CRANIAL ELECTROTHERAPY STIMULATION

IN

THE TREATMENT OF PRIMARY ALCOHOLISM

by

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THIS THESIS IS DEDICATED

TO MY PARENTS

TADEUSZ and ZOFIA MALEC

"That I may be largely wrong
I am free to admit - who can
be right altogether in science,
which is essentially progressive
and corrective?"

Michael Farraday
(1791-1867)

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FOREWORD AND ACKNOWLEDGEMENTS

During my medical studies at Cracow Medical University in Poland, I was fascinated by the problem of chemical substance abuse, especially by chronic alcoholism. The problem, which tragically affects individual lives and is burdensome on the family and, undoubtedly, on society as a whole, is very complex.

After arriving in Montreal, I was fortunate to meet Dr. Maurice Dongier who further inspired my interest in alcoholism. Under his supervision, I carried out my graduate studies in the Department of Psychiatry of McGill University.

My interest was directed toward therapy of chronic alcoholism. I have, therefore, greatly appreciated the task which Dr. Dongier assigned to me, to assess the clinical effectiveness of Cranial Electrotherapy Stimulation as a potential method of biological intervention. The study involved treatment of a large group of alcoholics. It allowed me not only to learn more about the problems related to chronic alcohol abuse, but also to learn the specific methodology involved in this study.

In addition to the continuous guidance I received from my supervisor, Dr. Dongier, I greatly appreciate

the full interest of Dr. Ante L. Padjen, a member of the Department of Pharmacology, who co-supervised my work. Dr. Padjen was directly responsible for carrying out this project, which was sponsored by **Neuroscope**. He guided the treatment procedure used, the collection of data and the analysis. Thanks to his constant assistance, I was able to learn about experimental procedures and computer application in the analysis of clinical data.

I want to express my sincere thanks to Dr. George Schwartz for his constant help and inspiring discussions on scientific methodology and statistics. The Repeated Measure of ANOVA procedure was demonstrated to me by Dr.Lorentz Amabol. I would also like to thank other members of the Department of Psychiatry, namely Professor Juan Carlos Negrete and Professor Robert Pihl.

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In conclusion, I have to state that all these people, and the study itself, have contributed to an unforgettable experience. Without all the help that I received, I would not have been able to complete the difficult task that faced me. I sincerely hope that the results of this study will add to our knowledge of chronic alcoholism.

THADEUS STANISLAS MALEC, M.D.

Montreal, May 31, 1990.

ABSTRACT

The present study reports on the treatment of 62 primary alcoholic patients using either Cranial Electrotherapy Stimulation (CES) or sham stimulation in a control group. The treatment consisted of half-hour sessions given five days per week during a four-week period. The variables analyzed included age, socio-demographic status, duration of alcohol dependence, frequency and volume of consumption, craving, and duration of sleep. Michigan Alcoholism Screening Test (MAST), Alcohol Dependence Scale (ADS), Drinking Behavior Inventory (DBI), Symptoms Checklist - 90 Revised (SCL-90R), Hamilton Psychiatric Rating Scale for Depression, Hamilton Anxiety Scale and Diagnostic Interview Schedule concerning organic brain syndrome, were used. Laboratory tests included serum Gamma-Glutamyl Transferase (GGT) and Mean Corpuscular volume (MCV). Significantly greater improvements were obtained in the active treatment group in the depression subscale and in the Positive Symptoms Distress Level of SCL-90R. The active stimulation reduced alcohol consumption significantly during weekends, but no significant difference in the average weekly consumption was observed between active and sham treatment groups. Consistently more favorable results were observed in the active treatment group in other dependent variables. CES seems to be a safe method of treatment which deserves further investigation concerning its mode of action and clinical application.

SOMMAIRE

Ce travail présente les résultats du traitement de 62 sujets alcooliques primaires, comparant les effets de la stimulation électrique crannienne (SEC) à ceux d'une stimulation factice chez un groupe contrôle. Le traitement a consisté de séances d'une demi-heure administrées cinq jours par semaine pendant une durée de quatre semaines. Les variables analysées incluent: l'âge, le status socio-démographique, la durée de l'alcoolo-dépendance, la fréquence et le volume de la consommation d'alcool, l'appétence pour l'alcool et la durée du sommeil. Les mesures psychologiques utilisées ont été: le test de dépistage de l'alcoolisme du Michigan (MAST), l'échelle de dépendance vis-à-vis de l'alcool (ADS), l'inventaire du comportement vis-à-vis de l'alcool (DBI), le "Symptoms Check List-90-R" (SCL-90-R), les échelles de mesure de Hamilton pour la dépression d'une part, l'anxiété d'autre part, une fraction du "Diagnostic Interview Schedule" concernant le syndrome organique cérébral. Les tests de laboratoire incluent: le dosage dans le sérum de la gamma

glutamyl transferase (GGT) et le volume corpusculaire moyen (VCM). Des améliorations significativement plus marquées ont été observées dans le groupe de traitement actif en ce qui concerne l'échelle de dépression et le "Positive Symptoms Distress Level" du SCL-90-R. La stimulation active a réduit significativement la consommation d'alcool pendant les fins de semaine, mais on n'observe pas de différence significative dans la consommation hebdomadaire moyenne lorsque l'on compare le traitement factice et le traitement actif. Dans l'ensemble des résultats non-significatifs mais avec une tendance favorable ont été observée dans la majorité des autres variables dépendantes (Hamilton anxiété, Hamilton dépression et les autres échelles du SCL-90-R). La SEC semble être une méthode sécuritaire de traitement qui mérite une investigation plus approfondie concernant son mode d'action et ses applications cliniques.

CHAPTER I - CRANIAL ELECTROTHERAPY STIMULATION

1. General Comments

Perhaps one of the least established applications of electric current in medical therapy is Cranial Electrotherapy Stimulation (CES), also known as "electrosleep" or Cranial Electrotherapy. CES makes use of low intensity electric current impulses applied via extracranial electrodes, and it acts by possibly influencing the Central Nervous System (CNS).

Cranial Electrotherapy Stimulation must be differentiated from other CNS "electrotreatments" such as Electroconvulsive Therapy (ECT) and Electro-anesthesia, which use much higher intensities of current and produce immediate and dramatic responses: ECT (current intensity 200-1600 mA) produces convulsions and retrograde amnesia; electro-anesthesia (current intensity 20-160 mA) produces CNS inhibition, initial analgesia followed by electronarcosis with true "anesthetic sleep".

In contrast to the above, Cranial Electrotherapy Stimulation results in neither convulsions nor in loss of consciousness. The current intensities used range from 0.05 to 1.5 mA. The patient remains fully conscious and

perceives, at the most, a slight tingling at the electrode site.

Years of experimentation have not yet determined the degree of CES efficacy although it has been demonstrated that it is harmless. Even experiments with animals, where higher current values and longer applications were used, has proven the treatment harmless. The safety parameters have been acknowledged by the US Food and Drug Administration (Brown 1975, Smith 1985).

At the present time, CES arouses interest as a potential treatment of chemical dependencies including alcohol dependence. As a biological intervention without use of pharmacological agents, it would be of value without any doubt. The present study was designed to evaluate CES in the treatment of alcohol abuse and dependence.

2. History

Historically, CES stems from research on Electroanesthesia. Both methods differ in applied current intensities and in the resulting outcomes. Many of the procedures developed and equipment manufactured, after appropriate parameter modification and adjustment, may be used in both: CES and Electroanesthesia (Schuy and Pfurtscheller 1970).

The history of research on electrical current applic-

ations in medicine started at the beginning of this century, when Leduc in 1902 commenced his first experiments with electricity to induce general anesthesia in dogs, and then repeated the experiment on himself. He showed that only pulsating and not steady intensity current has the ability to induce electro-anesthetic changes (Limoge 1975).

Continuing Leduc's experimentation, his pupil, Robinovich, in 1914 made the first claim about the clinical usefulness of the method in the treatment of insomnia. His work remained forgotten until the early sixties, when scientific interest in medical applications of small intensity electrical currents had its renaissance. It began with the work of Ananev et al (1960) and Gilyarovski et al (1958) and the publication of the book titled "Electrosleep". The current intensities used in their experiments were well below those used previously in electroanesthesia.

It was not until 10 years later that CES aroused more interest on this continent. However, many clinical studies had been carried out in the USSR and Europe, with regular meetings of the International Symposia on Electrosleep and Electroanesthesia. Contrary to what was implied by the old name "electrosleep", CES is not a method of indu-

cing sleep. Furthermore, it has been found that occurrence of sleep is not a sine-qua-non condition for the resulting effects (van Poznak 1969). Taking this into consideration, the US Food and Drug Administration has accepted the term "Cranial Electrotherapy Stimulation" as a standard nomenclature.

3. Mechanism of action

The mechanism of CES action remains unknown and controversial. Many theories, sometimes contradictory, are presented.

The most popular theory, at the present time, presumes a direct influence of the intracranial current flow on the brain cells. The controversy, whether small current intensities as those employed for CES are able to penetrate the cranium in sufficient magnitude to evoke CNS response, has been resolved in many ways. A direct measure of current intensities during CES session in the human brain, done by Dymond et al (1975) in patients undergoing diagnostic procedure with implantation of intracerebral electrodes, detected a measurable electrical field inside the brain tissues. The current intensities in the brain were also studied during experiments on animals undergoing CES, and in models of the human head (Driscoll and Rush 1970, Sances

and Larson 1965,1966, Jarzembski 1985, Liventsev 1967). It was proven that the current could penetrate the cranium and flow through the brain tissues. In turn, experiments by Dymond et al (1975) and Terzulo and Bullock (1956) demonstrated that the observed current intensities were sufficient to modify neuronal functioning. Indirect evidence of the current flow in the brain was obtained with special techniques of electro-encephalography (EEG). It has been stated that at least "...a certain proportion of the electric current passing through the head reaches deeper structures in the cerebrum and has a sufficient effect on the latent period and amplitude of responses from certain systems in this area (reticular formation, thalamus)" (Schuy and Pfurtscheller 1970) Also, some of the hormonal changes: alteration of thyroxine as well as changes in urinary-free catecholamines and 17 ketosteroids, may suggest involvement of the hypothalamus (Briones and Rosenthal 1973, Rosenthal 1973).

A second theory on the CES mode of action postulated the presence of a conditioned-reflex from the skin sensory receptors. In disfavor of this theory, it has been found that stimulation below the sensation threshold, as well as the use of the under-skin electrodes or elimination of the sensory stimuli with local anesthesia, did not prevent

occurrence of CES effects (Smith 1985, Liventsev 1967).

The most skeptical point of view maintains that the CES results are due to suggestion. To test this hypothesis, Ryan and Souheaver (1977) conducted an experiment with CES, comparing the degree of outcome in subjects representing low and high suggestibility. The study did not reveal a significant difference between groups. A specially-designed study to measure placebo effects failed to reveal any significant difference (Smith 1985, Schmitt et al 1986).

4. Neurophysiological bases of CES effects

Two main schools of neurophysiology represent different approaches. The first, based on Pavlovian theory, has the strongest support in the USSR. The other, relying on neurological and biochemical bases, is especially popular in North America.

The Pavlovian theory explains CES action on the basis of the "parabiosis" phenomenon. Parabiosis is defined as a neuronal protective inhibition spreading from the focus in the cortex through the brain. Pavlov describes it "...prolonged stimulation of one and the same point in the cortex leads to profound inhibition at this point in the cortex and this inhibition of course irradiates all over the hemispheres and descends to the subjacent parts of the

brain." Further, it is specified that three kinds of external stimuli can "...cause direct inhibition of the cerebral cortex: very weak, very strong and unusual ones" (Pavlov 1949, Liventsev 1967). From this point of view, CES represents a kind of very weak stimuli, and electro-anesthesia a very strong stimuli. The alternative neuro-physiological approach advocates a more specific mode of action of CES based on the presence of specific centers of the brain.

It was found in 1929, during experiments with cats, that a specific area of thalamus which, if treated with electrical stimulation, caused sleep. It was also shown that the same area of the brain may respond in a different way to varying characteristics of the current. Later, existence of "a waking center" in formatio reticularis of the brain stem was postulated. The theory of localized brain centers was supported with the findings that stimulation of precise sites of the limbic system evokes a specific reaction: anxiety, fear, attention, aggression, aversion, compulsive motion and sexuality. In addition to that, the vegetative function of the heart, blood vessels, bladder, intestine, gall bladder, erectors pillorum, pupils, etc. can be modified via stimulation of limbic area

(Wolff 1967).

Although unproven, a specific action of CES on certain localized structures in the brain is likely and it should be investigated using neurophysiological and neuroanatomical bases.

5. CES in the treatment of substance abuse and dependency

A number of clinical studies with CES has been conducted to treat chemical substance abuse and dependency. It is known that anxiety, depression, decreased stress tolerance, cognitive disturbances and insomnia frequently coexist with chemical addictions and constitute a part of the clinical picture. CES has been reported to alleviate those symptoms in single and poly-drug abusers (Bourgeois et al 1982, Jarzembski 1985, Patterson et al 1984, Smith 1985). Also, it was claimed that poly-drug abusers responded to the treatment as favorably as a group of "pure" alcoholics, (Schmitt et al 1984).

Apart from secondary psychopathologies, a reduction of withdrawal symptoms and craving was claimed in cocaine and opiate abuse as well as in nicotine addiction (Patterson et al 1984, Gossop et al 1984, Ellison et al 1987, Bourgeois et al 1982).

In alcoholics, CES was reported to reduce the withdrawal symptoms including withdrawal seizure and tremors

(Glimer 1973, Smith and O'Neil 1975, Smith et al (1979). In addition, cognitive brain dysfunction, as measured by Benton Visual Retention Test (short memory deficit) and by revised Beta examination (Beta IQ test for perceptive dysfunctions), showed a significantly greater improvement in actively treated subjects than in a sham group. As a matter of fact, only long-term abstinence could bring a similar degree of improvement in non-CES treated patients. Symptoms of depression and anxiety, both psychopathological conditions, were found to be most favorably affected by CES treatment (Schmitt et al 1984, Smith and O'Neil 1975).

In spite of the diversity of studies conducted, to my knowledge there are no reports evaluating craving and alcohol consumption in CES treated patients. In addition, the published results on CES effectiveness still require further corroboration and validation.

6. Clinical studies with CES

Years of experimentation with CES have not proven unequivocally the clinical effectiveness of the treatment, though it was studied in a surprisingly wide spectrum of pathologies. Many of the enthusiastic reports require a critical evaluation becuase of inadequate design used during the study. Another problem is lack of uniformity

in the methodology: different apparatus, different current parameters, non-homogeneous and badly characterized patient groups etc. (Smith 1985). This might partly explain the controversial findings.

CES was used primarily as a treatment of Central Nervous System disturbances. In fact, most of the clinical experiments have been carried out in psychiatry and neurology.

CES has been claimed to be of value in the therapy of neurosis as measured by means of psychopathological scales and confirmed by subjective reports of symptom relief of anxiety and depression. Controversial reports exist on CES usefulness in the treatment of insomnia (Frankel et al 1973, Weiss 1973). In spite of the confusing name "electrosleep", CES does not necessarily induce sleep during the treatment session. CES was used as a treatment of organic brain conditions resulting from head injuries, cerebral atherosclerosis, surgical treatment of the brain and cerebral vessels. In the treatment of psychosis, CES was applied in schizophrenia, manic-depressive and involutional psychosis and borderline states (Chumakova and Kirillova 1967). The results obtained are not unequivocal and less encouraging than in the treatment of neurosis (Banshchikov 1967). The problem

of chemical dependence, being of special interest in the present study, will be described further in this study.

Clinical trials with CES were conducted in refractory trophic ulcer, writer's cramp, viral encephalitis, disseminated sclerosis, hypothalamic and diencephalic disorders. The results obtained were encouraging (Chumakova and Kirillova 1967).

Surprisingly, the patients treated for psychiatric and neurologic disorders often experienced relief in coexisting somatic disorders. (The same was also observed after use of Electroanesthesia). These findings promoted additional experimentations with CES in other brances of medicine (Sergeev 1967, Banshchikov 1967, Wagenender 1970, Flemenbaum 1975, Smith 1985). The validity of the reported results must be evaluated carefully, as the studies were frequently uncontrolled, differed greatly in procedures used and patient characteristics. The multitude of different areas where CES was reported as beneficial is also suspicious. Up to this day, CES has not as yet established itself as a method of treatment.

7. Margin of CES safety; side effects; contraindications

The US Food and Drug Administration (FDA) has reported no hazards or injuries associated with CES treatment. In addition, a classification of devices used was summarized

(Brown 1975, Smith 1985). Furthermore, a review of the results of 88 studies by the US National Research Council revealed that the current output used in CES is too low to be hazardous. In classifying the CES devices, FDA states that risk to health, associated with the procedure, includes skin irritation at the electrode sites, or worsening of the condition if the device is not effective and the patient does not receive conventional treatment. There is no absolute contraindication for CES use. Relative contraindications include:

a. heart pacemaker (careful monitoring of the patient with ECG is required during CES session (Finsterbusch et al 1970);

b. traumatic cystic arachnoiditis and cerebral atherosclerosis (CES may intensify headaches).

The following conditions make electrode placements difficult:

iritis, iridocyclitis (Banshchikov 1967) - applies to "eye-pad electrodes";

weeping dermatitis - applies to skin electrode placement.

CHAPTER II - OBJECTIVES OF THE STUDY

Following is an outline of the three main objectives of the study: 1) Assessment of CES effectiveness to decrease craving and control alcohol consumption. It appears that evaluation of CES ability to decrease craving for alcohol and to control consumption has not been previously tested in clinical trials, using CES. In addition, recording of this data during periods of out-patient treatment was carried out when the subjects were not isolated from environmental influences. 2) Assessment of CES effectiveness in improving coexisting psychopathological conditions related to alcohol abuse, especially with reference to anxiety and depression. Both these conditions are reported to intensify during alcohol withdrawal and to improve during long-term alcohol abstinence. How these coexisting psychopathological conditions respond to CES intervention, in relation to the anticipated changes in alcohol consumption, represents the second objective of our studies. 3) Evaluation of individual tolerance to the treatment and incidence of side-effects. So far, no reports on adverse effects of CES have been recorded; nevertheless, any modification of the applied technique requires an investigation, as proposed in our studies.

CHAPTER III - METHODOLOGY

1. Physical parameters of CES

The characteristics of the current employed in CES vary greatly. Sometimes an incomplete description of the current parameters makes comparative study impossible and causes misinterpretations. For that reason, it may be useful to present an oscilloscopic picture of the current used in addition to its parameter description (Wolff 1967).

The following characteristics of the current must be defined:

Pulse shape

Current direction

Frequency or Pulse Repetition Rate

Pulse amplitude

Pulse width or Duty Cycle

Basal value of the current or Duty Cycle Bias

Pulse shape

Several varieties of current waves are used in CES. The current may flow continuously in the form of squared or sinusoidal pulses or it may be applied as a series of high frequency pulses (sinusoidal = Dernier's type, spike impulsations = Limoge's type, etc.) gated with lower Repetition Rate. Gated current waves are of special signific-

ance particularly in squared form, because of their ability to decrease polarizing electromotive force, as described in the paragraph about frequencies.

Current direction

CES uses a steady direction current. Most frequently, a pair of cathodes is placed in the ophryonic, or in the frontal, region just above the eyebrows; a pair of anodes is placed bilaterally in the mastoid region. The current direction, followed traditionally, was primarily chosen by Leduc in his experimentations. According to Liventsev (1966), this electrode placement enables obtaining the highest electrical field density in the brainstem.

An alternative electrode positioning is the bilateral one. The electrodes are placed at the joint point of occipital and temporal bones, slightly behind the ears.

Frequency or Pulse Repetition Rate

The term "frequency", often encountered in literature to describe the number of pulses in seconds, is appropriate only for the harmonic changes of the current. When no harmonic pulses are used, the more general term "Pulse Repetition Rate (PRR)" should be used.

Many of the issues described below were discovered during experimentations with Electroanesthesia, although

the findings can also be applied to the theoretical and practical assumptions concerning CES.

From the time of Leduc's pioneer work, it has been known that only a pulsatile current is able to evoke the required responses. A steady galvanic current was inactive and, at higher intensities, was found to be noxious (Limoge 1975). It seems that PRR is the most important characteristic responsible for CES biological properties (Wolff 1967, Brown 1975).

Although only a pulsating current produces the required responses during stimulation, there is a limit of the pulse rate which, if exceeded, makes the therapy ineffective without simultaneous increase of current intensity (Limoge 1975). In turn, an increase of current intensity may provoke undesirable muscle contractions. The same may happen even with a low PRR, when the current intensity increases beyond the critical value (Brown 1975, Limoge 1967). An explanation of this phenomenon is cited in Limoge (1967): "The passage of low frequency current through the tissues depends on the resistance of the latter, but a polarizing counterelectromotive force appears, which reduces conductivity and makes it necessary to increase the current intensity".

These assumptions about parameters responsible for current active properties seem to have a general dimension and they may be extrapolated to CES procedure. It should also be emphasized that CES is free from the danger of causing muscle contractures because only small PRR is used (1-250 pps) and current intensities are low.

It is known that frequency or PRR are of great importance for CES, although some questions still remain:

a. Is there an optimal current frequency?

b. Is there any relation between frequency and type of induced effects?

c. Is the response to a given frequency universally the same for all individuals?

The controversy remains to this day and it may be partially responsible for the inconsistent results of experiments (Gruenner 1970, Wolff 1967, Edel 1970).

Pulse amplitude

Current intensities during CES are reported in two ways and may be responsible for the confusion in the literature. There is a single pulse current intensity (being the sum of the pulse amplitude and the Duty Cycle Bias, defined further in this text), and there is an average current intensity, which describes average current flow

over time. The latter always has a lower value and it is recommended that it be calculated from the formula rather than measured directly (substantial measure bias: Wolff 1967).

The single pulse current intensity, in CES usually identical with pulse amplitude (Duty CYcle Bias equals 0), ranges from 0.5-1.5 mA (Brown 1975, Smith 1985).

The Average Current Intensity can be easily counted using the formula:

$$\text{Average Current} = \int_0^{T1} Idt/(T1+T2)$$

For rectangular current, the formula simplifies to:

$$\text{Average Current} = JxT1/(T1+T2)$$

T1 - pulse width, or time ON for the current flow, and

T2 - distance between two adjoining pulses, time OFF for current flow.

Both of the above have to be increased by the value of basal current level - Duty Cycle Bias, if used. Hence:

Total Current = Average Current + Duty Cycle Bias

The role of current intensities in CES is not well understood but its importance is unquestionable. It is the current intensity that produces physical effects of electrical flow. The ionic flux, which is the physical effect of current work in the tissue, is not likely to remain

without influence on the function of nerve cells.

The above assumption led to the introduction of a new class of apparatus for CES treatment. Such an apparatus is equipped with the ability to sustain a steady, optionally adjusted, current intensity, independent from the circuit resistance. Special care must be taken in order not to produce a sudden increase in circuit resistance, for example taking off the electrodes when the current is flowing. Rapid voltage elevation results and may produce a disagreeable sensation at the electrode levels. For this reason, some of the CES apparatus are manufactured with fixed voltage but it does not assure a steady current intensity.

Two main approaches are used in setting current intensity for CES treatment. First method: the amperage is adjusted individually for each patient and for each session, depending on skin sensitivity; it is usually administered below the sensation level. This method does not assure the same treatment for all patients and the study groups. Second method: CES is administered, during each treatment session, to all study subjects with a fixed current amperage, assigned in advance. The advantage of this method is to assure the same conditions of treatment; the disadvantage is the difficulty in producing "blind

date" for the study.

In completing the description of current intensities in CES, it should be stated that, according to a review of literature carried out by the author, no correlation has been reported between current intensities and measures of outcome.

Duty Cycle

Duty Cycle describes timing of the puise during a single cycle of current flow. It is a ratio of the time when the current is ON, producing a pulse, over the time of a full cycle. It is often expressed in percent. The formula for DC reads:

$$DC = T1/(T1+T2)$$

T1 - is the pulse width (the time when the current flows, producing a pulse)

T2 - is the time between two following pulses.

T1+T2 is the time of a single cycle (beginning of one impulsation to the beginning of the next one). T1+T2 is reciprocal to PRR.

The reported T1 ranges from 0.1-4 msec (Liventsev 1967, Smith 1985).

Duty Cycle Bias

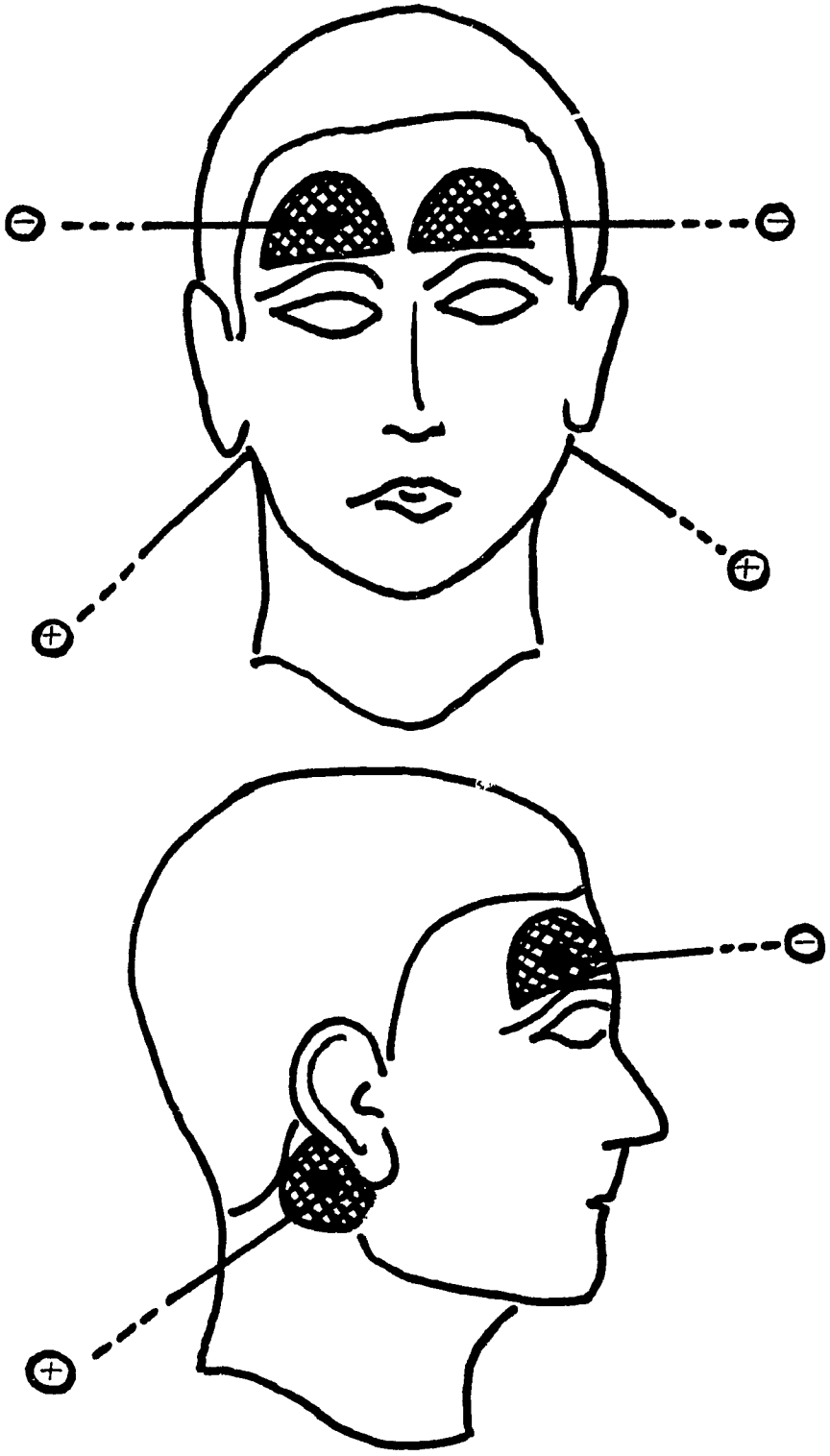
Duty Cycle Bias (DCBias) is a steady direction and

intensity basal current value, on which the pulsating current is superimposed. It is frequently used in electro-anesthesia. CES usually does not use Duty Cycle Bias.

2. Devices and parameters of treatment

The devices for stimulation in our study were constructed under the supervision of Dr.Padjen of the Department of Pharmacology, McGill University. The apparatus was driven by 9V batteries. High voltage output assured a steady intensity of current, independent from circuit resistance. A keyboard and display of parameters enabled us to determine the length of treatment and characteristics of the current (pulse current intensity and Pulse Repetition Rate (PRR)). The current was always administered with PRR = 100 pps, and consisted of a high frequency (10 kHz) sinusoidal continuous current flow, gated in the form of squared pulses. Current intensity (pulse amplitude) was adjusted on the basis of individual skin sensitivity threshold (methods of limits and signal detection procedure: Falmagne, 1985). The treatment was administered with a current intensity of 10 percent below the sensation threshold, and remained unchanged for each patient during four weeks of therapy. Duty Cycle Bias equalled zero; Duty Cycle was 50 percent. The device was equipped with special features:

FIGURE 1. ELECTRODE PLACEMENT



automatic stop after 30-min session and an alarm system (interruption of the current flow and alarm noise), should an accidental interruption of the circuit occur.

To assure similar sensation phenomena, the sham device produced superficial current flow in the frontal region without traversing the cranium. This feature further enforced the double-blind condition of the study.

Current was delivered via four disposable, self-adhesive electrodes (T.E.N.S. type T10001-38, production Ver-Med). Two of the electrodes, cathodes, were placed in the frontal regions symmetrically above the eyebrows; two others, anodes, were placed in the mastoid regions just behind the ears.

3. Determination of sample size

The high drop-out rate frequently observed in ambulatory treatment of alcoholism, reaching up to 90% of the initial sample size (Jaffe 1984), must be taken into account. Previous experience with ambulatory-treated patients in our research clinic showed an attrition over 20% over a 4-week period. The drop-out is believed to be due in part to fluctuation in motivation and also to prolonged withdrawal symptoms, with related affective disturbances (anxiety, depression, insomnia) leading to early relapse.

In order to determine the sample size, a power analysis was conducted. To detect 20% difference in improvement observed between actual and sham treatment groups in psychopathological variables (by means of 2-tailed t-test for independent samples) at the 5% level of significance with power of 80%, each group (active and sham) should have 75 subjects who completed the treatment. Assuming a 20% risk of drop-out, the initial enrollment should be 180 subjects.

The present data, obtained for 62 subjects who completed the treatment so far, should still be considered as a pilot study in preparation for a possible larger-scale clinical trial.

4. Recruitement of patients

Inclusion criteria:

1. Gender: only male alcoholics were included to increase homogeneity of the study group. Female alcoholism has a somewhat different history of development; female alcoholics are believed to seek more help for psychiatric or physical illnesses; they are believed to be more depressed and show a tendency to periodic drinking (Cadoret 1979, Hesselbrock 1979).

2. Age: 18-65 yrs.

3. DSM-IIIR criteria for alcohol abuse and/or depend-

ence (305-0X and 303.9X).

4. Duration of alcohol abuse more than 6 months.

5. Average daily consumption of 5 standard drinks (alcohol amount adjusted to body weight; See Appendix A).

6. Primary alcoholism (see Excluded Criteria 1 and 2 below).

7. Willingness to accept the protocol of the study and to sign the consent form.

Exclusion criteria:

1. Intermittent alcoholism (ability to abstain from alcohol consumption for a minimum 2 months, at least 3 times during the last 3 years).

2. Secondary alcoholism. According to the concept of primary and secondary psychiatric conditions, the term "primary" refers to a psychiatric condition that exists either alone or, if another condition is present, began sooner than the second condition. "Secondary" refers to a psychiatric condition that began later than the primary condition. Anti-social personality disorder, borderline personality and psychosis (diagnosed according to DSM-IIIR criteria) represent secondary causes of alcohol abuse and, therefore, were excluded.

3. Alcohol dependence coexisting with other substance

abuse.

4. Presence of a physical illness requiring immediate or continuous treatment, or taking medication on a regular basis.

5. Distance from the hospital making attendance doubtful.

Initial interview and consent form

The recruitement of subjects was carried out by means of advertisement in the media. Both French and English newspapers were used. The subjects' willingness to participate in the study was screened initially on the telephone. Before beginning the study, subjects were told about the treatment procedure, consisting of the double-blind study design, and signed a consent form. They were free to quit the study at any time and without obligation. Nevertheless, to decrease attrition, subjects who completed the treatment received payment for their time and transportation costs.

5. Data collection

The initial assessment for each patient was done before commencement of the therapy. It consisted of:

1. Complete medical examination:

A. Medical history and physical examination: Hgb, Htc, RBC, WBC, and differential smear, ESR,

proteins, B12, folates: (RBC and serum), T4, AST, ALT, GGT, direct and indirect bilirubin, alkaline phosphatase, albumins, globulins, Na, Cl, HCO3, Ca (corrected for albumins), Mg, PO4, BUN, creatinine, VDRL.

B. Laboratory tests: urine, routine examination.

2. Socio-demographic data collection: age, language, marital status, education, type of employment, employment status, income, legal problems.

3. Assessment of baseline average consumption of alcohol.

During the screening interview, subjects were queried about their daily average consumption, about the kind of alcohol ingested and about frequency of alcohol use: days per week. Daily average consumption of alcohol above 5 standard drinks (amount of alcohol adjusted to body weight, Appendix A) and frequency of abuse not less than 5 days/week were the criteria included to establish a group of heavy drinkers with a continuous drinking pattern. Baseline alcohol consumption was the point of reference for evaluating treatment outcome.

4. Assessment of drinking behavior and psychopathology.

MAST (Michigan Alcoholism Screening Test) in its

original form of 25 items was used to corroborate the diagnosis of alcoholism. The test is well validated and widely used as a screening test. A score of five or more is the positive indicator (Seltzer 1971).

ADS (Alcohol Dependence Scale) was used to establish the level of alcohol dependence in a quantitative way during the last 12 months before the interview (Skinner and Horn 1984). The test was given at baseline.

DBI (Drinking Behavior Inventory) has been shown to be a good quantitative measure of alcohol impairment over a defined preceding period. It is also sensitive as a measure of effectiveness in the treatment of alcoholism. The test was given at baseline and after completing the study (Shelton et al 1969).

HPRSD (Hamilton Psychiatric Rating Scale for Depression) is the most widely used depression scale. It consists of 21 items grouped into 6 factors: anxiety/somatization, cognitive disturbances, depression/retardation, retardation, sleep disturbances and weight loss. The scale measures the severity of depression and is not a diagnostic tool. The scale was administered at baseline and after completing the therapy (Hamilton 1967).

HAS (Hamilton Anxiety Scale) is one of the oldest and

the most widely used anxiety scale. It has been shown that the scale is sensitive to antianxiety agents. Two subscales are derived from HAS: "psychic anxiety" and "somatic anxiety". HAS is not a diagnostic instrument. The scale was administered at baseline and after completing the therapy (Guy 1976).

SCL-90R (Hopkins Symptoms Checklist 90 Revised) is a multidimensional self-report inventory comprising nine factors to measure specific areas of distress: I) somatization, II) obsessive-compulsive symptoms, III) interpersonal sensitivity, IV) depression, V) anxiety, VI) hostility, VII) phobic anxiety, VIII) paranoid ideation, IX) psychoticism. In addition to these, three general scores may be derived: GSI (General Symptomatic Index), PSDI also called PSDL (Positive Symptoms Distress Index/Level), PST (Positive Symptoms Total). The scale has been shown to be useful not only in discerning patients with respect to severity of illness but also in measuring therapeutic changes over time. The scale is sensitive also to a variety of non-pharmacological aspects of the treatment. The checklist was administered at baseline and after completing the therapy (Derogatis et al 1973).

DIS (Diagnostic Interview Schedule) - Version IIIA,

Items 239-258, was used to examine Organic Brain Syndrome. The validity of DIS III in the general population has been evaluated (Heltzer et al 1985). The schedule was administered at baseline and after completing the therapy.

5. Daily monitoring of patients.

Before each treatment session, the following data were collected:

a) Alcohol consumption. Patients were asked about the type and amount of alcohol ingested during the 24 hours preceding the treatment. They were provided with a "diary of consumption" form on which they reported the quantity of alcohol ingested. Consumption was then calculated and expressed in amount of pure alcohol adjusted to body weight (based on the Addiction Research Foundation "Risk-O-Graph" part of the "Assist" interview: Appendix A).

b) Alcohol saliva screening test. A stick saturated with saliva showed blood alcoholemia at the time of treatment (change in color was compared with a pattern block). The test was done to ensure reliability of reports on consumption.

c) Craving was measured with a visual analog scale. A line of 10 cm representing the range of craving from zero (left) to the maximum craving (right) experienced by any

patient in this group, was indicated on this scale. Each patient was asked to show the degree of craving experienced for the entire day on this line. Data on craving was obtained for the treatment days only (Monday to Friday).

d) Duration of sleep was recorded.

e) Patients were asked to list any health problems or side effects which they experienced.

Note: No data was collected during weekends on craving for alcohol and sleeping hours; these were recorded for treatment days only.

6. Application of treatment

Treatment was administered in the Alcohol Research Centre of Douglas Hospital on an out-patient basis. In case a medical emergency occurred, an arrangement was made with the Emergency Service of the Hospital. Patients were placed in a tranquil environment during the CES session to help them achieve relaxation and to supervise them. Patients were asked to refrain from any activity. Trained technicians were responsible for the application of the treatment, supervision and daily data collection. A nurse experienced in the treatment of alcoholics was in charge of the selection of subjects, assessment and follow-up, under the supervision of a psychiatrist. After the daily

data was collected, the following procedure was followed: a) the skin was cleansed with alcohol and four electrodes were placed: two in the frontal and two in the mastoid regions; b) the patient was placed in a reclining position on a long chair; c) the treatment device was programmed, connected with the electrodes and switched on; d) the experiences of the patient were recorded; e) after completion of the treatment, the apparatus was disconnected and the electrodes removed; f) patient's comments following the treatment were recorded.

7. Final assessment and follow-up monitoring

The final evaluation was made after completion of the four weeks of therapy by the patients. All the tests from the baseline assessment were repeated with the exception of: MAST (diagnostic test), ADS (test establishing level of alcohol dependence during 12 months preceding interview), and sociodemographic data (no short term changes in these variables were anticipated). Furthermore, patients were asked to estimate the results of the treatment. The "blind study" was checked by recording the therapists' "guess" as to whether the active or the sham procedure was used.

After completing the four weeks of therapy, patients were followed via telephone for a period of 6 months.

Three interviews (after 8, 16 and 24 weeks) were conducted. Subjects were asked about their weekly average consumption of alcohol, craving frequency on a five-point scale (no craving at all, 1-2 times per week, once daily, more than once daily, continuous) and duration of sleep.

CHAPTER IV - RESULTS

1. Characteristics of patient's group

Sixty-seven subjects met the admission criteria for the study. During the period of four weeks of therapy, five subjects dropped out, a rate of 7.5%. None of the premature terminations was due to intolerance of the treatment. These patients allegedly had personal grounds for discontinuing their participation in the study. The reasons given included: change of job, living conditions, etc.

The age of the patients ranged from 23-62 yrs (mean 40.1 yrs; SD 10.0). The socio-demographic characteristics of the treatment group are shown in Table 2. All of the subjects met the DSM-IIIR criteria for alcohol abuse and dependence. The baseline data of the study group, which included the Michigan Alcoholism Screening Test (MAST), Alcohol Dependence Scale (ADS) and Drinking Behavior Interview (DBI), are listed in Table 1.

test	mean	SD	range
MAST	26.05	9.03	9-46
ADS	17.24	6.24	6-34
DBI	49.42	22.59	8-97

Table 1: Results of MAST, ADS & DBI testing in the patient's group under study.

table 2. TABLE OF SOCIO-DEMOGRAPHIC DATA

CHARACTERISTIC	SUBDIVISION	frequency	PERCENT
GENDER	MALES	62	100
AGE	MEAN 40.1 yrs (SD 10.0) AGE RANGE 23 - 62 yrs		
LANGUAGE	FRENCH	55	88.7
	ENGLISH	7	11 3
MARITAL STATUS			
	SINGLE	16	25.8
	COMMON LAW	10	16.1
	MARRIED	17	27.4
	SEPARATED OR DIVORCED	19	30 6
LIVING WITH			
	MATE OR FAMILY	26	41.9
	OTHER RELATIVES	35	56.5
	ALONE	1	1 6
TYPE OF EMPLOYMENT			
	UNSKILLED LABOURER	5	8.1
	SEMI-SKILLED	4	6.5
	SKILLED OR FARMER	7	11.3
	WHITE COLLAR	4	6.5
	TECHNICIAN-PARAPROFESSI	7	11 3
	MANAGERIAL	10	16.1
	PROFESSIONAL	20	32.3
	CLERICAL	2	3 2
	SELF EMPLOYED AND OTHE	3	4 8
EMPLOYMENT STATUS			
	NOT EMPLOYED	25	40.3
	IRREGULAR	6	9.7
	REGULAR	30	48.4
	RETIRED OR SICK	1	1 6
INCOME			
	NONE	1	1.7
	5,000$ OR LESS	2	3.3
	5,001-15,000$	13	21.7
	15,001-30,000$	25	40
	OVER 30,000$	21	33.3
EDUCATION			
	PRIMARY	0	0
	SECONDARY	16	25.8
	POSTSECONDARY	21	33.9
	COLLEGE OR UNIVERSITY	25	40 3
LEGAL PROBLEMS			
	NONE	56	91
	MINOR	3	4.5
	NO ANSWER	3	4 5

These results clearly imply that our group of subjects was more dependent than those in the "heavy drinkers" category and suffered from psychological, physical and social consequences of alcohol abuse. Laboratory tests showed that in 75.8% of cases, macrocytic anemia was evident (MCV greater than 92 fL) and the GGT level was above normal in 22.8% of subjects. Both values are reported to be increased in alcohol abuse and may be used as objective indices of alcohol consumption on a long-term basis.

2. Test of successful randomization

No statistically significant difference between the active and sham treatment group was found in any of the baseline measures: mean age, socio-demographic characteristics, MAST, ADS, DBI, Hamilton Depression Scale, Hamilton Anxiety Scale, SCL90R, baseline consumption, years of alcohol abuse, number of days per week alcohol consumed, MCV and GGT (Table 3).

3. Test of blindness

After completing the therapy, patients' and therapists' "guess" on the applied treatment was recorded. Possible responses were coded as "active", "sham", "don't know". The answers obtained were tested using Chi^2. Most patients thought that they received active treatment (Table 4,5,6)

TABLE 3. TESTS OF SUCCESSFUL RANDOMIZATION.

CHARACTERISTICS	ACTIVE		SHAM		P
	MEAN	SD	MEAN	SD	
Age	40.18	9.76	39.97	10.36	0.94
Income group	5.15	2.80	5.97	2.69	0.26
Years of education	13.93	3.83	13.44	3.30	0.59
Baseline alcohol consumption	10.46	5.23	13.06	6.96	0.11
Years of alcohol abuse	9.68	7.75	12.29	9.81	0.26
Alcohol abuse (days/week)	6.32	0.98	6.09	1.33	0.45
Current intensity during therapy	592.86	379.99	544.12	266.22	0.56
MAST	25.75	9.82	26.29	8.48	0.82
ADS	17.00	6.24	17.44	6.32	0.78
DBI	46.82	20.03	51.56	24.60	0.42
GGT	81.04	86.68	78.67	91.36	0.92
MCV	96.25	3.88	95.27	5.55	0.43
SCL90R :					
depression	1.41	0.71	1.29	0.71	0.49
anxiety	1.08	0.86	1.05	0.79	0.90
PST	51.43	20.79	49.91	18.01	0.76
GSI	1.02	0.62	1.00	0.61	0.92
PSDL	1.65	0.49	1.69	0.50	0.75
Hamilton Depression	19.61	7.70	19.82	7.45	0.91
Hamilton Anxiety	15.11	8.02	16.21	8.93	0.62
DIS (organic brain syndrome)	37.82	6.74	36.79	7.74	0.58

Table shows baseline characteristics of active and sham treatment group.
P - significance of independent samples t-tests comparing between groups means.
In italics subscales and global scores of SCL90R

TESTS OF STUDY 'BLINDNESS'.

Test of patients 'blindness'.

TABLE 4.

ACTUAL TREATMENT	GUESS			TOTAL
	active	sham	doubt	
ACTIVE *(row percentage)*	**14** 54%	**6** 23%	**6** 23%	**26** 100%
SHAM *(row percentage)*	**22** 67%	**7** 21%	**4** 12%	**33** 100%

N=59
Chi^2 sign P=0.49

Test of the 1-st therapeutists 'blindness'.

TABLE 5.

ACTUAL TREATMENT	GUESS		TOTAL
	active	sham	
ACTIVE *(row percentage)*	**17** 61%	**11** 39%	**28** 100%
SHAM *(row percentage)*	**17** 51%	**16** 49%	**33** 100%

N=61
Chi^2 sign P=0.47

Test of the 2-d therapeutists 'blindness'.

TABLE 6.

ACTUAL TREATMENT	GUESS		TOTAL
	active	sham	
ACTIVE *(row percentage)*	**18** 64%	**10** 36%	**28** 100%
SHAM *(row percentage)*	**14** 42%	**19** 58%	**33** 100%

N=61
Chi^2 sign P=0.09

significance of Chi^2 = 0.49. The null hypothesis, that patient's guess was due to chance only, cannot be rejected. The therapists guessed correctly the treatment applied in 54%, resp. 61% of cases. Chi^2 statistics, which tested the null hypotheses that their responses were due to chance, were 0.47 and 0.09 (before Yates correction). The null hypotheses cannot be rejected. The analysis proved "double blindness" of the procedure.

4. Hopkins Symptoms Checklist - 90 Revised (SCL-90R)

The data was analyzed in two ways: 1) the baseline scores and end-scores of all the subscales and the global measures of SCL-90R were compared in paired t-test within each of the treatment groups (active and sham). As shown in Table 7, both groups improved significantly during the treatment period. 2) the degree of improvement (change score equals final score less baseline score) between active and sham group was compared using independent t-test. The results are listed in Table 8 and demonstrate that the active group improved significantly when compared to the sham group in the depression subscale of SCL-90R (p=0.01) and in one of the global scores of SCL-90R - the Positive Symptoms Distress Level (p=0.04).

Figure 2.

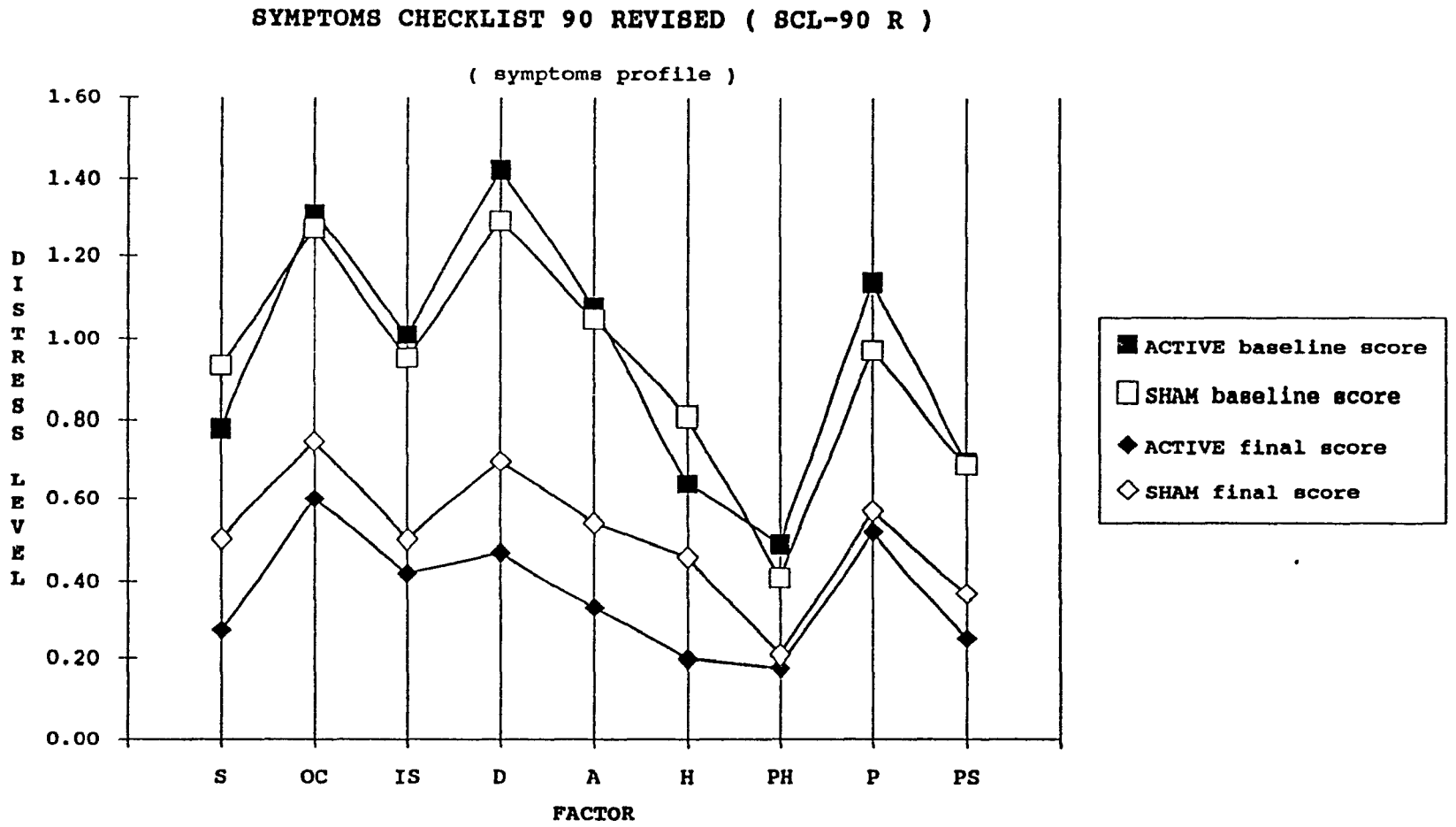

SYMPTOMS CHECKLIST 90 REVISED (SCL-90 R)
(symptoms profile)
DISTRESS LEVEL
1.60
1.40
1.20
1.00
0.80
0.60
0.40
0.20
0.00
S
OC
IS
D
A
H
PH
P
PS
FACTOR
ACTIVE baseline score
SHAM baseline score
ACTIVE final score
SHAM final score

TABLE 7. SYMPTOMS CHECKLIST - 90 R (SCL-90R)

FACTOR SCORE	ACTIVE					SHAM				
	B-LINE	SD	END	SD	P	B-LINE	SD	END	SD	P
SOMATIZATION	0.78	0.64	0.27	0.29	<0.001	0.92	0.76	0.5	0.46	<0.001
OBSESSIVE-COMPULSIVE	1.31	0.75	0.4	0.43	<0.001	1.27	0.73	0.75	0.69	<0.001
INTERPERSONAL SENSITIVITY	1.01	0.78	0.42	0.42	<0.001	0.95	0.72	0.5	0.55	<0.001
DEPRESSION	1.42	0.71	0.47	0.37	<0.001	1.29	0.71	0.7	0.56	<0.001
ANXIETY	1.08	0.86	0.33	0.37	<0.001	1.05	0.79	0.55	0.6	<0.001
HOSTILITY	0.64	0.57	0.2	0.2	<0.001	0.81	0.85	0.46	0.61	<0.001
PHOBIC ANXIETY	0.49	0.66	0.18	0.29	0.005	0.41	0.47	0.21	0.4	0.001
PARANOID IDEATION	1.14	0.79	0.53	0.51	<0.001	0.97	0.81	0.57	0.59	<0.001
PSYCHOTICISM	0.7	0.57	0.25	0.29	<0.001	0.69	0.49	0.37	0.53	<0.001
POSITIVE SYMPTOMS TOTAL	51.43	20.79	28.39	19.18	<0.001	49.91	18.01	32.44	20.54	<0.001
GENERAL SYMPTOMATIC INDEX	1.02	0.62	0.38	0.29	<0.001	1.00	0.61	0.56	0.497	<0.001
POSITIVE SYMPTOMS DISTRESS LEVEL	1.65	0.49	1.14	0.16	<0.001	1.69	0.499	1.39	0.41	<0.001

TABLE 8. SYMPTOMS CHECKLIST - 90 R (SCL-90R)

CHANGE IN FACTOR	ACTIVE	SD	SHAM	SD	P
SOMATIZATION	-0.52	0.45	-0.42	0.47	0.42
OBSESSIVE-COMPULSIVE	-0.7	0.63	-0.52	0.57	0.23
INTERPERSONAL SENSITIVITY	-0.59	0.66	-0.45	0.49	0.34
DEPRESSION	-0.95	0.57	-0.59	0.54	0.013
ANXIETY	-0.75	0.73	-0.51	0.53	0.13
HOSTILITY	-0.45	0.5	-0.36	0.43	0.45
PHOBIC ANXIETY	-0.31	0.54	-0.19	0.31	0.32
PARANOID IDEATION	-0.62	0.68	-0.4	0.42	0.15
PSYCHOTICISM	-0.46	0.43	-0.32	0.4	0.19
POSITIVE SYMPTOMS TOTAL	-23.04	15.75	-17.47	13.18	0.14
GENERAL SYMPTOMATIC INDEX	-0.64	0.5	-0.44	0.35	0.08
POSITIVE SYMPTOMS DISTRESS LEVEL	-0.51	0.43	-0.29	0.37	0.04

Table 7. shows paired t-tests comparing baseline- and final- score within each treatment group.

Table 8. shows independent t-tests comparing change scores (end - b.line) between both treatment groups.

Reported values: means and standard deviations.
P - significance level.
Significant values - bolded.

5. Hamilton Psychiatric Rating Scale for Depression

The analysis was carried out in two steps. Firstly, the baseline scores of the factors and the total scores were compared with the final scores using paired t-tests with each treatment group. Wilcoxon Matched-pairs Signed Rank Test was used to test the weight factor, which did not meet the criteria for parametric testing. The results of the analysis are shown in Table 9.

Secondly, the change scores in all the factors and in the total scores (change score = final score - baseline score; measure of improvement) were compared between the active and the sham treatment groups. The analysis was carried out with independent t-test. Mann-Whitney U test was used to compare the change scores in the weight factor. The results of the analysis are shown in Table 10.

Both treatment groups scored significantly lower after completing the treatment in totals and in all factors except weight loss.

As shown in the second analysis, the degree of improvement (change scores) does not differ significantly between the treatment groups. The null hypothesis (Ho: degree of improvement obtained during the therapy does not depend on whether the active or the sham procedure is used) cannot be

TABLE 9 **HAMILTON PSYCHIATRIC RATING SCALE FOR DEPRESSION**

(paired t-tests)

FACTOR	ACTIVE					SHAM				
	B-LINE	SD	END	SD	P	B-LINE	SD	END	SD	P
ANXIETY/SOMATIZATION	5.68	2.26	3.00	1 74	**<0.001**	6.44	2.16	3.71	2.07	**<0.001**
WEIGHT*										
COGNITIVE DISTURBANCES	5.11	2 82	2.68	1.74	**<0.001**	4 94	2 88	2 94	2.65	**<0.001**
DIURNAL VARIATION	1.21	1.52	0.25	0.93	**0.004**	1.03	1.55	0.32	1.01	**0.019**
RETARDATION	4.61	2.01	2.04	1 67	**<0.001**	4.32	1.82	2.44	2.06	**<0.001**
INSOMNIA	2.86	1.80	1.29	1.46	**<0.001**	3.03	2.17	1.68	1.75	**<0.001**
TOTAL	19.61	7.70	9.25	5.71	**<0.001**	19 82	7.45	11.12	6.78	**<0.001**
(analysis was done with Wilcoxon Matched-pairs Signed Rank Test)										
WEIGHT*	median=	0	median=	0	0.068	median=	0	median=	0	0.59
	mean=	0 14	mean=	0		mean=	0.06	mean=	0.03	

Reported values are average baseline and average final scores within each treatment group, SD - standard deviations;
P - level of significance of paired comparisons of baseline- and final-score within each treatment group

TABLE 10 **HAMILTON PSYCHIATRIC RATING SCALE FOR DEPRESSION**

(Independent samples t-test)

FACTOR Improvement	ACTIVE	SD	SHAM	SD	P
ANXIETY/SOMATIZATION	-2 68	2.58	-2.74	2 17	0.93
WEIGHT**					
COGNITIVE DISTURBANCES	-2.43	2.81	-2.00	2.22	0.50
DIURNAL VARIATION	-0 96	1 62	-0.71	1.66	0.54
RETARDATION	-2 57	1 89	-1.88	1.95	0.17
INSOMNIA	-1.57	1.99	-1.35	1.92	0.66
TOTAL	-10 36	7 68	-8.71	5 33	0.34
(analysis was done with Mann-Withney test)					
WEIGHT**	median=	0	median=	0	0.19
	mean=	-0 14	mean=	-0 03	

Reported values are means of change-scores (measure of improvement) ; change score = (end score - baseline score) ;
Negative value of the change score signifies improvement I.e. final score was lower than baseline score; P - level of significance;

rejected.

6. Hamilton Anxiety Scale

A significant decrese in the total score and in both the psychic and somatic anxiety factors was observed after four weeks of the experiment in both treatment groups, as confirmed by the paired t-test (Table 11).

The degree of improvement was compared using independent sample t-tests. Although greater improvement was observed in the active group, the results were not significantly different from those observed in the sham-treated group.

The results of the analysis are shown in Table 12.

7. Other tests

Other tests included Drinking Behavior Interview (DBI), Diagnostic Interview Schedule - section on organic brain syndrome (DIS), Mean Corpuscular Volume (MCV) and Serum Gamma-Glutamyl Transferase (GGT).

Statistical analysis with paired t-tests (Table 13) demonstrated: a) a significant decrease in DBI scores at the end of the therapy in both treatment groups; b) no significant changes in DIS (section on organic brain syndrome); c) a significant decrease of MCV only in the sham group; d) a significant lowering of GGT level in both treatment groups.

TABLE 11. HAMILTON ANXIETY SCALE

FACTOR	ACTIVE					SHAM				
	B-LINE	SD	END	SD	P	B-LINE	SD	END	SD	P
PSYCHIC ANXIETY	10.04	5.07	5.00	3.19	<0.001	10.24	4.92	5.76	4.20	<0.001
SOMATIC ANXIETY	5.07	3.71	1.86	1.80	<0.001	5.97	4.94	3.24	3.34	<0.001
TOTAL SCORE	15.11	8.02	6.86	4.30	<0.001	16.21	8.93	9.00	6.79	<0.001

Reported values are means and standars deviations of baseline- and end-score.
P - significance of paired t-test comparing end-score with baseline-score within each treatment group

TABLE 12. HAMILTON ANXIETY SCALE

FACTOR CHANGE	ACTIVE	SD	SHAM	SD	P
PSYCHIC ANXIETY	-5.04	4.91	-4.49	4.45	0.65
SOMATIC ANXIETY	-3.21	3.27	-2.73	2.92	0.55
TOTAL CHANGE	-8.25	7.22	-7.21	6.20	0.55

Table presents means and standard deviations of the change score in both groups.
Change score = (end score - baseline score).
P - significance of independent samples t-test comparing change scores between groups.

DBI, DIS*, MCV, GGT.

TABLE 13.

TEST	ACTIVE b-line	SD	end	SD	P	SHAM b-line	SD	end	SD	P
DBI	46.82	20.03	14.57	14.06	**<0.001**	51.56	24.6	18.06	17.56	**<0.001**
DIS*	37.82	6.74	35.29	5.59	0.058	36.79	7.74	36.5	6.78	0.79
MCV	96.25	3.88	95.93	3.89	0.45	95.27	5.55	94.41	4.94	**0.006**
GGT	78.33	82.01	54.71	50.79	**0.026**	78.67	91.36	53.82	52.07	**0.014**

TABLE 14.

TEST	active	SD	sham	SD	P
DBI	-32.29	17.98	-33.53	26.33	0.83
DIS*	-2.54	6.79	-0.29	6.38	0.19
MCV	-0.32	2.21	-0.85	1.69	0.29
GGT	-23.62	44.96	-24.85	54.65	0.93

DBI - Drinking Behavior Inventory;
DIS* - Diagnostic Interview Schedule (section on organic brain syndrom);
MCV - mean corpuscular volume (lab.test);
GGT - serum gamma-Glutamyl Transferase;

Table 13. - reported values are means and standard deviations of the baseline- and the final-result .
P - designates significance level of paired t-test comparing final-score with baseline-score within each treatment group.

Table 14. - reported values are the means and standard deviations (SD) of obtained improvements.
P - designates significance level of independent samples t-test comparing change scores between groups.
Measure of improvement is the change score= final score - baseline score;
Negative sign indicates that final-score was smaller than baseline-score;

When the degree of improvement in these variables in the active group was compared with the control group t-test, no significant differences were found. The null hypothesis cannot be rejected, stating that changes in DBI, DIS, MCV, GGT do not differ between the active and sham treatment groups (Table 14).

8. Daily alcohol consumption during the therapy

Data on alcohol consumption were obtained during 25 days of therapy.

Figure 3 shows the average daily alcohol consumption in the two treatment groups. It can be seen that the active and sham groups present different patterns of daily consumption.

In the active treatment group, there is no significant variation in frequency of days with higher consumption. In contrast, the sham treatment group showed significantly higher alcohol consumption levels during weekends. The consumption was highest and most constant on Saturdays. The next highest level appeared on Fridays, except for the first treatment week. Alcohol consumption was less dramatic on Sundays, but nevertheless showed a constant increase. On Thursday of the second week, an episode of high consumption was registered.

Figure 3.

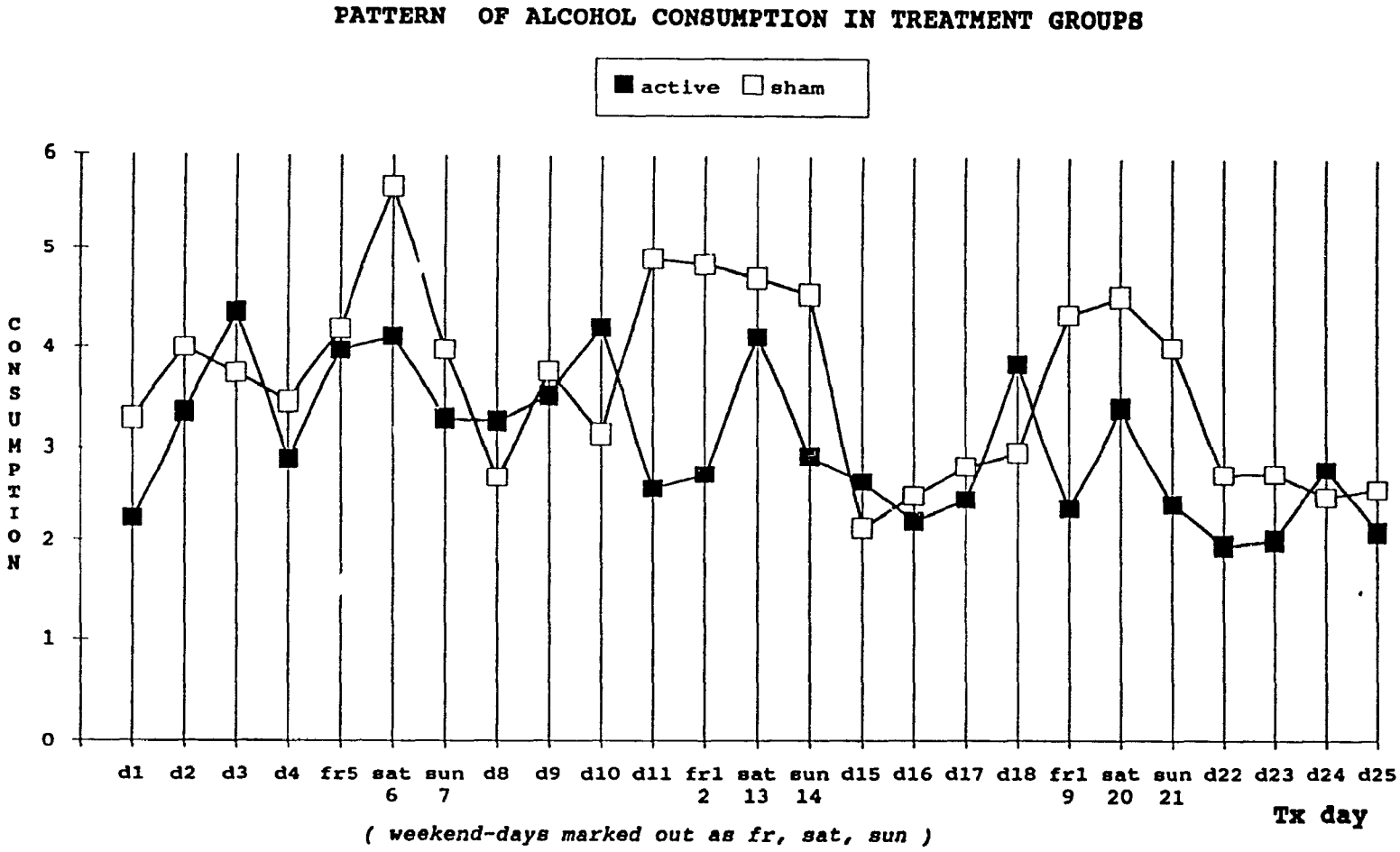

PATTERN OF ALCOHOL CONSUMPTION IN TREATMENT GROUPS
active
sham
CONSUMPTION
0
1
2
3
4
5
6
d1 d2 d3 d4 fr5 sat 6 sun 7 d8 d9 d10 d11 fr1 2 sat 13 sun 14 d15 d16 d17 d18 fr1 9 sat 20 sun 21 d22 d23 d24 d25
Tx day
(weekend-days marked out as fr, sat, sun)

TABLE 15.

ALCOHOL CONSUMPTION
(week-days & week-ends)

Week of treatment	ACTIVE		SHAM	
	week-days	week-end	week-days	week-end
week 1	3.20 (3.95)	3.78 (4.38)	3.60 (4.13)	4.62 (5.13)
week 2	3.37 (3.90)	3.25 (4.47)	3.61 (4.65)	4.69 (5.28)
week 3	2.77 (3.70)	2.71 (3.43)	2.60 (3.90)	4.30 (4.52)
week 4	2.01 (3.18)	not available	2.73 (4.24)	not available

week-days - represents average consumption from Monday to Thursday
week-end - represents average consumption of Friday, Saturday and Sunday
In parenthesis - Standard Deviations

TABLE 16.

WEEKLY AVERAGE CONSUMPTION
(number of standard drinks per day)

Week of treatment	ACTIVE	SHAM
week 1	3.45 (3.96)	4.03 (4.15)
week 2	3.32 (3.58)	4.07 (4.60)
week 3	2.65 (3.01)	3.41 (4.00)
week 4 *	2.01 (3.18)	2.73 (4.24)

In parenthesis - Standard Deviations;
* average of four treatment days only;

It should also be noted that in the sham group, the alcohol consumption was higher than in the active group on 19 out of 25 treatment days. Within the active treatment group, the number of days with increased alcohol consumption was lower than in the sham group. On Mondays, the level of consumption in both groups returned to a lower level. A period of three weeks of therapy is included in the analysis.

The statistical analysis was performed with the Repeated Measures ANOVA (SPSS/PC + computer program). Factors analyzed were: a) kind of treatment: active and sham (between-subjects factor); b) the treatment weeks: week 1, week 2, week 3 (within-subjects factor); c) weekend versus weekdays condition representing subdivision of consumption into weekdays (Monday to Thursday) and weekends (Friday to Sunday); this is the second within-subjects factor (Table 17).

The following null hypotheses were tested:

a) First Null Hypothesis: The mean consumption during the entire treatment period is not significantly different between active and sham treatment groups. This null hypothesis cannot be rejected at .05 level of significance.

b) Second Null Hypothesis: In the sample group as a

table 17. **ALCOHOL CONSUMPTION ANALYSIS - DAILY PATTERN**
(standard output of ANOVA procedure)

Source of variation	SS	DF	MS	F	Sig	
within cells	5013.25	60	83.55			
constant	4620.12	1	4620.12	55.29	**<0.001**	
A	47.92	1	47.92	0.57	0.452	
Source of variation	SS	DF	MS	F	Sig	
within cells	691.26	120	5.76			
B	36.94	2	18.47	3.21	**0.044**	***
AxB	0.75	2	0.38	0.07	0.937	
Source of variation	SS	DF	MS	F	Sig	
within cells	402.50	60	6.71			
C	44.78	1	44.78	6.68	**0.012**	
AxC	29.67	1	29.67	4.42	**0.04**	
Source of variation	SS	DF	MS	F	Sig	
within cells	706.73	120	5.89			
BxC	2.22	2	1.11	0.19	0.828	
AxBxC	6.78	2	3.39	0.58	0.564	

FACTORS OF REPEATED MEASURE ANOVA:

A - type of treatment (active vs. sham, between subjects factor)
B - week of treatment (within subjects factor)
C - weekend (Fr,Sat,Sun) vs. the weekdays (Mo,Tu,We,Th) condition (within subjects factor)
AxB - interaction of week and type of treatment
AxC - interaction of type of treatment with weekend vs. weekdays condition
BxC - interaction of week of treatment with weekend vs. weekdays condition
AxBxC - interaction of type of treatment, week of treatment and weekend vs. weekdays condition

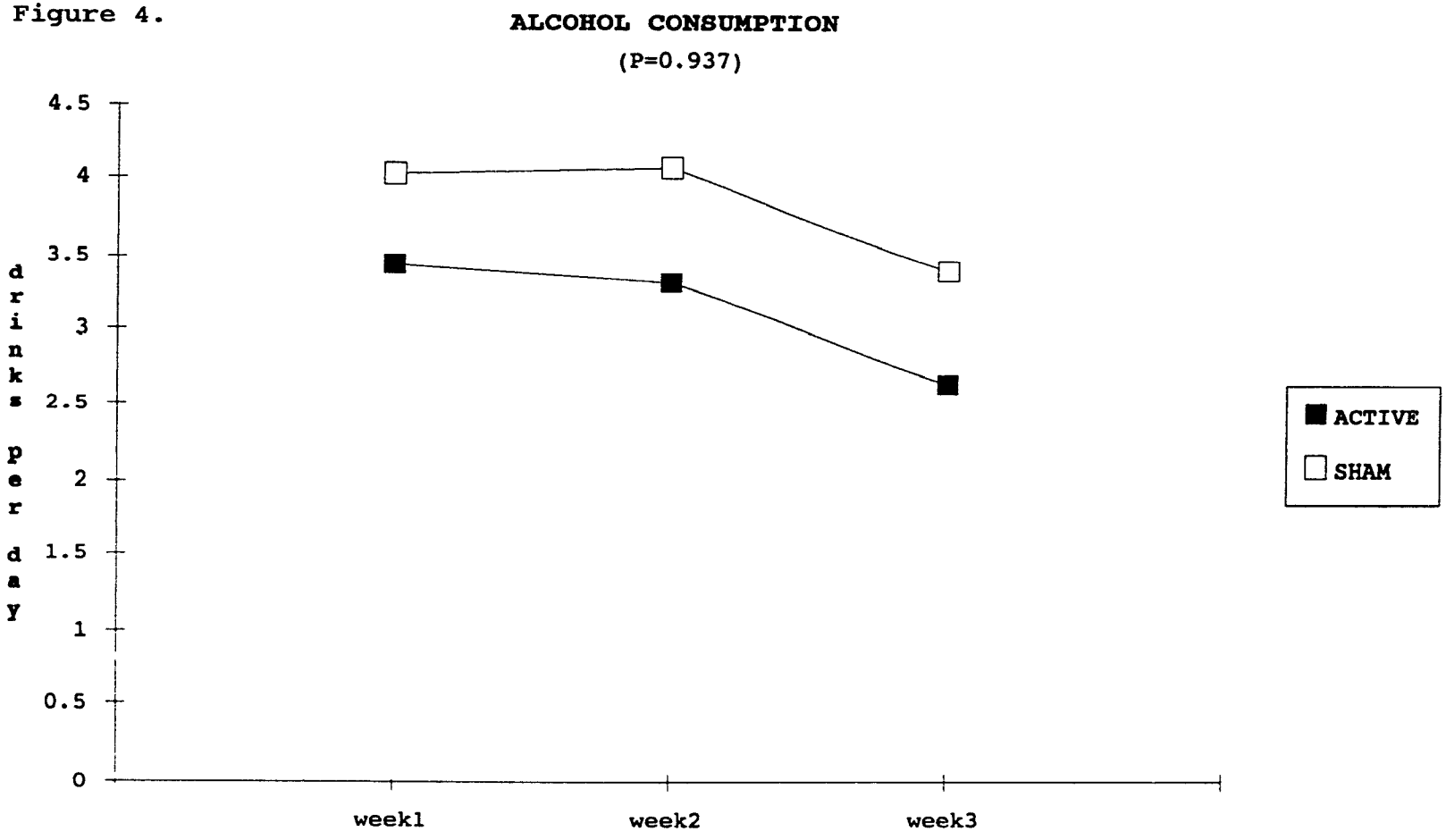
*** Mauchly sphericity test, W=0.926 with significance=0.104; Greenhaus-Geisser Epsilon =0.93
After adjustment of degrees of freedom: numerator df=1.86 and denominator df=111.74
Adjusted F=3.21; significance obtained from tables by interpolation, P=0.05

whole, there is no change in the level of average weekly consumption during treatment (factor B). This null hypothesis may be rejected at the P 0.05 level of significance (with Greenhause-Geisser correction applied to adjust the degrees of freedom). The alternative hypothesis may be stated as follows: in sample group as a whole, the consumption decreased significantly during treatment (See Fig.4).

c) Third Null Hypothesis: There is no interaction between the kind of stimulation and weekly average consumption (interaction AxB). This null hypothesis cannot be rejected; there are no significant differences in weekly average consumption between the treatment groups irrespective of the treatment week (Table 16).

d) Fourth Null Hypothesis: In the sample group as a whole, the average weekend consumption does not differ from the weekdays average (testing factor C). This null hypothesis may be rejected with 0.012 probability of incorrect decision. Tables 15,17, show that there is greater mean level of consumption on weekends compared with weekdays. To explain the above, the following hypothesis may be stated: that the sample group as a whole significantly increased the overall weekend consumption when compared with weekday levels.

Figure 4.


ALCOHOL CONSUMPTION
(P=0.937)
4.5
4
3.5
3
2.5
2
1.5
1
0.5
0
drinks per day
week1
week2
week3
ACTIVE
SHAM

e) Fifth Null Hypothesis: There is no interaction between the kind of stimulation (active, sham) and the weekend versus weekday consumption changes. In other words, the observed increase in weekend consumption is not significantly different between the active and sham group (interaction AxC). This null hypothesis may be rejected at P 0.44 level of significance. Table 15 and Fig.3 show that the increases in weekend consumption are greater in the sham group. An alternative hypothesis can be formulated as follows: the active CES stimulation has an ability to reduce high weekend consumption of alcohol.

f) Sixth Null Hypothesis: In the sample group as a whole, during the entire treatment period, weekends are characterized by a constant increase of alcohol consumption level (interaction BxC). This null hypothesis cannot be rejected.

g) Seventh Null Hypothesis: There is no interaction between the kind of stimulation, week of treatment and weekend versus weekdays factor (interaction AxBxC). This null hypothesis cannot be rejected.

In summary, the following can be stated:

1. The overall average consumption of the treatment period was not significantly different comparing active

and sham groups.

2. Both groups (active and sham) demonstrated a significant (P=0.01) decrease in their weekly average consumption.

3. The treatment groups did not significantly differ in their average weekly consumption.

4. The weekend periods were characterized by a significant increase of alcohol consumption (P=0.01).

5. The active stimulation, as compared with the sham, significantly reduced (P=0.05) weekend consumption.

6. The changes observed in weekend versus weekday consumption presented a constant pattern during the whole therapy.

7. Time did not produce significant changes in the ability of the active group to control weekend consumption, nor did it influence the high weekend consumption in the sham group.

9. Record of days without alcohol consumption

The treatment groups did not significantly differ in the average number of days without alcohol consumption during the treatment period. The analysis, using the independent t-test, showed the following results:

1.Active group: 12.9 days (SD 8.5) 2.Sham group: 12.2 days (SD 8.6)

Significance: P=0.78

10. Abstinence at the end of the treatment period

The proportion of patients able to abstain from alcohol during the last ten days of therapy did not differ significantly between the two treatment groups tested, using Chi-square:

Treatment Group	No. of Patients Abstinent	No. of Patients Non-abstinent
active	6 (10.0% of Group)	21 (35.0% of Group)
sham	6 (10.0% of Group)	27 (45.0% of Group)

Table 18: Abstinence during last ten days of therapy. Analysis included 60 subjects with no missing data. Chi-Square = .15; P=0.70 (before Yates correction).

11. Changes in consumption comparing baseline, treatment and follow-up periods

Table 19 and Fig.5 show the changes (mean values) in consumption during the period preceding the treatment, the three weeks of therapy and during the follow-up period. Forty-three subjects, whose data are available for these periods, have been included in the analysis. The following factors were analyzed:

A Factor: the between-subjects factor is the type of stimulation: either active or sham.

B Factor: the within-subjects factor is the time when the data on consumption were obtained: baseline, at treat-

ment period (three weeks) and follow-up periods (three follow-ups: at month 2, month 4 and month 6).

The following null hypotheses were tested (Table 20):

a) First Null Hypothesis: There is no overall difference between the active and sham group in the amount of alcohol consumed during the entire observation time, i.e. from the baseline to the end of follow-up (testing Factor A). The null hypothesis cannot be rejected with 0.05 significance.

b) Second Null Hypothesis: Consumption in the sample group as a whole does not change significantly during the observation period from the baseline to the end of follow-up, i.e. time does not influence consumption of both experiment groups combined (testing Factor B). The null hypothesis can be rejected with less than 0.001 level of significance. The alternative hypothesis may be formulated that consumption of alcohol changes significantly during the time of observation in the sample group as a whole.

c) Third Null Hypothesis: The active and the sham groups do not differ in the alcohol consumption level in any of the observation periods: baseline, treatment-time and follow-up (AxB interaction). The null hypothesis cannot be rejected.

Figure 5.

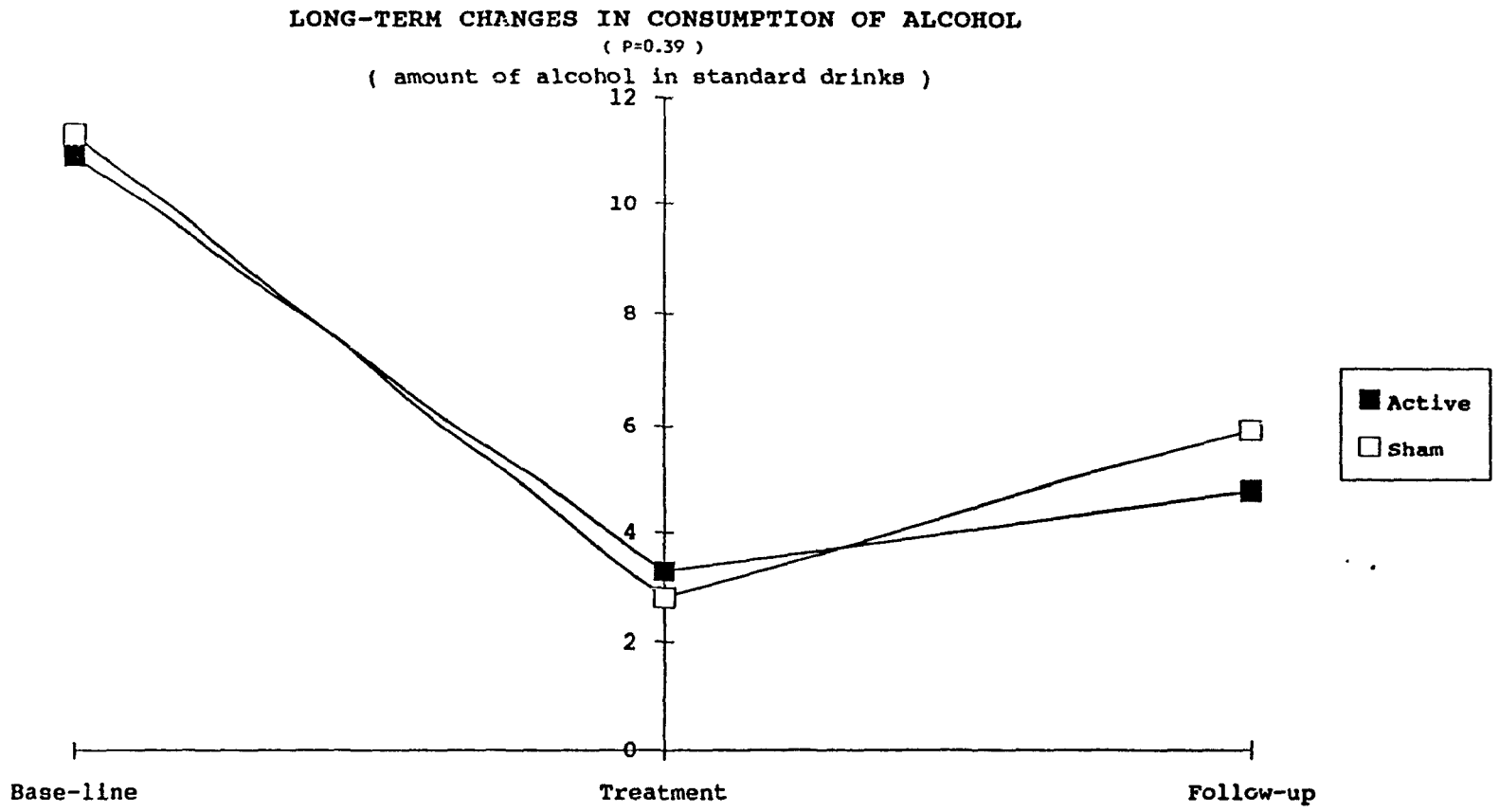

LONG-TERM CHANGES IN CONSUMPTION OF ALCOHOL
(P=0.39)
(amount of alcohol in standard drinks)
12
10
8
6
4
2
0
Base-line
Treatment
Follow-up
Active
Sham

TABLE 19. **ALCOHOL CONSUMPTION: BASELINE, TREATMENT, FOLLOW-UP.**

Average cons.	Active		Sham	
	Mean	SD	Mean	SD
Base-line	10.86	5.38	11.71	5.75
Treatment	3.24	3.62	2.78	3.62
Follow-up	4.73	4.05	5.92	5.19

* note ,that whenever data missing - subject excluded
** count of valid cases : N active=21; N sham=22;

TABLE 20. **ALCOHOL CONSUMPTION: BASELINE, TREATMENT, FOLLOW-UP.**
(analysis of variences)

Source of variation	SS	DF	MS	F	Sig.
within cells	1552.33	41	37.86		
constant	5513.04	1	5513.04	145.61	**<0.001**
A	9.00	1	9.00	0.24	0.628

Source of variation	SS	DF	MS	F	Sig.	
within cells	1150.75	82	14.03			
B	1566.19	2	783.09	55.80	**<0.001**	***
AxB	16.37	2	8.19	0.58	0.56	

A - kind of treatment (active; sham).
B - time factor (baseline; 3 weeks of treatment; 3 follow-up).
*** Mauchly sphericity test, W=0.9105 ; Greenhause-Geisser Epsilon = 0.918;
Adjusted degrees of freedom : numerator df=1.84 ; denominator df = 75.28 ;
Adjusted F = 55.67; significance of F obtained from tables by extrapolation, P <0.001;

Long term consumption changes - Summary of results:

1. The treatment groups did not differ in their overall average consumption during the entire observation time (from baseline to end of follow-up).

2. Alcohol consumption in the sample group as a whole strongly depended on the measurement period. As shown in Table 13 and Fig. 5 , in both groups high baseline consumption decreased considerably during treatment period and showed a tendency to increase after completing the therapy. These changes were statistically significant. Post-hoc analysis with paired t-tests showed that, after the therapy, patients in the sample group as a whole significantly increased their consumption; nevertheless, consumption level was significantly lower than at the baseline.

3. There was no significant difference in the average alcohol consumption between the active and sham treatment groups at the baseline, during treatment period or during follow-up.

12. Analysis of craving for alcohol

Data obtained during four weeks of treatment (five-treatment-days per week) were analyzed using the Repeated Measure ANOVA on SPSS/PC+ computer program. The type of given stimulation was the between-subjects factor (Factor A);

Figure 6.

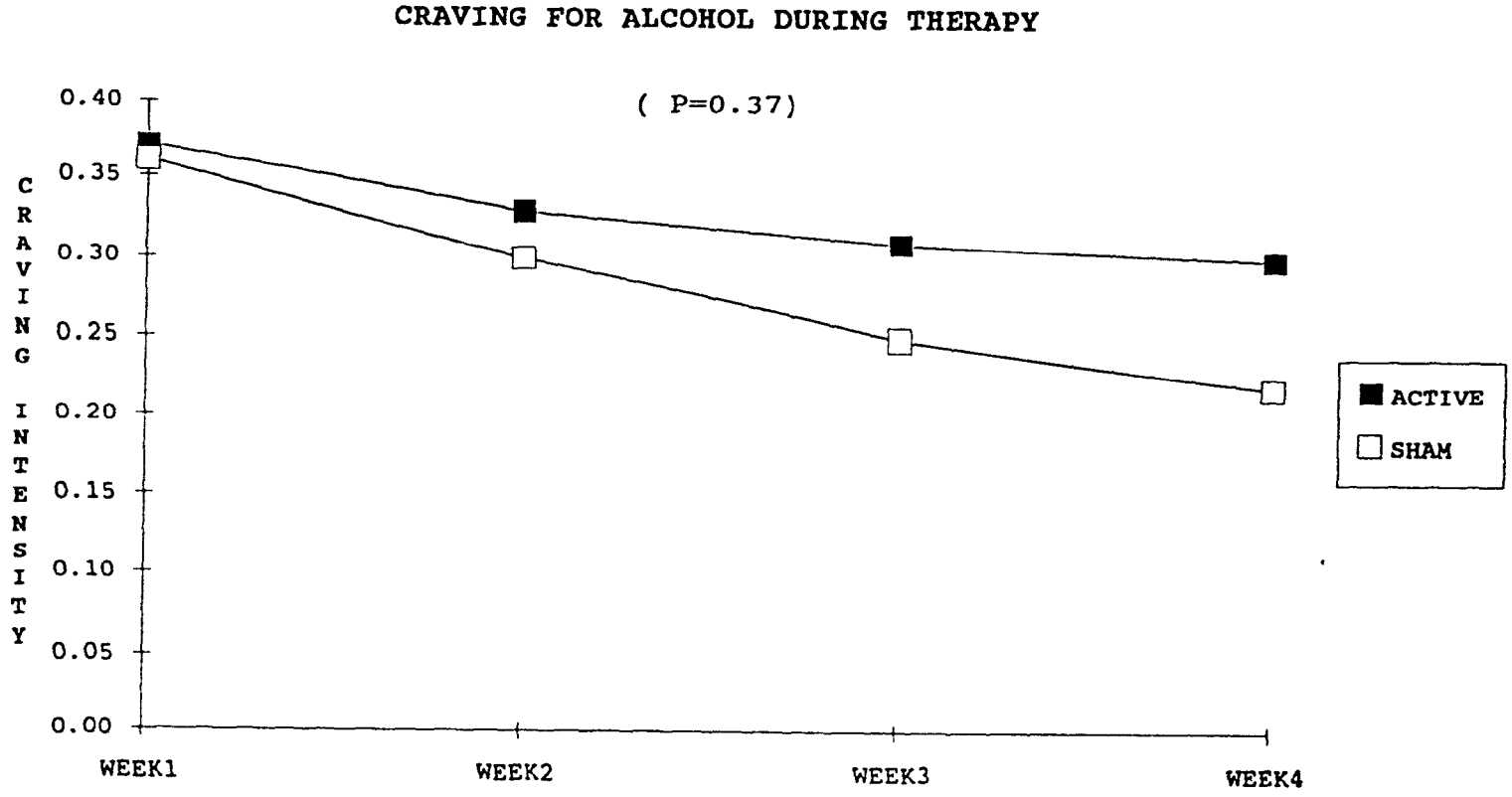

CRAVING FOR ALCOHOL DURING THERAPY
(P=0.37)
CRAVING INTENSITY
0.40
0.35
0.30
0.25
0.20
0.15
0.10
0.05
0.00
WEEK1
WEEK2
WEEK3
WEEK4
ACTIVE
SHAM

TABLE 21. **ANALYSIS OF CRAVING FOR ALCOHOL (during 4 weeks* of treatment)**

Source of variation	SS	DF	MS	F	Sig
within cells	7.37	60	0.12		
constant	23.03	1	23.03	187.55	**<0.001**
A	0.12	1	0.12	1	0.321

Source of variation	SS	DF	MS	F	Sig	
wihin cells	2.25	180	0.01			
B	0.41	3	0.14	10.93	**<0.001**	***
AxB	0.04	3	0.01	1.06	0.369	

* measures of craving were done during treatment-days i.e. five days/week
A - kind of treatment :active vs sham; (betweensubjects factor)
B - factor of time: week of the therapy ; (withinsubjects factor)
*** Mauchly sphericity test, W = 0.5698 significance < 0.001 ; Greenhouse-Geisser Epsilon = 0.767
Adjusted degrees of freedom : numerator df = 138.06 ; denominator df = 2.30 ; Adjusted F = 9.00 ;
Significance of adjusted F obtained by interpolation from tables, P <0.001 ;

the week of treatment was the within-subjects factor (Factor B, Time factor). Table 21 shows the results.

The following null hypotheses were tested:

a) First Null Hypothesis: The overall average craving during the whole treatment period does not differ significantly between the treatment groups (testing Factor A). As shown in Table 21, the null hypothesis cannot be rejected.

b) Second Null Hypothesis: Four weeks of treatment does not produce significant changes in craving for alcohol in the sample group as a whole (testing Factor B). The null hypothesis may be rejected with 0.001 level of significance. The alternative hypothesis may be stated as follows: Craving for alcohol during four weeks of therapy decreased significantly in the sample group as a whole.

c) Third Null Hypothesis: There is no interaction between the type of stimulation and weekly average craving for alcohol (AxB interaction). The null hypothesis cannot be rejected at the .05 level of significance.

Craving for alcohol - Summary of results:

As shown in Fig. 6, a decreasing tendency in craving is observed in both treatment groups. The sample group as

a whole showed a significant decrease in craving for alcohol, although statistical analysis demonstrated no significant difference between the two groups.

13. Analysis of sleeping hours during the therapy period

The analysis included calculation of average sleeping hours obtained on the basis of five treatment days per week. (See Fig.7 and Table 22).

The type of treatment (active or sham) was the between-subjects factor (Factor A).

The time factor (Factor B) was the within-subjects factor (four weeks of therapy). Repeated Measure ANOVA with SPSS computer program was used.

The following null hypotheses were tested:

a) First Null Hypothesis: There is no overall difference between the active and sham group in average of sleeping hours.

b) Second Null Hypothesis: The sample group as a whole does not show significant time changes in the average sleeping hours during four weeks of therapy (Factor B).

c) Third Null Hypothesis: There is no interaction between the weekly average of sleeping hours and the kind of stimulation (interaction AxB).

None of the above null hypotheses can be rejected

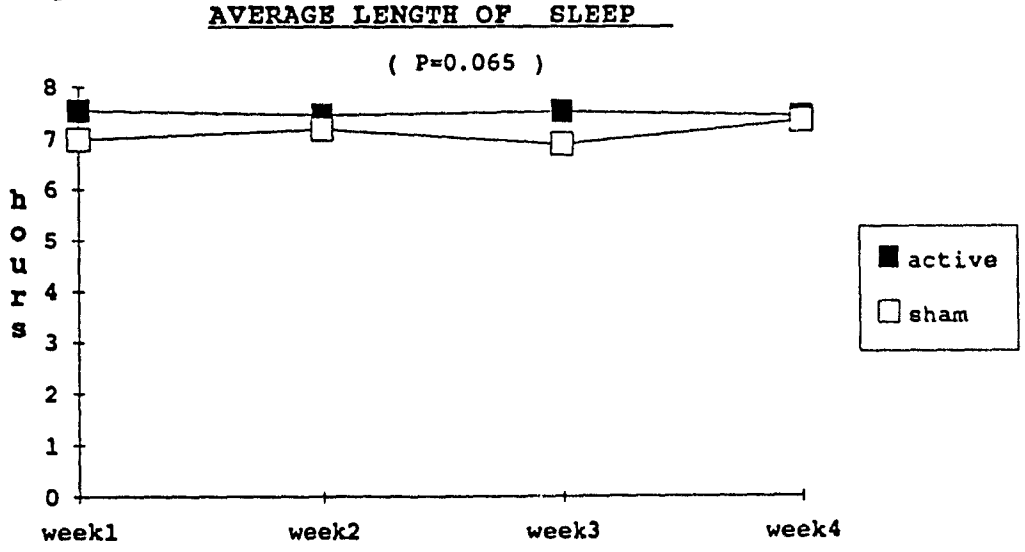
TABLE 22. AVERAGE LENGTH OF SLEEP DURING THERAPY
(analysis of variences)

source of variation	SS	DF	MS	F	Sig
within cells	261.75	59	4.44		
constant	12751.9	1	12751.9	2874.31	<0.001
A	9.5	1	9.5	2.14	0.149

source of variation	SS	DF	MS	F	Sig
within cells	68.93	177	0.39		
B	0.98	3	0.33	0.84	0.474
AxB	2.86	3	0.95	2.45	0.065

factor A - kind of treatment (active , sham);
factor B - week of treatment (4 weeks);

Figure 7.



at .05 significance level.

Sleeping hours - Summary of results

Neither the active treatment nor the sham procedure produced any significant changes in the length of sleep. A trend, to sleep longer, was observed in the active group (AxB interaction, P=0.065).

14. Follow-up analysis

Three variables were the subject of investigation: average consumption, craving for alcohol and average sleeping hours. The craving was estimated according to the five point scale. The follow-up data represent an average of the week preceding phone interviews. The interviews were carried out at 8, 16 and 24 weeks after completion of the therapy (See Tables 23,24,25). Statistical analysis was carried out using the Repeated Measure of ANOVA procedure (SPSS/PC+ computer program).

The following factors were tested:

The kind of previously received treatment stimulation represents a between-subjects factor (Factor A).

The procedure of first, second and third follow-up interview is the repeated measure: within-subjects factor (Factor B).

The above-mentioned factors and their interaction have

FOLLOW-UP ANALYSIS

TABLE 23. **alcohol consumption**

Source of variation	SS	DF	MS	F	Sig
within cells	2680.37	41	65.37		
constant	3658.92	1	3658.9	55.97	**<0.001**
A	45 96	1	45.96	0 7	0 407

Source of variation	SS	DF	MS	F	Sig
within cells	1119.99	82	13.66		
B	35.84	2	17.92	1.31	0.275
AxB	44.53	2	22.26	1.63	0 202

TABLE 24. **sleeping hours**

Source of variation	SS	DF	MS	F	Sig
within cells	126.89	36	3.52		
constant	5559.53	1	5559.5	1577 3	<0 001
A	1.64	1	1 64	0 46	0 5

Source of variation	SS	DF	MS	F	Sig
within cells	69.55	72	0.97		
B	0.66	2	0.33	0 34	0.712
AxB	1 29	2	0 65	0 67	0 516

TABLE 25. **craving auto-evaluation**

Source of variation	SS	DF	MS	F	Sig
within cells	119.59	39	3.07		
constant	363.1	1	363.1	118 41	**<0.001**
A	5 02	1	5 02	1 64	0 208

Source of variation	SS	DF	MS	F	Sig
within cells	77.34	78	0.99		
B	4.61	2	2 31	2 33	0 104
AxB	0 06	2	0 03	0 03	0 971

A - kind of treatment :active vs.sham; (between subjects factor).
B - time factor: followup interview ; (within subject factor).

been analyzed. For each variable (consumption, sleeping hours, craving), the following null hypotheses were tested:

a) First Null Hypothesis: There is no difference between the treatment groups in the overall post-treatment averages of consumption, sleeping hours and craving (Factor A).

b) Second Null Hypothesis: In the sample group as a whole, there is no significant time change in the variables examined during the follow-up period (Factor B).

c) Third Null Hypothesis: There is no interaction between time and type of stimulation in any of the variables examined: alcohol consumption, craving, sleeping hours (interaction AxB).

None of the above-stated null hypotheses can be rejected at the .05 level of significance.

Follow-up analysis - Summary of results

From the 8th week, after completing the treatment, through to week 16 and week 24, there was no significant difference between the sham and active treatment groups in any of the variables examined (alcohol consumption, craving, sleeping hours).

CHAPTER V - DISCUSSION

In spite of some established therapeutic approaches, the treatment of alcoholism remains controversial. This situation may be due to unclear etiology and the lack of unequivocally-accepted diagnostic criteria. At the present time, alcoholism is considered to be a syndrome rather than a well-defined clinical condition. DSM III-R does not employ the term "alcoholism", but rather defines it as a phenomena of alcohol-dependence and alcohol-abuse. Long-term studies have demonstrated a complexity of factors affecting the development of alcohol dependence and abuse in some individuals, while others seem to be protected.

The heterogeneous character of alcoholism would suggest the need for a more individualized therapeutical approach (Dongier 1989). Social, psychological and biological factors are implicated to a different degree in each case. The biological factors may possibly contain a genetic component.

The classical therapeutic approach relies on several general assumptions. It consists of a pharmacological intervention such as the use of benzodiazepines, phenytoine, neuroleptics, to control the withdrawal symptoms during detoxification. It is usually followed by psycho-

social renabilitation period, usually attendance at Alcoholic Anonymous (AA) meetings. Behavioral therapy (controlled drinking) and various other forms of psychotherapy are used alternatively.

Etiologically directed pharmacological adjuvants to the existing treatments rely on the effects of alcohol abuse on the brain neurotransmitter system which is possibly responsible for the dependence phenomena. Serotoninergic, dopaminergic, GABA-ergic and opiatergic systems seem to be involved. Pharmacologically specific modification of these systems remains as yet in the experimental stage (Naranjo et al 1984,1987, Lhuintre et al 1985, Dongier 1989). From this point of view, the ideal medication would eliminate or reduce the withdrawal symptoms and craving for alcohol without inducing cross-dependence.

In the present study, an attempt was made to evaluate a possible alternative biological intervention - the Cranial Electrotherapy Stimulation. At the current state of knowledge concerning the mechanism of action of CES, several hypotheses may be considered, including previously-mentioned changes in the brain neurotransmitters. The obvious advantage of CES when compared to the pharmacological interventions include a lack of side effects, an absence of inter-

action with alcohol and an easier acceptance by such influential groups as Alcoholic Anonymous (AA). AA is opposed to any use of "drugs" to fight a drug addiction (alcohol dependence), irrespective of whether the medication is potentially addictive (e.g. benzodiazepines) or not.

Concurrent psychopathology and psychological distress, aggravated during withdrawal period, present a major problem in the treatment of alcoholism. Depression frequently coexists with prolonged alcohol abuse. According to Cadoret (1979), depression is observed in up to 60 percent of alcoholics; yet their reciprocal relation remains controversial and difficult to disentangle. Both alcoholism and depression, as a primary or secondary condition, are significantly involved in the vicious circle of distress and abuse. Often, an alcoholic erroneously uses alcohol as a "self-medication" attenuating symptoms of depression immediately after consumption. The exacerbation of symptoms to even greater extent, as a long-term consequence of alcohol abuse, is not realized. Episodes of depression frequently occur in alcoholic patients during detoxification, even if they previously did not suffer from mood disturbances. During the first year after detoxification,

alcoholic patients still show a tendency to mood swings ranging from depression to anxiety. These dysphoric states are partly responsible for relapses. A therapeutic intervention to alleviate depression would undoubtedly be of value.

Our current study revealed the ability of CES to alleviate psychological distress. The improvement of depression in the actively-treated group, as measured by SCL-90R, significantly exceeded the result of the sham procedure. Changes in the other subscales of SCL-90R, though not statistically different in both treatment groups, were consistently more positive in the active group. These results, undoubtedly, contributed to the significantly greater improvement in the active treatment group when considering the global measure of psychological distress - the Positive Symptoms Distress Level.

Interestingly, the improvement of depression, as measured by the Hamilton Psychiatric Rating Scale for Depression, was not significantly greater in the active group. This somewhat paradoxical finding can perhaps be explained by the fact that Hamilton Psychiatric Rating Scale for Depression is heavily influenced by somatic symptoms of depression, which may be confused with the

complaints caused by alcohol abuse and by related organic problems.

Both treatment groups showed highly significant improvements on the Hamilton Anxiety Scale from the baseline, although the active stimulation did not produce significantly better results than the sham.

It is striking that, in spite of a lack of significance in both Hamilton Scales, the total scores (as well as the factors constituting them) were consistently more favorably affected in the active treatment group than in the sham group.

The analysis of consumption on a daily basis showed that the high weekend consumption formed a constant pattern in the sham treatment group, which was not the case in the active group. The reduction of weekend alcohol consumption in the active group, as compared with the sham group, reached the level of statistical significance ($p=0.04$). Weekends may represent an increased risk of alcohol consumption because of no work obligations and social encouragement. The active stimulation seemed to counteract this risk factor. The effects of active stimulation on consumption carried over during the following Saturdays and Sundays, which were the days without treatment. The other

aspects of drinking behavior analyzed during the study were the number of days when the patients were able to abstain from alcohol and the final result of the treatment measured as an abstinence at the end of the therapy. The active treatment group did not differ from the sham group in the average number of days without alcohol consumption. When the last ten days of treatment are considered, the proportions of completely abstinent patients at the end of the experiment were not significantly different between the treatment groups. CES was not shown to be significantly better than sham procedure in controlling weekly average consumption. Craving and sleeping hours were not significantly affected by the type of stimulation.

A time effect was observed in both treatment groups at the level of alcohol consumption. The consumption had decreased dramatically from the highest baseline level reaching the lowest value during the therapy period. During the follow-up period, consumption increased but it remained lower than at the baseline.

The follow-up revealed no difference in alcohol consumption level between the active and sham groups.

No significant differences between the treatment groups were found in the degree of improvement in Diagnos-

tic Interview Schedule (DIS) - section testing for organic brain syndrome, in Drinking Behavior Inventory (DBI), and in the laboratory tests: Mean Corpuscular Volume (MCV) and serum Gamma-Glutamyl Transferase (GGT). No significant time changes in DIS may correspond to the overall low scoring of the patients in the Organic Brain Syndrome (OBS) section. None of these patients met the diagnosis of OBS.

With respect to DBI, patients in both treatment groups improved significantly during the treatment period, independent of the kind of received stimulation. A significant decrease in GGT observed in both groups during the treatment period may reflect a concomitant strong reduction in alcohol consumption.

The study group was characterized by a high compliance and cooperativeness. The five patients (7.5%), who dropped out during the therapy, were all from the active treatment group. Possibly, the reasons for dropping out from the program were not related to the treatment itself. Changes in housing, work, death in the family, were stated as motives by the patients.

The present study corroborates the safety of Cranial Electrotherapy Stimulation (CES) intervention and the lack of side effects attributable to the active stimulation.

Subjects in both groups described the treatment sessions as relaxing and helpful in regaining control over alcohol consumption. In many instances, alcohol abuse during weekend periods was explained as being due to social events such as social gatherings with family or friends and parties. Whether the results would be improved if the treatment was continued during weekends remains hypothetical.

Finally, the strong therapeutic effect of participation in our study should be emphasized. This resulted in high overall improvement irrespective of whether an active or a sham procedure was used.

CHAPTER VI - CONCLUSIONS

1) Consumption of alcohol

Cranial Electrotherapy Stimulation affected the weekly pattern of alcohol consumption by significantly reducing alcohol abuse during weekends. The weekly average consumption was decreased independently of the type of stimulation. A carry-over effect of Cranial Electrotherapy Stimulation on drinking behavior should be considered since no treatment was administered during weekends, which constitute a risk factor for higher alcohol consumption.

2) Associated psychopathological conditions

The present study reveals a significant effect of Cranial Electrotherapy Stimulation in alleviating depression symptoms and the subjectively experienced, psychological distress, as measured by depression subscale and by Positive Symptoms Distress Level of Self-Checklist-90 Revised (SCL90R). Changes in other subscales of SCL90R, as well as Hamilton Psychiatric Rating Scale for Depression (HPRSD) and Hamilton Anxiety Scale (HAS) were not significantly different between the sham and the treatment groups, though a consistent tendency to more pronounced improvement was observed in the actively treated group.

The fact that co-existing psychopathological conditions improve, irrespective of what treatment procedure is used, indicates that they may be related to the dramatic reduction of alcohol consumption.

3) Craving for alcohol

In both groups, craving during the treatment period decreased significantly to the same extent.

4) Effect on sleep

No significant changes were observed in average sleeping hours although a trend towards longer sleep was noticed in the active treatment group.

5) Alcohol-related social and biological impairment

The scores in Drinking Behavior Inventory (DBI) and level of Serum Gamma-Glutamyl Transferase (SGGT) were significantly decreased in both treatment groups (active and sham). These tests indicated decreased social impairment and improved liver function during the treatment period. These changes are related to the reduction of alcohol consumption independent of the type of stimulation.

6) Side-effects and safety of Cranial Electrotherapy Stimulation

The safety of CES and the lack of side effects, attributable to CES, have been demonstrated.

7) Complexity of alcohol-related problems

It appears that psychobiological correlations as well as social factors interact and influence patients' drinking behavior, especially when treatment is carried out on an outpatient basis. The current study revealed the complexity of problems related to the accurate assessment of results in the treatment of alcoholism during clinical trials.

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A P P E N D I X

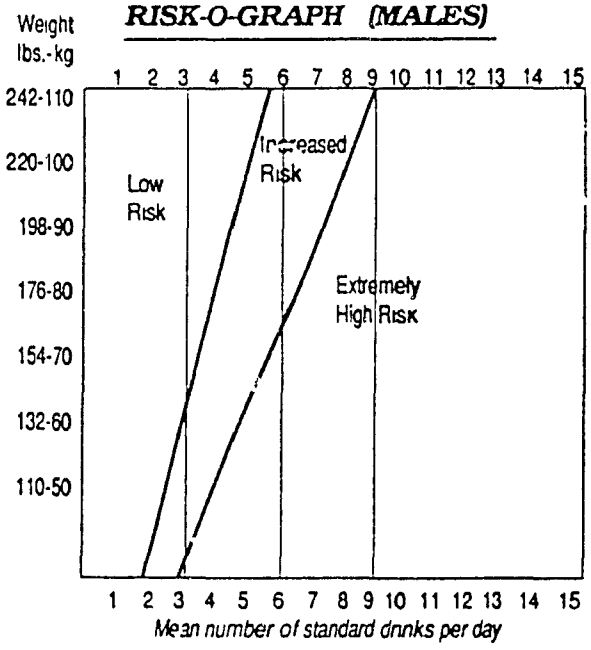
APPENDIX "A"

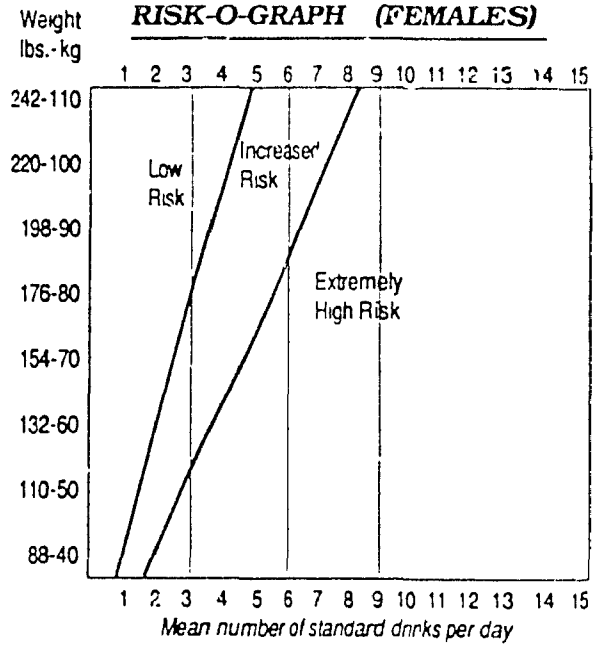
ALCOHOL EQUIVALENTS (STANDARD DRINKS)

Beverage	Usual Serving	Standard Drinks per Serving	Usual Size Bottle	Standard Drinks per Bottle
Beer (5%)	340 mL (12 oz)	1 0	340 mL (12 oz)	1 0
Table Wine	140. mL (5 oz)	1.0	750 mL (26 4 oz)	5 3
			1000 mL (35 2 oz)	7 0
			1500 mL (52 8 oz)	10 6
Fortified Wine	99 mL (3 5 oz)	1 0	750 mL (26 4 oz)	7 5
Spirits	43. mL (1 5 oz)	1.0	340 mL (12 oz)	8 0
			710 mL (25 oz)	16 6
			1135. mL (40 oz)	26 6

Conversion Factor 1 Imperial ounce = 28.4 mL

1 Standard Drink = 340 mL (12 oz) Beer
140 mL (5 oz) Table Wine
99 mL (3 5 oz) Fortified Wine
43 mL (1 5 oz) Spirits





Risk-O-Graph

A graphic representation of the Risk of Physical Damage from alcohol consumption as a function of average daily alcohol intake and body weight for a person of medium fat content. For fatter persons (less body water by proportion) the risk is greater For leaner individuals the risk is a bit less For healthy people the best evidence indicates that consumption of more than 3 or 4 standard drinks daily by an individual of 70 kg (155 lbs) carries an increasing risk to health. Consumption of 6 or more "standard drinks" daily by the same individual is generally accepted as the level at which definite physical damage begins to accrue These graphs assume a steady rate of daily ingestion however, one may have a low average daily intake yet still drink hazardously on sporadic occasions This carries great risk of acute physical damage, accidents and death even though average daily consumption is in the minimal risk range.

The effects of alcohol depend on the amount taken (dose), the frequency of consumption, the rate of absorption, the metabolic rate, the body weight, the proportion of body water, and the general state of health of the consumer. These factors all vary somewhat not only between individuals but within a given individual at different times However, with few exceptions, body weight and dose X frequency are the major relevant variables which predict the risk of damage from drinking in healthy individuals The longer a given level of drinking is maintained (weeks, months, years) the greater the probability of permanent damage.

The figure provides a graphic representation of the risks of physical damage associated with various levels of alcohol consumption for individuals of various weights. However it must be recognized that with certain types of diseases the risk increases faster than the graph indicates It should be emphasized that the boundaries between risk levels are arbitrary and the increasing risk is part of a continuum When in doubt one should err on the side of caution in assessing the hazard of physical damage.

Note. For females, consumption of alcohol during pregnancy, especially during the first trimester, can contribute significantly to the development of Fetal Alcohol Syndrome (FAS)

APPENDIX "B" (1)

Initials._____
Code:_____
Date:_____

MICHIGAN ALCOHOLISM SCREENING TEST (MAST)

1 Do you feel you are a normal drinker?

yes	no	
0	2	_____

2. Have you ever awakened the morning after some drinking the night before and found that you could not remember a part of the evening before?

yes	no	
2	0	_____

3 Does your wife (or parent) ever worry or complain about your drinking?

yes	no	
1	0	_____

4 Can you stop drinking without a struggle after one or two drinks?

yes	no	
0	2	_____

5 Do you feel bad about your drinking?

yes	no	
1	0	_____

6. Do friends or relatives think you are a normal drinker?

yes	no	
0	2	_____

7. Do you ever try to limit your drinking to certain places?

yes	no	
0	0	_____

8 Are you always able to stop drinking when you want to?

yes	no	
0	2	_____

9 Have you ever attended a meeting of Alcoholics Anonymous (AA)?

yes	no	
5	0	_____

10 Have you gotten into fights when drinking?

yes	no	
1	0	_____

11. Has drinking ever created problems with you and your wife?

yes	no	
2	0	_____

12. Has your wife (or other family member) ever gone to anyone for help about your drinking?

yes	no	
2	0	_____

13. Have you ever lost friends or girlfriends/boyfriends because of drinking?

yes	no	
2	0	_____

APPENDIX "B" (2)

Initials _____
Code _____
Date _____

14 Have you ever gotten into trouble at work because of drinking?

yes	no
2	0

_____

15. Have you ever lost a job because of drinking?

yes	no
2	0

_____

16. Have you ever neglected your obligations, your family, or your work for two or more days in a row because you were drinking?

yes	no
2	0

_____

17 Do you ever drink before noon?

yes	no
1	0

_____

18 Have you ever been told you have liver trouble? Cirrhosis?

yes	no
2	0

_____

19. Have you ever had delirium tremens (DTs), severe shaking, heard voices or seen things that weren't there after heavy drinking?

yes	no
2	0

_____

20 Have you ever gone to anyone for help about your drinking?

yes	no
5	0

_____

21. Have you ever been in a hospital because of drinking?

yes	no
5	0

_____

22. Have you ever been a patient in a psychiatric ward of a general hospital where drinking was part of the problem?

yes	no
2	0

_____

23. Have you ever been seen at a psychiatric or mental health clinic, or gone to a doctor, social worker, or clergyman for help with an emotional problem in which drinking had played a part?

yes	no
2	0

_____

24. Have you ever been arrested, even for a few hours, because of drunk behaviour?

yes	no
2	0

_____

25. Have you even been arrested for drunk driving or driving after drinking?

yes	no
2	0

_____

TOTAL: _____

Initials:_____
Code:_____
Date:_____

DRINKING BEHAVIOUR INTERVIEW (DBI)

Shelton, Hollister and Gocka
(First part of three)

Type of Drinking

1. Number of days per week some alcoholic beverage taken:
   Less than 3 days (0) More than or equal to 3 days (1)

2. Daily average intake of alcoholic beverage when drinking:
   Score 1 for each 3 ounces of hard liquor
   9 ounces of wine
   24 ounces of beer

3. Any use of non-beverage alcohol (bay rum, rubbing)
   (Be subtle First ploy: Anything else you've ever drunk?)

   None (0) Yes (5)

4. Drinks taken at work (not socially in business)
   (Exclude business luncheon or dinner, business cocktail party; really refers to sneak drinks at work)

   None (0) Yes (2)

5. Number of times "drunk" past two months:
   (Obviously a matter of some definition. State number of times you've had enough to drink which you would have been arrested for drunk driving if you had been driving and were stopped)

   0 (0) 1 (3) 2 (6) 3 (9) 4 (12) 5 (15)

APPENDIX "C" (2)

Initials _____
Code: _____
Date: _____

6. Longest period of time between drinks (hours).
(This question aims at abstinence of less than 24 hours)
If less than 12 hours, score 2

More than 12 hours	Less than 12 hours	
(0)	(2)	_____

7. Longest period of uninterrupted drinking (hours).
If more than 6 hours, score 2.

Less than 6 hours	More than 6 hours	
(0)	(2)	_____

8. Days a week in which "eye-opener" drink taken.

0	Positive answer	More than 1 day	
(0)	(2)	(3)	_____

9. Days a week in which a surreptitious "sneak" taken

0	Positive answer	More than 1 day	
(0)	(2)	(3)	_____

10. Number of meals a week missed because of drink.

0	Positive answer	1 to 3	More or equal to 4	
(0)	(1)	(2)	(3)	_____

11. Percent of time drinks alone.

Less than 25%	More than 25%	More than 50%	
(0)	(1)	(2)	_____

12. Number of "black-outs" in past two months.
(Periods for which he has no memory)

0	1	2	3	4	
(0)	(3)	(6)	(9)	(12)	_____

13. Number of "shakes" in past two months.
(Severe tremors: also include marked anxious states)

0	1	2	3	4	
(0)	(3)	(6)	(9)	(12)	_____

Initials:_____
Code:_____
Date:_____

14. Number of illnesses attibutable to drinking past 2 months.
(Some may be dismissed as "colds" or intestinal flu)

0	1	2	3	4	
(0)	(3)	(6)	(9)	(12)	_____

15. Number of severe hangovers in past two months.
(Did he need to take aspirin the next morning, or skip breakfast?)

0	1	2	3	4	5	6	
(0)	(2)	(4)	(6)	(8)	(10)	(12)	_____

16. Number of arrests associated with drinking past six months.
(Any kind: driving, disturbing the peace, etc.)

0	1	2	3	
(0)	(5)	(10)	(15)	_____

17 Number of injuries attributable to drinking in past 6 months.
(Many of these will be vague. a burn which he can't account for)

0	1	2	3	4	
(0)	(3)	(6)	(9)	(12)	_____

SCL—90—R

INSTRUCTIONS:

Below is a list of problems and complaints that people sometimes have. Please read each one carefully. After you have done so, please fill in one of the numbered circles to the right that best describes HOW MUCH DISCOMFORT THAT PROBLEM HAS CAUSED YOU DURING THE PAST WEEK INCLUDING TODAY. Mark only one numbered circle for each problem and do not skip any items. If you change your mind, erase your first mark carefully. Read the example below before beginning, and if you have any questions please ask the technician.

SEX

MALE ○

FEMALE ○

Initials _____

Code _____

Date _____

DATE			ID. NUMBER	AGE
MO	DAY	YEAR		

VISIT NUMBER _____

EXAMPLE HOW MUCH WERE YOU DISTRESSED BY	NOT AT ALL	A LITTLE BIT	MODERATELY	QUITE A BIT	EXTREMELY
1. Bodyaches	⓪	①	②	●	④

HOW MUCH WERE YOU DISTRESSED BY		NOT AT ALL	A LITTLE BIT	MODERATELY	QUITE A BIT	EXTREMELY
1.	Headaches	1	⓪	①	②	③	④
2.	Nervousness or shakiness inside	2	⓪	①	②	③	④
3.	Repeated unpleasant thoughts that won't leave your mind	3	⓪	①	②	③	④
4.	Faintness or dizziness	4	⓪	①	②	③	④
5.	Loss of sexual interest or pleasure	5	⓪	①	②	③	④
6.	Feeling critical of others	6	⓪	①	②	③	④
7.	The idea that someone else can control your thoughts	7	⓪	①	②	③	④
8.	Feeling others are to blame for most of your troubles	8	⓪	①	②	③	④
9.	Trouble remembering things	9	⓪	①	②	③	④
10.	Worried about sloppiness or carelessness	10	⓪	①	②	③	④
11.	Feeling easily annoyed or irritated	11	⓪	①	②	③	④
12.	Pains in heart or chest	12	⓪	①	②	③	④
13.	Feeling afraid in open spaces or on the streets	13	⓪	①	②	③	④
14.	Feeling low in energy or slowed down	14	⓪	①	②	③	④
15.	Thoughts of ending your life	15	⓪	①	②	③	④
16.	Hearing voices that other people do not hear	16	⓪	①	②	③	④
17.	Trembling	17	⓪	①	②	③	④
18.	Feeling that most people cannot be trusted	18	⓪	①	②	③	④
19.	Poor appetite	19	⓪	①	②	③	④
20.	Crying easily	20	⓪	①	②	③	④
21.	Feeling shy or uneasy with the opposite sex	21	⓪	①	②	③	④
22	Feelings of being trapped or caught	22	⓪	①	②	③	④
23.	Suddenly scared for no reason	23	⓪	①	②	③	④
24.	Temper outbursts that you could not control	24	⓪	①	②	③	④
25.	Feeling afraid to go out of your house alone	25	⓪	①	②	③	④
26.	Blaming yourself for things	26	⓪	①	②	③	④
27.	Pains in lower back	27	⓪	①	②	③	④
28.	Feeling blocked in getting things done	28	⓪	①	②	③	④
29.	Feeling lonely	29	⓪	①	②	③	④
30.	Feeling blue	30	⓪	①	②	③	④
31.	Worrying too much about things	31	⓪	①	②	③	④
32.	Feeling no interest in things	32	⓪	①	②	③	④
33.	Feeling fearful	33	⓪	①	②	③	④
34.	Your feelings being easily hurt	34	⓪	①	②	③	④
35.	Other people being aware of your private thoughts	35	⓪	①	②	③	④

Please continue on the following page ▶

APPENDIX "D" (2)

Initials _____ Code: _____ Date: _____

			NOT AT ALL	A LITTLE BIT	MODERATELY	QUITE A BIT	EXTREMELY
36.	Feeling others do not understand you or are unsympathetic	36	0	1	2	3	4
37.	Feeling that people are unfriendly or dislike you	37	0	1	2	3	4
38	Having to do things very slowly to insure correctness	38	0	1	2	3	4
39.	Heart pounding or racing	39	0	1	2	3	4
40	Nausea or upset stomach	40	0	1	2	3	4
41.	Feeling inferior to others	41	0	1	2	3	4
42	Soreness of your muscles	42	0	1	2	3	4
43.	Feeling that you are watched or talked about by others	43	0	1	2	3	4
44	Trouble falling asleep	44	0	1	2	3	4
45.	Having to check and double-check what you do	45	0	1	2	3	4
46.	Difficulty making decisions	46	0	1	2	3	4
47.	Feeling afraid to travel on buses, subways, or trains	47	0	1	2	3	4
48.	Trouble getting your breath	48	0	1	2	3	4
49.	Hot or cold spells	49	0	1	2	3	4
50.	Having to avoid certain things, places, or activities because they frighten you	50	0	1	2	3	4
51.	Your mind going blank	51	0	1	2	3	4
52	Numbness or tingling in parts of your body	52	0	1	2	3	4
53.	A lump in your throat	53	0	1	2	3	4
54	Feeling hopeless about the future	54	0	1	2	3	4
55.	Trouble concentrating	55	0	1	2	3	4
56.	Feeling weak in parts of your body	56	0	1	2	3	4
57.	Feeling tense or keyed up	57	0	1	2	3	4
58.	Heavy feelings in your arms or legs	58	0	1	2	3	4
59.	Thoughts of death or dying	59	0	1	2	3	4
60	Overeating	60	0	1	2	3	4
61.	Feeling uneasy when people are watching or talking about you	61	0	1	2	3	4
62.	Having thoughts that are not your own	62	0	1	2	3	4
63.	Having urges to beat, injure, or harm someone	63	0	1	2	3	4
64	Awakening in the early morning	64	0	1	2	3	4
65.	Having to repeat the same actions such as touching, counting, or washing	65	0	1	2	3	4
66.	Sleep that is restless or disturbed	66	0	1	2	3	4
67.	Having urges to break or smash things	67	0	1	2	3	4
68.	Having ideas or beliefs that others do not share	68	0	1	2	3	4
69.	Feeling very self-conscious with others	69	0	1	2	3	4
70.	Feeling uneasy in crowds, such as shopping or at a movie	70	0	1	2	3	4
71.	Feeling everything is an effort	71	0	1	2	3	4
72.	Spells of terror or panic	72	0	1	2	3	4
73.	Feeling uncomfortable about eating or drinking in public	73	0	1	2	3	4
74.	Getting into frequent arguments	74	0	1	2	3	4
75.	Feeling nervous when you are left alone	75	0	1	2	3	4
76.	Others not giving you proper credit for your achievements	76	0	1	2	3	4
77.	Feeling lonely even when you are with people	77	0	1	2	3	4
78	Feeling so restless you couldn't sit still	78	0	1	2	3	4
79.	Feelings of worthlessness	79	0	1	2	3	4
80	The feeling that something bad is going to happen to you	80	0	1	2	3	4
81.	Shouting or throwing things	81	0	1	2	3	4
82.	Feeling afraid you will faint in public	82	0	1	2	3	4
83.	Feeling that people will take advantage of you if you let them	83	0	1	2	3	4
84	Having thoughts about sex that bother you a lot	84	0	1	2	3	4
85.	The idea that you should be punished for your sins	85	0	1	2	3	4
86.	Thoughts and images of a frightening nature	86	0	1	2	3	4
87.	The idea that something serious is wrong with you body	87	0	1	2	3	4
88.	Never feeling close to another person	88	0	1	2	3	4
89.	Feelings of guilt	89	0	1	2	3	4
90	The idea that something is wrong with your mind	90	0	1	2	3	4

APPENDIX "E" (1)

Initials _____
Code _____
Date _____

DOUGLAS HOSPITAL

HAMILTON PSYCHIATRIC RATING SCALE FOR DEPRESSION

INSTRUCTIONS: For each item select the "cue" which best characterizes the patient

1. **DEPRESSED MOOD** (Sadness, hopless, helpless, worthless)
   - 0 - Absent
   - 1 - These feeling states indicated only on questioning
   - 2 - These feeling states spontaneously reported verbally
   - 3 - Communicates feeling states non-verbally - i e , through facial expression, posture, voice, and tendency to weep
   - 4 - Patient reports **VIRTUALLY ONLY** those feeling states in his spontaneous verbal and non-verbal communication

2. **FEELINGS OF GUILT** ____
   - 0 - Absent
   - 1 - Self-reproach, feels he has let people down
   - 2 - Ideas of guilt or rumination over past errors or sinful deeds
   - 3 - Present illness is a punishment Delusions of guilt
   - 4 - Hears accusatory or denunciatory voices and/or experiences threatening visual hallucinations

3. **SUICIDE** ____
   - 0 - Absent
   - 1 - Feels life is not worth living
   - 2 - Wishes he were dead or any thoughts of possible death to self
   - 3 - Suicidal ideas or gesture
   - 4 - Attempts at suicide *(any serious attempt rates 4)*

4. **INSOMNIA EARLY** ____
   - 0 - No difficulty falling asleep
   - 1 - Complains of occasional difficulty falling asleep - i e , more than 1/2 hour
   - 2 - Complains of nightly difficulty falling alseep

5. **INSOMNIA MIDDLE** ____
   - 0 - No difficulty
   - 1 - Patient complains of being restless and disturbed during the night
   - 2 - Waking during the night - any getting out of bed rate 2 *(except for purposes of voiding)*

6. **INSOMNIA LATE** ____
   - 0 - No difficulty
   - 1 - Waking in early hours of the morning but goes back to sleep
   - 2 - Unable to fall asleep again if he gets out of bed

7. **WORK AND ACTIVITIES** ____
   - 0 - No difficulty
   - 1 - Thoughts and feelings of incapacity, fatigue or weakness related to activities, work or hobbies
   - 2 - Loss of interest in activity: hobbies or work - either directly reported by patient or indirectly in listlessness, indecision and vacillation *(feels he has to push self to work or activities)*
   - 3 - Decrease in actual time spent in activities or decrease in productivity In hospital, rate 3 if patient does not spend at least three hours a day in activities *(hospital jobs or hobbies)* exclusive of ward chores
   - 4 - Stopped working because of present illness In hospital, rate 4 if patient fails to perform ward chores unassisted

____

Initials:_____
Code._____
Date._____

8. **RETARDATION** (Slowness of thought and speech; impaired ability to concentrate; decreased activity)
   - 0 - Normal speech and thought
   - 1 - Slight retardation at interview
   - 2 - Obvious retardation at interview
   - 3 - Interview difficiult
   - 4 - Complete stupor

9. **AGITATION** _____
   - 0 - None
   - 1 - "Playing with" hands, hair, etc.
   - 2 - Hand wringing, nail-biting, hair pulling, biting of lips

10. **ANXIETY PSYCHIC** _____
   - 0 - No difficulty
   - 1 - Subjective tension and irritability
   - 2 - Worrying about minor matters
   - 3 - Apprehensive attitude apparent in face or speech
   - 4 - Fears expressed without questioning

11. **ANXIETY SOMATIC** _____

   Physiological concomitants of anxiety, such as: Gastrointestinal: dry mouth, wind, indigestion, diarrhea, cramps, belching
   Cardiovascular: palpitations, headaches
   Respiratory: hyperventilation, sighing
   Urinary frequency
   Sweating

   - 0 - Absent
   - 1 - Mild
   - 2 - Moderate
   - 3 - Severe
   - 4 - Incapacitating

12. **SOMATIC SYMPTOMS GASTROINTESTINAL** _____
   - 0 - None
   - 1 - Loss of appetite but eating without staff encouragement. Heavy feelings in abdomen
   - 2 - Difficulty eating without staff urging Requests or requires laxatives or medication for bowels or medication for G.I symptoms

13. **SOMATIC SYMPTOMS GENERAL** _____
   - 0 - None
   - 1 - Heaviness in limbs, back or head. Backaches, headaches, muscle aches. Loss of energy and fatigability
   - 2 - Any clear-cut symptom rates 2

14. **GENITAL SYMPTOMS** _____

   Symptoms such as: loss of libido, menstrual disturbances

   - 0 - Absent
   - 1 - Mild
   - 2 - Severe

15. **HYPOCHONDRIASIS** _____
   - 0 - Not present
   - 1 - Self-absorption *(bodily)*
   - 2 - Preoccupation with health
   - 3 - Frequent complaints, requests for help, etc.
   - 4 - Hypochondriacal delusions

_____

Initials _____
Code _____
Date _____

16. **LOSS OF WEIGHT** *Rate either a or b*

a. When rating by history:
0 - No weight loss
1 - Probably weight loss associated with present illness
2 - Definite *(according to patient)* weight loss
3 - Not assessed

b. On weekly ratings by ward psychiatrist, when actual weight changes are measured
0 - Less than 1 lb. weight loss in week
1 - Greater than 1 lb weight loss in week
2 - Greater than 2 lb. weight loss in week
3 - Not assessed

17. **INSIGHT** _____
0 - Acknowledges being depressed and ill
1 - Acknowledges illness but attributes cause to bad food, climate, overwork, virus, need for rest, etc
2 - Denies being ill at all

18 **DIURNAL VARIATION** _____

a. Note whether symptoms are worse in morning or evening. If NO diurnal variation, mark none
0 - No variation
1 - Worse in A.M.
2 - Worse in P.M.

b. When present, mark the severity of the variation. Mark "None" if NO variation
0 - None
1 - Mild
2 - Severe

19. **DEPERSONALIZATION AND DEREALIZATION** _____
0 - Absent *Such as. feelings of unreality, nihilistic ideas*
1 - Mild
2 - Moderate
3 - Severe
4 - Incapacitating

20. **PARANOID SYMPTOMS** _____
0 - None
1 - Suspicious
2 - Ideas of reference
3 - Delusions of reference and persecution

21. **OBSESSIONAL AND COMPULSIVE SYMPTOMS** _____
0 - Absent
1 - Mild
2 - Severe

_____

APPENDIX "F"

Initials:_____
Code:_____
Date:_____

DOUGLAS HOSPITAL

HAMILTON ANXIETY SCALE

INSTRUCTIONS: Rate each symptom construct by the term which best describes the patient's present condition

0 = Not Present 1 = Mild 2 = Moderate 3 = Severe 4 = Very Severe

1.	ANXIOUS MOOD Worries, anticipation of the worst, fearful anticipation, irritability	0 1 2 3 4
2.	TENSION: Feelings of tension, fatigability startle response, moved to tears easily, trembling, feelings of restlessness, inability to relax.	0 1 2 3 4
3	FEARS. Of dark, of strangers, of being left alone, of animals, of traffic, of crowds.	0 1 2 3 4
4	INSOMNIA. Difficulty in falling asleep, broken sleep, unsatisfying sleep and fatigue on waking, dreams, nightmares, night terrors	0 1 2 3 4
5	INTELLECTUAL Difficulty in concentration, poor memory.	0 1 2 3 4
6.	DEPRESSED MOOD: Loss of interest, lack of pleasure in hobbies, depression, early waking, diurnal swing.	0 1 2 3 4
7.	SOMATIC (Muscular): Pains and aches, twitchings, stiffness, myoclonic jerks, grinding of teeth, unsteady voice, increased muscular tone	0 1 2 3 4
8.	SOMATIC (Sensory): Tinnitus, blurring of vision, hot and cold flushes, feelings of weakness, pricking sensation.	0 1 2 3 4
9.	CARDIOVASCULAR SYMPTOMS: Tachycardia, palpitations,pain in chest, throbbing of vessels, fainting feelings. sighing, dyspnea	0 1 2 3 4
10.	RESPIRATORY SYMPTOMS: Pressure or constriction in chest, choking feelings, sighing, dyspnea	0 1 2 3 4
11	GASTROINTESTINAL SYMPTOMS: Difficulty in swallowing, wind, abdominal pain, burning sensations, abdominal fullness, nausea, vomiting, borborygmi, looseness of bowels, loss of weight, constipation	0 1 2 3 4
12.	GENITOURINARY SYMPTOMS: Frequency of micturition, urgency of micturition, amenorrhea, menorrhagia, development of frigidity, premature ejaculation, loss of libido, impotence.	0 1 2 3 4
13	AUTONOMIC SYMPTOMS: Dry mouth, flushing, pallor, tendency to sweat, giddiness, tension headache, raising of hair.	0 1 2 3 4
14.	BEHAVIOUR AT INTERVIEW: Fidgeting, restlessness or pacing, tremor of hands, furrowed brow, strained face, sighing or rapid respiration, facial pallor, swallowing, etc	0 1 2 3 4