

___ Total score used Y/N

___ Means estimated from graph (Y=1; N=2)

___ Rater blind to Tax (Y=1; N=2)

APPENDIX B

Appendix B

Medically Supervised Detoxification Withdrawal Protocols for TRMC and 12 & 12 Treatment Centers

Group 1. - For all patients

1. Lindane shower upon admission.
2. Regular diet or diet as tolerated.
3. Thiamine 100 mg.- 1 tablet po daily.
4. Therageneric + M - 1 tablet po daily.
5. Tylenol 325 mg. - 2 tablets q4 hr. for pain, as needed.
6. Iburprophen 200 mg. - if needed 3 tablets q4 hr, as needed....if Tylenol does not help.
7. Maalox (30 cc) po q4 hr. pm - indigestion, as needed.
8. Milk of Magnesia (30 cc) po 4q hr. - constipation, as needed.
9. Kaopectate - for diarrhea, as needed
10. Benadryl 25 mg. - 1 capsule po 3 times daily for restlessness and itching, as needed
11. Clonidine 0.1 mg. - po 2 times daily if systolic BP > 180 or diastolic > 100.
12. Lomotil 2.5 mg. - tablet 1 po to start, then 1 q4 hr. for diarrhea, as needed.
13. Hydroxyzyine 25 mg. - capsule 1 po q4 hr. pm for nausea/vomiting.
14. Robutussin 2 tsp. - q4 hr not to exceed 6 doses in 24 hr. for cough, as needed.
15. Phenergan 50 mg. I.M.- as ordered.
16. Phenergan "R" suppository - insert 1 q6-8 hr. pm n/v, as needed.
17. Sudafed 30 mg. po 2 tablets every 4-6 hrs. not exceed 8 tablets in 24 hrs, as needed
18. Emetrol - pm as directed for c/o nausea.
19. Auralgan Ear Drops - 4 gtts Tid of Qid pm., as needed.

Group II. - For all patients with alcohol abuse (either dose order, as ordered)

1. Librium 25 mg. to 50mg. - po q6 for obvious withdrawal signs and symptoms X 3 days.
2. Librium 25 mg. to 50mg. - po q4 for severe withdrawal signs and symptoms X 3 days.

Group III. - For patients with other drug abuse

1. Ativan 2.0 mg - 1 tab po 2 or 3 X daily for 3 days.

Group IV. - For patients with a history of seizures on alcohol withdrawal epilepsy, or patients who have been abusing benzodiazepines or barbiturates.

1. Dilantin 100 mg. - po 3 X daily for complete stay in detoxification.
2. Dilantin 100 mg. - po c phenobarbital 15 mg po Tid for complete stay in detoxification., given only as ordered by doctor.
3. Mg SO_4 50% solution - 2cc I. M. X 3 days as needed for alcohol withdrawal.

APPENDIX C

Appendix C

CRANIAL ELECTROTHERAPY STIMULATION PROTOCOL
INFORMED CONSENT

I, _____, have been asked to participate in a clinical study entitled:

A PLACEBO-CONTROLLED, DOUBLE-BLIND, RANDOMIZED, COMPARISON OF CRANIAL ELECTROTHERAPY STIMULATION (CES) PLUS STANDARDS SUBSTANCE ABUSE TREATMENT WITH SIMULATED CES PLUS STANDARD SUBSTANCE ABUSE TREATMENT IN SUBSTANCE DEPENDENT PATIENTS.

I understand the following:

A. Terms of the Study.

This study is being conducted for research purposes to compare the safe and effective use of cranial electrostimulation (CES) used in conjunction with the institution's standard treatment for substance dependency, with a simulated CES treatment used in conjunction with the institution's standard treatment for substance dependency. The CES devise used in this study, the LB2000, is an electrical device approved by the Food and Drug Administration (FDA) for the treatment of anxiety, insomnia and depression, symptoms which are common among alcohol and drug dependent patients.

I understand that I am volunteering to participate in the study that will involve my participation for at least two weeks but no more than four weeks during my hospital stay. The purpose of the study is to see if CES is useful in the treatment for substance dependence among patients seeking medical treatment at this institution.

I understand that I will continue in all phases of the normal treatment program of this institution while participating in this study and I can decline to participate now or terminate my participation at any time later without prejudice of my treatment in any way.

I understand that patients will be assigned to various study groups in a random and unbiased fashion. Patients will be assigned to the study groups in order of their admission to the hospital. Each third patient will not be assigned to use CES and will be designated a control subject. Patients not volunteering for the study will not be used as controls.

The study will be "double blind" which means that some devices will provide minimal or no stimulation. "Double-blind" means that neither the patient nor the therapist knows which devices will actually provide CES treatment or alternatively provide only simulated CES treatment. This is to ensure that there is no bias in the study.

I understand that after preliminary and psychological testing each patient will be given actual or simulated CES treatments daily for 45 minutes, with 15 treatments anticipated. I further understand that after this treatment period I will be retested on the original psychological tests. This study will continue until at least 60 patients have completed their participation.

Following the study the results will be made available to any participant who requests them. Patients receiving simulated treatment will be offered regular CES treatment following the study.

B. Risks or Discomforts to the Subjects.

I understand in this study I may receive a placebo treatment or CES treatment in addition to the standard treatment for dependence. A placebo treatment is not an active CES treatment. I understand that the LB2000, a CES device, puts out a very small electrical burst of energy which has as its source 9 volt batteries. After an initial tingle behind the ear, I will not feel the electrical stimulation during the study. Devices similar to the LB2000 have been used in Europe since the early 1950's and in the Far East since the late 1950's. Physicians in the United States began using devices similar to the LB2000 in the early 1970's with increased use through the 1980's and 1990's. Thousands of patients have received this kind of treatment with no reports of any harmful effects.

I understand that I may experience some unknown side effects. Although, the probability is extremely low, based on the evidence of all previous studies. I agree that I have been fully informed of the potential side effects and that my questions in regard to these possible side effects have been answered to my satisfaction. I understand that I may be taking medications which react negatively with the purposes of this study and that I may be asked to discontinue these medications, which can usually be done without any risks. However, if my health worsens as the result of any discontinuation of medication I will be removed from the study and allowed to resume prior medications.

I understand that in the event physical injury, disability, or illness resulting from participation in this research no compensation will be available from the treatment center or any individual involved in this study, and that I must obtain any necessary medical care in the same manner in which I obtained any other medical care, i.e., through my person physician.

C. Benefits.

I understand that similar devices have been previously approved for use in persons with symptoms common in alcohol and drug dependent patients (i.e., anxiety, insomnia and depression). I understand that negative effects have not been reported by patients being treated with CES devices similar to the LB2000.

I understand that all examinations, tests, and assessment procedures required by this study are provided free of charge.

Short term and long term benefits of CES in regard to dependency have been described in previous studies. The use of CES may be helpful in the management of your chemical dependence.

D Available Alternative Courses of Treatment.

I understand that there are several alternative treatments for dependency which include several other CES devices. A decision not to participate in this study will not influence the quality or availability of medical care and psychological care that I would normally receive from Dr. Cody and this institution.

E. Confidentiality of Records.

I understand that all study data and medical records will be considered confidential. However, appropriate representatives of Life Balance Int., Inc. and the University of Tulsa may inspect my records. In order to meet the obligations of Federal law, you understand that my records may be subject to review by the Food and Drug Administration (FDA). I also understand that I will not be identified by name in any publication(s) relating this research.

F. Medical Treatment and Costs.

Emergency medical treatment will be provided without charge to me by the institution's doctors in the event of an injury which is documented to be study-related. No other compensation will be provided. However, this does not constitute a waiver of any rights I may otherwise have under federal or state laws or regulations.

G. Available Information.

If I have any questions or desire further information with respect to this study, the availability of medical care, or if I experience a study-related injury, I may contact:

Dr. Mary Ellen O'Connor
Department of Psychology
The University of Tulsa
Telephone (918) 631-2248

If I have any questions concerning my rights as a research subject, I may contact:

Dr. Dan H. Fieker, D.O., Chairman
Institutional Review Board
Tulsa Regional Medical Center
Telephone (918) 587-2561

D Available Alternative Courses of Treatment.

I understand that there are several alternative treatments for dependency which include several other CES devices. A decision not to participate in this study will not influence the quality or availability of medical care and psychological care that I would normally receive from Dr. Lyons and this institution.

E. Confidentiality of Records.

I understand that all study data and medical records will be considered confidential. However, appropriate representatives of Life Balance Int., Inc. and the University of Tulsa may inspect my records. In order to meet the obligations of Federal law, you understand that my records may be subject to review by the Food and Drug Administration (FDA). I also understand that I will not be identified by name in any publication(s) relating this research.

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Dr. Mary Ellen O'Connor
Department of Psychology
The University of Tulsa
Telephone (918) 631-2248

If I have any questions concerning my rights as a research subject, I may contact:

Mr. Jerry Carlton
Executive Vice President
12 & 12 Transition House, Inc.
12 E. 12th Street
Tulsa, OK. 74119
Telephone (918) 584-1212

H. Termination.

I may discontinue your participation at any time during the study, without penalty or loss of benefits to which you are otherwise entitled. If I choose to discontinue my participation in this study I will continue to be given the standard treatment for dependency that is normally provided by this institution. No prejudice will be shown me for my medical care or future participation in research studies.

In addition, my participation may be terminated by the physician if I violate the study plan, need additional medication, experience a study-related injury or for administration reasons.

Upon withdrawal from the study I agree to return to the research any devices provided for this study.

I. Significant New Findings.

Any new important information that develops during the course of the study which may influence my willingness to continue participation in this study will be made available to me.

I, _____voluntarily consent to participate in this clinical study.

Subject:_____
Signature Date

Researcher:_____
Signature Date

Witness:_____
Signature Date

APPENDIX D

Appendix D

SEMI-STRUCTURED INTERVIEW FOR CES DATA COLLECTION

DAILY***TEN TO FIFTEEN MINUTE INTERVIEW***

***Structure the interview such that the order of the questions are varied between interviews. Use sundry wording while maintaining the key thrust of the question (do not vary meaning of underlined words). Substitute day, since yesterday, since I saw you last, since we last meet, ect. for phrases containing the words 24 hours. Do not allow interviews to become routine or monotonous. Allow no less than ten and no more than fifteen minutes to elapse during the interview. Allow approximately one minute per question.

1. How have you been feeling since in the last 24 hours.
   - A. How much of the time in the last 24 hours have you felt this way?
2. Have you had any physical discomfort in the last 24 hours?
   - A. If yes: what are the symptoms and for how long?
3. Have you had any craving for drugs in the last 24 hours?
   - A. If yes: how often and for what length of time before the craving stopped?

   Please describe your mood in the last 24 hours.
   - A. Try to the get subject to be as specific as possible in describing his state of affect.
5. Have you been worried about anything in the last 24 hours?
   - A. If yes: tell me what you've been worried about and how much of last day have you worried.
6. Have you felt self-critical or down on yourself in the last 24 hours?
   - A. If yes: how much of the time?
   - B. If not, have you been feeling particular good about yourself?
     (1). If yes: how much of the time.
7. Have you felt angry at anybody in the last 24 hours?
   - A. If yes: tell me what happened and how long you were angry.
8. Do you feel anyone has been giving you a bad time or working against you in the last 24 hours.
   - A. If yes: tell me about it.
9. Have you had any problems with concentration or memory in the last 24 hours?
   - A. If yes: tell me what problems you had and how much of the time it was a problem.
10. Have you had any unusual or special experiences or thoughts in the last 24 hours?

A. If yes: tell me about them.

11. During the last 24 hours has there been anything that happened to you that was particularly pleasing or upsetting.

A. If yes: tell me about it.

12. Note:***Ten to fifteen minute interview***

Is there anything else you would like to talk about?

***Ask the above question only if ten to fifteen minutes have not elapsed since the beginning of the interview.

13. ***Politely end the conversation after ten to fifteen minutes have elapsed.

DAILY BPRS CHECKLIST FOR CES CD SUBJECTS

1. APPEARANCE:

Grooming-- 1) hair combed; 2) shaved for males, make-up for females. Clean-- 3) clothes unstained and unsoiled. Neat-- 4) clothes unwrinkled and appropriately worn. Personal hygiene-- 5) body clean, 6) no noticeable body or breath odor.

0.= Not present: 6 of 6
1.= Very mild: 5 of 6
2.= Mild 4 of 6
3.= Moderate 3 of 6
4.= Moderately Severe 2 of 6
5.= Several of 6
6.= Very Severe 0 of 6

2. DRUG CRAVING:

Patient expresses having felt a craving or desire for the ingestion of chemically dependent substances. Base ratings only on the duration of drug craving that is self-reported by the patient. Do not rate on basis of the intensity of drug craving or report by the staff.

0.= Not Present.
1.= 0 - 2 hours
2.= 2 - 6 hours
3.= 6 - 10 hours
4.= 10 - 12 hours
5.= 12 - 14 hours
6.= 14+

3. WITHDRAWAL SYMPTOMS:

Any physical symptoms relating to withdrawal (see symptom checklist) by patients self-report. Do not rate on the basis of intensity of withdrawal symptoms reported by the staff.

0.= Not present.
1.= 0 - 2 hours
2.= 2 - 6 hours
3.= 6 - 10 hours
4.= 10 - 12 hours
5.= 12 - 14 hours
6.= 14+

4. SLEEP DISTURBANCE:
1) early insomnia. 2) middle insomnia 3) late insomnia 4) leaves bed (except to void). 5) nightmares 6) night terrors.

0.= Not present.
1.= Very mild: 1 of 6
2.= Mild: 2 of 6
3.= Moderate: 3 of 6
4.= Moderately severe: 4 of 6
5.= Severe: 5 of 6
6.= Very severe 6 of 6
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness or cannot be assessed

5. ANXIOUS MOOD:
Worry, fear, or overconcern for present or future. Rate solely on the basis of verbal report of the patient's own subjective experiences pertaining to the past 24 hours. Do not infer anxiety from physical signs, symptoms or from neurotic defense mechanisms. Do not rate on a basis that is restricted to somatic concerns.

0.= Not present.
1.= Very mild: occasionally felt somewhat anxious.
2.= Mild: occasionally felt moderately anxious, or often felt somewhat anxious.
3.= Moderate: occasionally felt very anxious, or often felt moderately anxious
4.= Moderately severe: often felt very anxious
5.= Severe: felt very anxious most of the time
6.= Severe: felt very anxious nearly all of the time.
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness, or not assessed.

6. DEPRESSED MOOD:
Subjective report of feeling depressed, blue, "down in the dumps," etc. Rate only degree of reported depression. Do not rate on the basis of inferences concerning depression based upon general retardation and somatic complaints. Rate on the basis of reported (i.e. subjective) information pertaining to the past 24 hours.

0.= Not present.
1.= Very Mild: occasionally felt somewhat depressed.
2.= Mild: occasionally felt moderately depressed or often felt

somewhat depressed.
3.= Moderate: occasionally felt very depressed or often felt moderately depressed.
4.= Moderately severe: often felt very depressed.
5.= Severe: felt very depressed most of the time.
6.= Very severe: felt very depressed nearly all of the time.

7. UNCOOPERATIVENESS:
Evidence of resistance, unfriendliness, resentment, and a lack of readiness to cooperate with the interviewer. Rate only on the basis of the patient's attitude and responses to the interviewer and the interview situation. Do not rate on the basis of reported resentment or uncooperativeness outside the interview situation.

0.= Not present.
1.= Very mild: e.g. does not seem motivated.
2.= Mild: e.g. seems evasive in certain areas.
3.= Moderate: e.g. monosyllabic, fails to elaborate spontaneously, somewhat unfriendly.
4.= Moderately severe: e.g. monosyllabic, fails to elaborate spontaneously, quite unfriendly and/or refuses to answer questions occasionally.
5.= Severe: e.g. refuses to answer a number of questions.
6.= Very severe: e.g. refuses to answer most questions.

8. COGNITIVE DYSFUNCTION:
Difficulty or dysfunction with memory and/or concentration, distinguish from DISORIENTATION.

0.= Not present.
1.= Very mild: occasionally felt somewhat dysfunctional.
2.= Mild: occasionally felt moderately dysfunctional, or often felt somewhat dysfunctional.
3.= Moderate: occasionally felt very dysfunctional, or often felt moderately dysfunctional.
4.= Moderately severe: often felt very dysfunctional, or often felt moderately dysfunctional.
5.= Severe: felt very dysfunctional for 8 - 12 waking hours of the last 24 hours.
6.= Very severe: felt very dysfunctional for 12+ waking hours, all of the last 24 hours.
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardednes or not assessed.

9. EXCITED AFFECT:
Heightened emotional tone, including irritability and expansiveness (hypomanic affect). Do not infer affect from statements of grandiose delusions. Rate based on observations made during interview.

0.= Not present.
1.= Very mild and of doubtful clinical significance.
2.= Mild: e.g. irritable or expansive at times during the interview.
3.= Moderate: e.g. constantly irritable or expansive.
4.= Moderately severe: e.g. constantly irritable or expansive, or at times, enraged or euphoric.
5.= Severe: e.g. enraged or euphoric throughout most of the interview.
6.= Very severe: e.g. as above, but to such a degree that the interview must be terminated prematurely

10. BLUNTED AFFECT:
Diminished affective responsiveness, as characterized by deficits in facial expression, body gesture, and voice pattern. Distinguish from EMOTIONAL WITHDRAWAL in which the focus is on interpersonal impairment rather than affect. Consider degree and consistency of impairment. Rate based on observations made during interview.

0.= Not present.
1.= Very mild.
2.= Mild: e.g. somewhat diminished facial expression or somewhat monotonous voice or somewhat restricted gestures.
3.= Moderate: e.g. as above, but more intense, prolonged, or frequent.
4.= Moderately severe: e.g. flattening of affect, including at least two of the three features; severe lack of facial expression, monotonous voice, or restricted body movements.
5.= Severe: e.g. profound flattening of affect.
6.= Very severe: e.g. totally monotonous voice, and lack of expressive gestures throughout the evaluation.

11. PSYCHOMOTOR AGITATION:
Rate motor restlessness (agitation) observed during the interview. DO NOT rate on the basis of subjective experiences reported by the patient. Disregard suspected pathogenesis (e.g. tardive dyskinesia).

0.= Not present.
1.= Very mild: occasionally fidgets.
2.= Mild: e.g. frequently fidgets.
3.= Moderate: constantly fidgets, or frequently fidgets, wrings, hands,

and pulls clothing.

4.= Moderately severe: e.g. constantly fidgets, wrings hands and, pulls clothing.

5.= Severe: e.g. cannot remain seated (i.e. must pace).

6.= Very severe: e.g. paces in a frantic manner.

12. PSYCHOMOTOR RETARDATION:

Reduction in energy level evidenced in slow movements. Rate on the basis of observed behavior of the patient only. Do not rate on the basis of the patient's subjective impression of his or her own energy level.

0.= Not present.

1.= Very mild and of doubtful clinical significance

2.= Mild: e.g. conversation is somewhat retarded, movements somewhat slowed.

3.= Moderate: e.g. conversation is noticeably retarded but not strained.

4.= Moderately severe: e.g. conversation is strained, moves, very slowly.

5.= Severe: e.g. conversation is difficult to maintain, hardly moves at all.

6.= Very severe: e.g. conversation is almost impossible, does not move at all throughout the interview.

SEMI-STRUCTURED INTERVIEW FOR CES DATA COLLECTION

*****PER/POST DETOX/CES TREATMENT*****
*****TEN TO FIFTEEN MINUTE INTERVIEW*****

NAME_____ PATIENT NO._____

1. How have you been felling since we meet a week ago?
   A. How much of the time in the last week have you felt this way?
2. Have you had any physical discomfort since I saw you a week (or so) ago?
   A. If yes what are the symptoms and for how long?
3. Have you had any desire for drugs in the last week ?
   A. If yes, how often and for what length of time before the craving stopped?

Please describe your mood in last week.

   A. Try to the get subject to be as specific as possible in describing his state of affect.

5. Have you been worried about anything during the week (or so)?
   A. If yes, tell me what you've been worried about and how much of last day have you worried.
6. Have you felt self-critical or down on yourself in the last week (or so)?
   A. If yes, how much of the time?
   B. If not, have you been feeling particular good about yourself?
      (1). If yes, how much of the time.
7. Have you felt angry at anybody since we meet a week (or so) ago?
   A. If yes, tell me what happened and how long you were angry.
8. Do you feel anyone on the unit has been giving you a bad time or working against you in the last week (or so)?
   A. If yes, tell me about it.
9. Have you had any problems with concentration or memory in the last week (or so) ?
   A. If yes, tell me what problems you had and how much of the time it was a problem.
10. Have you had any unusual or special experiences or thoughts since we last meet a week (or so) ago?
    A. If yes, tell me about them.
11. Since we talked a week (or so) ago has there been anything that has happened to you that was particularly pleasing or upsetting.
    A. If yes, tell me about it.

12. Note:***Ten to Fifteen minute interview***

Is there anything else you would like to talk about?

***Ask the above question only if ten minutes have not elapsed since the beginning of the interview.

13. ***Politely end the conversation after ten to fifteen minutes have elapsed.

WEEKLY BPRS CHECKLIST FOR CES CD SUBJECTS

NAME_____ PATIENT NO._____

DATE__/__/__

1. APPEARANCE:

Grooming-- 1) hair combed; 2) shaved for males, make-up for females. Clean-- 3) clothes unstained and unsoiled. Neat-- 4) clothes unwrinkled and appropriately worn. Personal hygiene-- 5) body clean, 6) no noticeable body or breath odor.

0.= Not present: 6 of 6
1.= Very mild: 5 of 6
2.= Mild 4 of 6
3.= Moderate 3 of 6
4.= Moderately Severe 2 of 6
5.= Several of 6
6.= Very Severe 0 of 6

2. DRUG CRAVING:

Patient expresses having felt a craving or desire for the ingestion of chemically dependent substances. Base ratings only on the duration of drug craving that is self-reported by the patient. Do not rate on basis of the intensity of drug craving or as reported by the staff.

0.= None
1.= 1 day
2.= 2 days
3.= 3 days
4.= 4 days
5.= 5 days
6.= 6+ days

3. WITHDRAWAL SYMPTOMS:

Any physical symptoms relating to withdrawal (see symptom checklist) by patients self-report. Do not rate on the basis of intensity of withdrawal symptoms as reported by the staff.

0.= None
1.= 1 day
2.= 2 days
3.= 3 days
4.= 4 days
5.= 5 days

6.= 6+ days

4. SOMATIC CONCERNS:
Degree of concern over present bodily health. Rate the degree to which physical health is perceived as a problem by the patient, whether complaints have a realistic basis or not. Do not rate merely on reports of somatic symptoms as with withdrawal symptoms. Rate only concern for (or worrying about) physical problems (real or imagined). Rate on the basis of reported (i.e. subjective) information pertaining to the last week.

0.= Not present.
1.= Very mild: occasionally is somewhat concerned, or often is somewhat concerned about body, symptoms, or physical illness.
2.= Mild: occasionally is moderately concerned, or often is somewhat concerned.
3.= Moderate: occasionally is very concerned, or often is moderately concerned.
4.= Moderately Severe: often is very concerned.
5.= Severe: is very concerned most of the time.
6.= Very severe: is very concerned nearly all of the time.
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness, or not assessed.

5. SLEEP DISTURBANCE:
1) early insomnia. 2) middle insomnia 3) late insomnia 4) leaves bed (except to void). 5) nightmares 6) night terrors.

0.= Not present.
1.= Very mild: 1 of 6
2.= Mild: 2 of 6
3.= Moderate: 3 of 6
4.= Moderately severe: 4 of 6
5.= Severe: 5 of 6
6.= Very severe 6 of 6
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness, or not assessed.

6. ANXIOUS MOOD:
Worry, fear, or overconcern for present or future. Rate solely on the basis of verbal report of the patient's own subjective experiences pertaining to the past week. Do not infer anxiety from physical signs, symptoms or from neurotic defense mechanisms. Do not rate on basis that is restricted to somatic concern.

0.= Not present.
1.= Very mild: occasionally felt somewhat anxious.
2.= Mild: occasionally felt moderately anxious, or often felt somewhat anxious.

3.= Moderate: occasionally felt very anxious, or often felt moderately anxious.
4.= Moderately severe: often felt very anxious.
5.= Severe: felt very anxious most of the time.
6.= Very Severe: felt very anxious nearly of the time.
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness, or not assessed.

7. DEPRESSED MOOD:
Subjective report of feeling depressed, blue, "down in the dumps," etc. Rate only degree of reported depression. Do not rate on the basis of inferences concerning depression based upon general retardation and somatic complaints. Rate on the basis of reported (i.e. subjective) information pertaining to the past week.

0.= Not present.
1.= Very Mild: occasionally felt somewhat depressed.
2.= Mild: occasionally felt moderately depressed or often felt somewhat depressed.
3.= Moderate: occasionally felt very depressed or often felt moderately depressed.
4.= Moderately severe: often felt very depressed.
5.= Severe: felt very depressed most of the time.
6.= Very severe: felt very depressed nearly all of the time.
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness, or not assessed.

8. GUILT FEELINGS:
Overconcern or remorse for past behavior. Rate on the basis of the patient's subjective experiences or guilt as evidenced by the patient's verbal report pertaining to the last week. Do not infer guilt feelings from depression, anxiety, or neurotic defenses.

0.= Not present.
1.= Very mild: occasionally felt somewhat guilty.
2.= Mild: occasionally felt moderately guilty, or often felt somewhat guilty.
3.= Moderate: occasionally felt very guilty, or often felt moderately guilty.
4.= Moderately severe: often felt very guilty
5.= Severe: felt very guilty most of the time, or encapsulated delusion(s) of guilt.
6.= Very severe: agonizingly and constantly felt guilty, or pervasive delusion(s) of guilt.
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness, or not assessed.

9. GRANDIOSITY:
Inflated self-esteem (self-confidence) or inflated appraisal of one's talents, powers, abilities, accomplishments, knowledge, importance, or identity. Do not score mere grandiose quality of claims (e.g. "I'm the worst sinner in the world," "The entire country is trying to kill me.")

unless the guilt/persecution is related to some special exaggerated attributes of the individual. Also the patient must claim exaggerated attributes, e.g. if patient denies talents, powers, etc., even if he or she states that others indicate that he/she has these attributes, this item should not be scored. Rate on the basis of reported (i.e. subjective) information pertaining to the past week.

0.= Not present.
1.= Very mild: e.g. is more confident than most people, but of only possible clinical significance.
2.= Mild: e.g. definitely inflated self-esteem or exaggerates talents somewhat out of proportion to the circumstances.
3.= Moderate: e.g. inflated self-esteem clearly out of proportion to the circumstances, or suspected grandiose delusions.
4.= Moderately severe: e.g. a single (definite) encapsulated grandiose delusion, or multiple (definite) fragmentary grandiose delusions.
5.= Severe: e.g. a single (definite) grandiose delusion/delusional system, or multiple (definite) grandiose delusions with which the patient seems preoccupied.
6.= Very severe: e.g. as above, but nearly all conversation is directed toward the patient's grandiose delusion(s).
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness, or not assessed.

10. HOSTILITY:
Animosity, contempt, belligerence, disdain for other people outside the interview situation. Rate solely on the basis of the verbal report of feelings and actions of the patient toward others during the past week. Do not infer hostility from neurotic defenses, anxiety, or somatic complaints.

0.= Not present.
1.= Very mild: occasionally felt somewhat angry.
2.= Mild: often felt somewhat angry, or occasionally felt moderately angry.
3.= Moderate: occasionally felt very angry, or often felt moderately angry.
4.= Moderately severe: often felt very angry.
5.= Severe: has acted on his anger by becoming verbally or physically abusive on one or two occasions.
6.= Very severe: has acted on his anger on several occasions.
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness, or not assessed.

11. SUSPICIOUSNESS:
Belief (delusional or otherwise) that others have now, or have had in the past, malicious or discriminatory intent toward the patient. On the basis of verbal report, rate only those suspicions which are currently held whether they concern past or present circumstances. Rate

on the basis of reported (i.e. subjective) information pertaining to the past week.

0.= Not present.
1.= Very mild: rare instances of distrustfulness which may or may not be warranted by the situation.
2.= Mild: occasional instances of suspiciousness that are definitely not warranted by the situation.
3.= Moderate: more frequent suspiciousness or transient ideas of reference.
4.= Moderately severe: pervasive suspiciousness, frequent ideas of reference, or an encapsulated delusion.
5.= Severe: definite delusion(s) of reference or persecution that is (are) not wholly pervasive (e.g. an encapsulated delusion).
6.= Very severe: as above, but more widespread, frequent or intense.
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness, or not assessed.

12. UNCOOPERATIVENESS:
Evidence of resistance, unfriendliness, resentment, and a lack of readiness to cooperate with the interviewer. Rate only on the basis of the patient's attitude and responses to the interviewer and the interview situation. Do not rate on the basis of reported resentment or uncooperativeness outside the interview situation.

0.= Not present.
1.= Very mild: e.g. does not seem motivated.
2.= Mild: e.g. seems evasive in certain areas.
3.= Moderate: e.g. monosyllabic, fails to elaborate spontaneously, somewhat unfriendly.
4.= Moderately severe: e.g. monosyllabic, fails to elaborate spontaneously, quite unfriendly and/or refuses to answer questions occasionally.
5.= Severe: e.g. refuses to answer a number of questions.
6.= Very severe: e.g. refuses to answer most questions.

13. COGNITIVE DYSFUNCTION:
Difficulty or dysfunction with memory and/or concentration, distinguish from DISORIENTATION.

0.= Not present.
1.= Very mild: occasionally felt somewhat dysfunctional.
2.= Mild: occasionally felt moderately dysfunctional, or often felt somewhat dysfunctional.
3.= Moderate: occasionally felt very dysfunctional, or often felt moderately dysfunctional.
4.= Moderately severe: often felt very dysfunctional, or often felt moderately

dysfunctional.

5.= Severe: felt very dysfunctional for 8 - 12 waking hours of the last week.
6.= Very severe: felt very dysfunctional for 12+ waking hours, all of the last week.
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness, or not assessed.

14. HALLUCINATORY BEHAVIOR:

Perceptions (in any sensory modality) in the absence of an identifiable external stimulus. Rate only those experiences that have occurred during the last week. Do not rate "voices in my head" or "visions in my mind" unless the patient can't differentiate between these experiences and his/her thoughts.

0.= Not present.
1.= Very mild: suspected hallucinations only.
2.= Mild: definite hallucinations, but insignificant, infrequent, or transient (e.g. occasionally formless visual hallucinations, a voice calling the patient's name).
3.= Moderate: as above, but more frequent or extensive (e.g. frequently sees the devil's face, two voices carry on lengthy conversations).
4.= Moderately severe: hallucinations are experienced nearly all day, or are a source of extreme distress.
5.= Severe: as above, and has had a moderate impact on the patient's behavior (e.g. concentration difficulties leading to impaired work functioning).
6.= Very severe: as above, and has had a severe impact (e.g. attempts suicide in response to hallucinations)
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness, or not assessed.

15. EMOTIONAL WITHDRAWAL:

Deficiency in relating to the interviewer and to the interview situation. Overt manifestations of this deficiency include poor/absence of eye contact, failure to orient oneself physically toward the interviewer, and a general lack of involvement or engagement in the interview. Distinguish from BLUNTED AFFECT in which deficits in facial expression, body gesture, and voice pattern are scored. Rate on the basis of observations made during the interview.

0.= Not present.
1.= Very mild: e.g. occasionally exhibits poor eye contact.
2.= Mild: e.g. same as above, but more frequent.
3.= Moderate: e.g. exhibits little eye contact but still seems engaged in the interview and is appropriately responsive to all questions.
4.= Moderately severe: e.g. stares at the floor or orients self away from interviewer, but still seems moderately engaged.
5.= Severe: e.g. as above, but more persistent or pervasive.
6.= Very severe: e.g. appears "spacey" or "out of it" (total absence of emotional

relatedness) and is disproportionately uninvolved or unengaged in the interview (DO NOT SCORE IF EXPLAINED BY DISORIENTATION).

16. CONCEPTUAL DISORGANIZATION:
Degree of speech incomprehensibility, include any type of formal thought disorder (e.g. loose associations, incoherence, flight of ideas, neologisms). Do not include mere circumstantiality or pressured speech, even if marked. Do not rate on the basis of the patient's subjective impressions (e.g. "my thought are racing, I can't hold a thought, "my thinking gets all mixed up"). Rate only on the basis of observations made during the interview.

0.= Not present.
1.= Very mild: e.g. somewhat vague, but of doubtful clinical significance.
2.= Mild: e.g. frequently vague, but the interview is able to progress smoothly; occasional looseness of associations.
3.= Moderate: e.g. occasional irrelevant statements, infrequent use of neologisms or moderate loosening of associations.
4.= Moderately severe: as above, but more frequent.
5.= Severe: formal thought disorder is present for most of the interview, and the interview is severely strained.
6.= Very severe: very little coherent information can be obtained.

17. DISORIENTATION:
Confusion or lack of proper association for person, place or time. Rate based on observations made during the interview.

0.= Not present.
1.= Very mild: e.g. seems somewhat confused.
2.= Mild: e.g. indicated 1992 when, in fact, it is 1993.
3.= Moderate: e.g. indicates 1988.
4.= Moderately severe: e.g. is unsure where he/she is .
5.= Severe: e.g. has no idea where he/she is.
6.= Very severe: e.g. does not know who he/she is.
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness, or not assessed.

18. UNUSUAL THOUGHT CONTENT:
Severity of delusions of any type -- consider conviction and effect on actions. Assume full conviction if patient has acted on his or her feelings. Rate on the basis of reported (i.e. subjective) information pertaining to the past week.

0.= Not present.
1.= Very mild: delusion(s) suspected or likely.

2.= Mild: at times, patient questions his or her belief(s) (partial delusion).
3.= Moderate: full delusional conviction, but delusions(s) has little or not influence on behavior.
4.= Moderately severe: full delusional conviction,, but delusion(s) has only occasional impact on behavior.
5.= Severe: delusion(s) has significant effect, e.g. neglects responsibilities because of preoccupation with belief that he/she is God.
6.= Very severe: delusions(s) has major impact e.g. stops eating because believes food is poison.
9.= Cannot be assessed adequately because of severe formal thought disorder, uncooperativeness, or marked evasiveness/guardedness, or not assessed.

19. EXCITED AFFECT:
Heightened emotional tone, including irritability and expansiveness (hypomanic affect). Do not infer affect from statements of grandiose delusions. Rate based on observations made during interview.

0.= Not present.
1.= Very mild and of doubtful clinical significance.
2.= Mild: e.g. irritable or expansive at times during the interview.
3.= Moderate: e.g. constantly irritable or expansive.
4.= Moderately severe: e.g. constantly irritable or expansive, or at times, enraged or euphoric.
5.= Severe: e.g. enraged or euphoric throughout most of the interview.
6.= Very severe: e.g. as above, but to such a degree that the interview must be terminated prematurely.

20. BLUNTED AFFECT:
Diminished affective responsiveness, as characterized by deficits in facial expression, body gesture, and voice pattern. Distinguish from EMOTIONAL WITHDRAWAL in which the focus is on interpersonal impairment rather than affect. Consider degree and consistency of impairment. Rate based on observations made during interview.

0.= Not present.
1.= Very mild.
2.= Mild: e.g. somewhat diminished facial expression or somewhat monotonous voice or somewhat restricted gestures.
3.= Moderate: e.g. as above, but more intense, prolonged, or frequent.
4.= Moderately severe: e.g. flattening of affect, including at least two of the three features; severe lack of facial expression, monotonous voice, or restricted body movements.
5.= Severe: e.g. profound flattening of affect.
6.= Very severe: e.g. totally monotonous voice, and lack of expressive gestures

throughout the evaluation.

21. PSYCHOMOTOR AGITATION:
Rate motor restlessness (agitation) observed during the interview. Do not rate on the basis of subjective experiences reported by the patient. Disregard suspected pathogenesis (e.g. tardive dyskinesia).

0.= Not present.
1.= Very mild: occasionally fidgets.
2.= Mild: e.g. frequently fidgets.
3.= Moderate: constantly fidgets, or frequently fidgets, wrings, hands, and pulls clothing.
4.= Moderately severe: e.g. constantly fidgets, wrings hands and, pulls clothing.
5.= Severe: e.g. cannot remain seated (i.e. must pace).
6.= Very severe: e.g. paces in a frantic manner.

22. PSYCHOMOTOR RETARDATION:
Reduction in energy level evidenced in slow movements. Rate on the basis of observed behavior of the patient only. Do not rate on the basis of the patient's subjective impression of his or her own energy level.

0.= Not present.
1.= Very mild and of doubtful clinical significance.
2.= Mild: e.g. conversation is somewhat retarded, movements somewhat slowed.
3.= Moderate: e.g. conversation is noticeably retarded but not strained.
4.= Moderately severe: e.g. conversation is strained, moves, very slowly.
5.= Severe: e.g. conversation is difficult to maintain, hardly moves at all.
6.= Very severe: e.g. conversation is almost impossible, does not move at all throughout the interview.

23. MANNERISMS AND POSTURE:
Unusual and unnatural motor behavior. Rate only abnormality of movement. Do not rate simple heightened motor activity here. Consider frequency, duration, and degree of bizarreness. Disregard suspected pathogenesis.

0.= Not present.
1.= Very mild: odd behavior but of doubtful clinical significance, e.g. occasional unprompted smiling, infrequent lip movements.
2.= Mild: strange behavior but not obviously bizarre, e.g. infrequent head-tilting (side to side) in a rhythmic fashion, intermittent abnormal finger movements.
3.= Moderate: e.g. assumes unnatural position for a brief period of time, infrequent tongue protrusions, rocking, facial grimacing.

4.= Moderately severe: e.g. assumes and maintains unnatural position throughout interview, unusual movements in several body areas.
5.= Severe: as above, but more frequent, intense, or pervasive.
6.= Very severe: e.g. bizarre posturing throughout most of the interview, continuous abnormal movements in several
of the patient's body areas.

APPENDIX E

Appendix E

DATA COLLECTION FORM FOR CES CD TREATMENT RESEARCH

NAME:_____
DATE:_____
PATIENT NO:_____
SOCIAL SECURITY NO:_____
DOUBLE BLIND DEVICE NO:_____
DOUBLE BLIND SETTING:_____
DATE OF ADMISSIONS:_____
DATE OF BIRTH:_____
AGE:_____
SEX:_____
MARITAL STATUS:_____
RACE:_____
OCCUPATION:_____
HOME ADDRESS:_____
HOME PHONE:_____
BUSINESS PHONE:_____
NEXT OF KIN:_____
ADDRESS:_____
PHONE:_____
ATTENDING PHYSICIAN/THERAPIST_____
DIAGNOSIS:
ENTRY_____
EXIT_____

UA CONCENTRATION LEVELS:
ETOH <10.0 mg/dl NEGATIVE_____
AMPHETAMINE <300 ng/ml NEGATIVE_____
BARBITURATE <200 ng/ml NEGATIVE_____
BENZODIAZEPINE <200ng/ml NEGATIVE_____
CANNABINOID <25 ng/ml NEGATIVE_____
COCAINE <300 ng/ml NEGATIVE_____
OPIATE <200 ng/ml NEGATIVE_____
PCP <25 ng/ml NEGATIVE_____

MEDICATIONS_____
_____

PATIENT NAME....._____
PATIENT NO....._____

SUBJECTS SELF-REPORT OF DRUG USE:
DRUG LAST USED FREQUENCY AMOUNT

ETOH_____
AMPHETAMINE_____
BARBITURATE_____
BENZODIAZEPINE_____
CANNABINOID_____
COCAINE_____
CRACK COCAINE_____
OPIATE_____
PCP_____
PROPOXYPHENE_____
HALLUCINOGENS_____

NUMBER OF PREVIOUS CD ADMISSIONS:....._____
INFORMED CONSENT:.....Y or N

DATE OF PRETESTING:....._____
PRETEST SCORES:
SIGH-D....._____
SIGH-A....._____
BDI....._____
BAI....._____
SIGH-D....._____
SIGH-A....._____
SYMPTOM CHECKLIST INDEX....._____
BPRS-CD....._____

DATES, TIME, INITIAL AND THRESHOLD CURRENT OUTPUT, & ADMINISTRATOR OF TREATMENT:
1.....__/__/__,__:____,_____
2.....__/__/__,__:____,_____
3.....__/__/__,__:____,_____
4.....__/__/__,__:____,_____
5.....__/__/__,__:____,_____
6.....__/__/__,__:____,_____
7.....__/__/__,__:____,_____
8.....__/__/__,__:____,_____
9.....__/__/__,__:____,_____
10.....__/__/__,__:____,_____
11.....__/__/__,__:____,_____
12.....__/__/__,__:____,_____
13.....__/__/__,__:____,_____
14.....__/__/__,__:____,_____
15.....__/__/__,__:____,_____

DATE OF POSTTESTING....._____

POSTTEST SCORES:
SIGH-D.....
SIGH-A.....
BDI.....
BAI.....
SIGH-D.....
SIGH-A.....
SYMPTOM CHECKLIST INDEX.....
BPRS-CD.....

SUBJECTIVE OPINION OF ACTIVE TREATMENT BY SUBJECT.....Y or N
SUBJECTIVE OPINION OF ACTIVE TREATMENT BY RESEARCHER.....Y or N
DEBRIEFING GIVEN.....Y or N
DATE OF RELEASE FORM C.D. UNIT.....__/__/__
DAYS ON C.D. UNIT.....

DAILY SEMI-STRUCTURED INTERVIEW RATINGS

DAY # 1 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

DAY # 2 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD	0	1	2	3	4	5	6	X

DAY # 3 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

DAY # 4 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

DAY # 5 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

DAY # 6 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9

UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

DAY # 7 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

DAY # 8 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

DAY # 9 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

DAY # 10 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

DAY # 11 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

DAY # 12 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

DAY # 13 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X

COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

DAY # 14 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

DAY # 15 EXAMINER_____

APPEARANCE	0	1	2	3	4	5	6	9
DRUG CRAVING	0	1	2	3	4	5	6	9
WITHDRAWAL SYMPTOMS	0	1	2	3	4	5	6	9
SLEEP DISTURBANCE	0	1	2	3	4	5	6	9
ANXIOUS MOOD	0	1	2	3	4	5	6	9
DEPRESSED MOOD	0	1	2	3	4	5	6	9
HOSTILITY	0	1	2	3	4	5	6	9
UNCOOPERATIVENESS	0	1	2	3	4	5	6	X
COGNITIVE DYSFUNCTION	0	1	2	3	4	5	6	9
EXCITED AFFECT	0	1	2	3	4	5	6	X
BLUNTED AFFECT	0	1	2	3	4	5	6	X
PSYCHOMOTOR AGITATION	0	1	2	3	4	5	6	X
PSYCHOMOTOR RETARD.	0	1	2	3	4	5	6	X

APPENDIX F

Appendix F

Treatment Milieus at TRMC and 12 & 12 Treatment Centers

TIME	Monday Activities	Tuesday Activities	Wednesday Activities	Thursday Activities	Friday Activities	Saturday Activities	Sunday Activities
2400-0600	Sleep/Rest	Sleep/Rest	Sleep/Rest	Sleep/Rest	Sleep/Rest	Sleep/Rest	Sleep/Rest
0630-715	Breakfast	Breakfast	Breakfast	Breakfast	Breakfast	Breakfast	Breakfast
0715-0845	Personal Time	Personal Time	Personal Time	Personal Time	Personal Time	Personal Time	Personal Time
0900-0950	Meditation	Meditation	Meditation	Meditation	Meditation	Meditation	Meditation
1000-1100	Primary Group	Family Group	Primary Group	Family Group	Primary Group	Recovery Dynamics	Orientation
1130-1230	Lunch	Lunch	Lunch	Lunch	Lunch	Lunch	Lunch
1230-1450	Recovery Dynamics	Recovery Dynamics	Recovery Dynamics	Recovery Dynamics	Recovery Dynamics	Personal Time	Personal Time
1445-1630	Exercise	Laundry-Study	Exercise	Laundry-Study	Exercise	Structured Outing	Family Group
1630-1730	Orientation Free Time	Orientation Free Time	Orientation Free Time	Orientation Free Time	Orientation Free Time	Orientation Free Time	Orientation Free Time
1730-1815	Dinner	Dinner	Dinner	Dinner	Dinner	Dinner	Dinner
1815-1845	Personal Time	Personal Time	Personal Time	Personal Time	Personal Time	Personal Time	Personal Time
1900-2200	12 Step Meeting	12 Step Meeting	12 Step Meeting	12 Step Meeting	12 Step Meeting	12 Step Meeting	12 Step Meeting
2200-2230	Jobs / Quiet Time	Jobs / Quiet Time	Jobs / Quiet Time	Jobs / Quiet Time	Free Time	Free Time	Jobs / Quiet Time
2330-2400	Lights Out	Lights Out	Lights Out	Lights Out	Jobs Quiet Time	Jobs Quiet Time	Lights Out
2400	Sleep/Rest	Sleep/Rest	Sleep/Rest	Sleep/Rest	Lights Out	Lights Out	Sleep/Rest

APPENDIX G

Appendix G

ANCOVA Results

Summary of ANCOVA Results

Posttreatment outcome measures as analyzed by ANCOVA are summarized and presented in Table 18.

Table 18

Summary of ANCOVA Results

Assessments	SS Effect	df Effect	MS Effect	SS Error	df Error	MS Error	F	p
SCL	1040.852	2	520.426	7212.772	25	288.511	1.80383	.18545
BAI	119.686	2	59.842	1665.823	25	66.633	.89809	.42001
BDI	189.644	2	94.822	1400.393	25	56.016	1.69277	.20442
SIGH-A	727.034	2	363.517	954.227	25	38.169	9.52385	.00084*
SIGH-D	1052.365	2	526.184	695.424	25	27.817	18.9158	.000010*

Marked effects are significant at $p < .050$

Summary of Scheffe Tests (ANCOVA)

Posttreatment outcome measures as analyzed by Scheffe tests are summarized and presented in Table 19.

Table 19
Summary of Schaffe Tests (ANCOVA)

Assessments	CES treatment vs. Control	CES treatment vs. CES sham	CES sham vs. Control
SCL	.3548	.7289	.8170
BAI	.4809	.5499	.9934
BDI	.3192	.4511	.9688
SIGH-A	.0064*	.0176*	.9160
SIGH-D	.0002*	.0030*	.5182

Marked effects are significant at $p < .050$

Summary of Effect Sizes (ANCOVA)

Effect Sizes of posttreatment outcome measures are summarized and presented Table 20.

Table20

Summary of Effect Sizes (ANCOVA)

Assessments	CES treatment vs. Control	CES treatment vs. CES sham	CES sham vs. Control
SCL	.86	.34	.52
BAI	.60	.29	.30
BDI	.75	.68	.07
SIGH-A	1.84	1.52	.32
SIGH-D	2.72	1.98	.74

APPENDIX H

Appendix H

CES CD Treatment Study Variables

01. SUBJNUM: Subject Number
02. GROUPNUM: Group Number
03. DAYSNUM: Number of Days Given Treatment
04. PRESLC: Pretest - Symptom Checklist
05. PREBAI Pretest - Beck Anxiety Inventory (BAI)
06. PREBDI Pretest - Becks Depression Inventory (BDI)
07. PRESIGH-A: Pretest - Structured Interview Guide for the Hamilton Anxiety Rating Scale (HARS)
08. PRESIGH-D1: Pretest - Structured Interview Guide for the Hamilton Depression Rating Scale (HDRS-17 item)
09. PRESIGH-D2: Pretest - Structured Interview Guide for the Hamilton Depression Rating Scale (HDRS-21 item)
10. PTSCL: Posttest Symptom Checklist
11. PT1BAI: Posttest Detox - BAI
12. PT1BDI: Posttest Detox - BDI
13. PT1SIGH-A: Posttest Detox - SIGH-A
14. PT1SIGH-D1: Posttest Detox - SIGH-D
15. PT1SIGH-D2: Posttest Detox - SIGH-D
16. PT2SCL: Posttest Treatment - SCL
17. PT2BAI: Posttest Treatment - BAI
18. PT2BDI: Posttest Treatment - BDI
19 .PT2 SIGH-A: Posttest Treatment - SIGH-A
20. PT2SIGH-D: Posttest Treatment - SIGH-D
21. PT2SIGH-D2: Posttest Treatment - SIGH-D
22. AGE: Age of Subject
23. GENDER: Gender (1=male: 2=female)
24. MARRSTAT: Single=1; Married=2; Divorced=3
25. RACE: White=1; Black=2; Indian=3; Oriental=4
26. ADMSDATE: Date of Admission (1/1/93 = 93.001)
27. MED1VIT: Medication Protocol 1
28. MED2LIB: Medication Protocol 2
29. MED3ADA: Medication Protocol 3
30. MED4DIL: Medication Protocol 4
31. MED5OTHR: Medications not under Protocol
32. MAMP_TX: Mean milla Ampere during study period

33. LOCATION: 1=TRMC; 2=12&12
34. PREVADMT: Number of Previous Admissions to Detox
35. SUBJOPIN: Subjects Opinion of Treatment Level
36. RESOPIN: Researchers Opinion of Treatment Level
37. UA1ETHOL: Urine Analysis for Alcohol
38. UA2AMPH: Urine Analysis for Amphetamines
39. UA3BARB: Urine Analysis for Barbiturates
40. UA4BENZ: Urine Analysis for Benzodiazepines
41. UA5CANN: Urine Analysis for Cannabinoid
42. UA6COKE: Urine Analysis for Cocaine
43. UA7OPIA: Urine Analysis for Opiates
44. UA8PCP: Urine Analysis for PCP
45. UA9PROP: Urine Analysis for Propoxyphene
46. PSTDETOX: Post Detox Test Present
47. PSTTREAT: Posttreatment Test Present
48. TESTS: 1=Detox; 2=Treatment; 3=Both
49. NUMSTEST: 1=Posttest; 2=2 Posttests