THE EFFECT OF CEREBRAL ELECTROTHERAPY ON PATIENTS WITH ANXIETY NEUROSIS

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THE EFFECT OF CEREBRAL ELECTROTHERAPY ON PATIENTS WITH ANXIETY NEUROSIS

by

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ABSTRACT

The efficacy of cerebral electrotherapy in relieving the symptoms of anxiety neurosis was investigated in ten subjects with this condition. A blind cross-over experimental design, in which subjects received five consecutive days of active and five days of placebo treatment was employed, the order being counterbalanced. The subjects' experience of cutaneous sensation was identical for both treatment conditions. Anxiety levels were measured pre- and post-treatment using daily physiological and psychological measures. Weekly psychological measures, some of which were double-blind, were also obtained before and after each type of treatment as well as on one-week and one-month follow-up days. The results showed a statistically significant overall treatment effect, but no differences between active and placebo treatment. There was a post hoc finding of a significant correlation between response to the overall treatment procedure and high levels of extraversion as measured by the Eysenck Personality Inventory (EPI). The implications of these findings are that the therapeutic effectiveness of CET is attributable to the non-specific or placebo components of the treatment procedure and not to the direct effect of the electrical current on the brain. Furthermore, the personality dimensions of the EPI may be useful in predicting the degree of response to non-specific components inherent in the CET treatment procedure.

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CHAPTER I

INTRODUCTION

This chapter will begin with an overview of the origins and development of cerebral electrotherapy (CET) followed by a description of the technique and its variations. The theories of mode of action will then be examined, followed by an evaluation of the controlled studies of clinical effects of cerebral electrotherapy. This will lead to a discussion of the target disorder chosen for this study of CET effectiveness, and different ways of measuring anxiety from both a psychological and physiological point of view. The relevance of physiological findings will then be discussed. The rationale for this study will then be presented followed by the formulation of hypotheses relating CET and anxiety as they emerge from this review of the literature.

Overview

Electrosleep, or cerebral electrotherapy (CET) is a somatic therapy characterized by the passage of a low-amplitude, pulsating direct electrical current around and through the cranium. Originally, electrosleep was intended to induce a state of natural sleep, "a state of consciousness grossly indistinguishable from ordinary sleep, produced by the direct action of a weak rhythmic current on the brain

of a cooperative subject in a non-distracting environment" (Boblitt, 1969, p. 9). Pavlov is said to have provided the concept and rationale for electrically produced sleep therapy (Boblitt, 1969; Obrosow, 1959). His concept of cerebral protective inhibition was based on his work with dogs who were observed to experience

> a hypnotic-like period between the states of sleep and wakefulness that is characterized by changes in the processes of excitation and inhibition of the brain cells...Pavlov showed that the hypnotic phase may occur in the animal during a period of silence and rest as well as while under the influence of a prolonged, monotonous and weak stimulus of the central nervous system, the effects of which are intensified under conditions of comfort. A pulsating electric current was one such stimulus (Obrosow, 1959, p. 180).

The protective inhibition of artificial sleep was thought to be essential for combatting "overexhaustion of the nervous system. Sleep therapy affords the brain cells the best opportunity for complete rest and restoration" (Obrosow, 1959, p. 185). Investigations into the therapeutic effects of low-level direct electrical currents have been going on since the 19th century, and included in the techniques were those of electronarcosis, "a forced state of motor and sensory blockage, usually with loss of consciousness", and electroanesthesia, "a forced state of total anesthesia with or without total loss of consciousness and paralysis" (Boblitt, 1969, p. 9). The modern line of electrosleep therapy as a technique distinct from electronarcosis and electroanesthesia began with work done by the Soviet researcher Gilarojwskii in the early 1950s (Lewis, 1966).

The view that the sole purpose of electrosleep therapy was to induce a sleep-like state to promote functional recovery of cerebral cells influenced the type of research carried out in the Soviet Union and Europe. Most of it has been presented at the International Symposia for Electrosleep and Electroanesthesia, the first being held in 1966, with subsequent meetings in 1969 and 1972. It was after the first Symposium that the notion of a therapeutic, protective, artificial sleep was gradually replaced by the idea that the direct action of the current itself was the healing force: "Our experience has shown that sleep in the course of the individual session is not an absolute condition of success of therapy...the passage of the pulse current through the brain is of greater importance for a curative effect than the achievement of the condition of sleep in one course of treatment" (Van Poznak, 1969, p. 507). Accordingly, Wageneder proposed adopting the term cerebral electrotherapy (CET) to replace electrosleep in that it more accurately reflected the type of treatment involved (Wageneder & St. Schuy, 1970).

It was after the first International Symposium in 1966 that North American researchers became interested in CET. Before this, most of the work on CET had been done in the Soviet Union and Europe. Translations of these works revealed sweeping claims for the beneficial effects of CET in a wide variety of disorders in the fields of psychiatry, surgery, dermatology, obstetrics, and pediatrics, but experimental controls were inadequate on non-existent

(Van Poznak, 1969). The American interest in CET research marked the beginning of a somewhat more objective assessment of its effects.

The American and European studies included investigations of physiological effects produced by CET using human as well as animal subjects in assessing gastric secretion (Reigel, 1970; Wilson, Reigel, Unger, Larson & Sances, 1970), brain waves (Lechner, 1966; Itil, Gannon, Akpinar & Hsu, 1971; Weiss, 1973), a variety of autonomic nervous system responses (Forster, Post & Benton, 1963; Wolff, 1970; Grunner, 1970) and hormonal changes (Rosenthal, 1973). Most of the other studies focused on the target symptoms of chronic anxiety, insomnia and depression (Rosenthal & Wulfsohn, 1970; Frankel, Buchbinder and Snyder, 1973; Flemenbaum, 1974), and occasionally, alcoholism (Tomsovic & Edwards, 1973; Smith & O'Neill, 1975).

Whereas the Soviet and European researchers had presented a united front in their favourable opinion of the beneficial effects of CET, the American investigators were markedly divided in their opinions, in spite of the fact that the focus of CET research in America had been narrowed down to the main target disorders of anxiety, depression and insomnia and to certain physiological effects. In general, this division of opinion holds among authors of uncontrolled studies, as well as among those who used rigid double-blind procedures in their investigations.

Summary

The original rationale for electrosleep therapy or cerebral electrotherapy (CET) as it will be referred to hereafter, lay in Pavlov's concept of protective inhibition which an organism could enter into when faced with exhausting overstimulation. CET was considered to be a safe and simple way to achieve this recovery. In the Soviet Union and Europe, CET has been used to treat almost every kind of medical disorder, but scientifically acceptable trials have not been undertaken. Recent North American interest in CET has led to controlled studies of specific effects, the results of which have, so far, been inconclusive.

General CET Technique

A review of the literature reveals that CET encompasses a rather bewildering array of procedural parameters which may, in part, explain the contradictory findings and confusing opinions about its effectiveness. Everyone seems agreed that the best treatment setting is a quiet, darkened room in which the patient lies comfortably on a bed. A pair of electrodes is placed either directly on the eyelids (Lewis, 1966) or on the brow (Brown, 1975). A second pair is usually placed over the mastoids. The felt pads attached to the electrodes are either soaked in water (Weiss, 1973), a saline solution (Feighner, Brown & Olivier, 1973) or prepared with saline paste (Hearst, Cloninger, Crews &

Cadoret, 1974). The forehead electrodes are cathodes and those at the mastoids are the anodes, (Rosenthal, 1972a), but in some studies they are reversed (Marshall & Izard, 1974). Straus (1964) and others have used direct current (DC) and Tomsovic et al. (1973) and other researchers have used alternating current (AC). Sometimes a DC bias is added to the DC type of current (Marshall et al., 1974) and the AC type as well (Itil et al., 1971). The pulse frequency measured in impulses per second or Hertz (Hz) may vary from 30 Hz (Straus, 1964) to 100 Hz (Rosenthal, 1972a), with a pulse width ranging from 1 to 2 milliseconds (msec) (Straus, 1964). The supply voltage may vary from 10 to 120 volts. The current amplitude at which tingling is usually felt ranges from 0.1 to 0.5 milliamperes (ma). These current parameters depend on the type of CET device used, and no two American-made models seem to possess the same electrical characteristics (Brown, 1975). Treatment sessions have been known to vary from 30 minutes for one week (Rosenthal, 1972a) to two hours for several months (Wageneder, Iwanovsky & Dodge, 1969).

Patients require no specific preparation for CET. After the electrodes are applied the current is turned on, and the amplitude is increased slowly, to avoid any unpleasant sensation, until the level is reached at which a slight tingling sensation occurs. Some clinicians then reduce the current to a level at which the patient experiences no cutaneous sensation.

CET is an attractive treatment because it is said to have no cumulative side effects (Weiss, 1973), complications or contraindications, to be non-toxic and usable along with drugs and other therapies and to be a simple procedure to carry out (Weinberg, 1969). However, side effects such as blurring of vision, thought to be the result of electrode pressure on the eye, and dizziness, as well as slight burns on the skin at the electrode sites have been reported (Koegler, Hicks & Barger, 1971; Rosenthal, 1970; Frankel, 1974). Moreover, contraindications such as epilepsy, blood diseases, malignant tumours, cerebrovascular disorders and heart disease (Chumakova & Kirillova, 1976) and various forms of psychosis (Rosenthal, 1972b) have been reported.

Summary

CET technique involves a number of procedural options in electrode placement, current parameters and length of treatment. There are conflicting claims about side effects and contraindications.

Mode of Action

Central to the argument of CET efficacy is the theoretical question of its mode of action. There are two opposing schools of thought:

(1) The effects of CET treatment are due to the direct action of the current on the cerebral cells. Rush et al.

(1968), in their work on a theoretical model of current flow in the human head, assumed that the current entered the cranium via the frontal surface electrodes. Results indicated that 45% of the electrical output actually entered the brain. Those who favour the direct effect theory hold that the current traversing the brain induces protective inhibition which creates favourable recovery of cerebral cells along with sedation and normalization of the central nervous system (CNS) processes (Banshchikov, 1967; Brand, 1970).

(2) The effects of CET treatment are due to the indirect action of the current. The normalization of only the peripheral autonomic elements of the nervous system are involved and the effect on the CNS is a secondary one involving a variety of mechanisms (Dodge, 1967; Iwanovsky & Dodge, 1968). There are:

(a) relaxation, attributable to lying down in the setting of quietness, comfort and semidarkness.

(b) sensory stimulation, whereby the rhythmic cutaneous sensations experienced in treatment may alone account for the clinical effects. Such sensory stimuli have been found to induce sleep (Lovell & Morgan, 1942; Oswald, 1960).

(c) suggestion or placebo effect, whereby patients who are referred by physicians to undergo a special type of therapy involving neither drugs nor psychotherapy are under the impression that this represents a new type of cure for their specific ailment. Often they are told that they will

get better, and furthermore they are aware that no effort beyond lying quietly and accepting the supposedly healing current is required.

The proponents of the direct effect theory have mainly been the Soviet and European researchers who were influenced by the original Pavlovian concept of protective inhibition. Kalinowsky (1961) suggested the therapeutic usefulness of CET lay in the "rhythmic nature of a peripheral stimulation." Iwanovsky et al. (1968) described a Soviet work in which the author conclusively stated that CET was a rhythm therapy in which the electrical current provided a kind of "electromassage" which polarized cells and normalized tissue metabolism. The Soviet researcher Banshchikov was reported to have demonstrated conclusively that the effects of CET were due to current parameters and not to such variables as electrode pressure on the eye or reflex responses (Iwanovsky et al., 1968). The European researcher Brand (1970) asserted that although very little was known about the actual physiological mechanisms of CET, its therapeutic effect, characterized by feelings of general soothing and relaxation was one of sedation and normalization of CNS processes. The notion that a possible placebo effect played an unimportant role in CET research was promulgated by Giljarowskii, who in the words of Frankel (1974) "probably influenced many of the subsequent investigators to adopt a similar attitude--one that was to be reflected time and again in data interpretation, technological innovations and

experimental designs" (p. 95).

Reviewers of CET literature have repeatedly stressed the importance of the elements of suggestion inherent in a procedure which calls for patients to lie comfortably in a quiet, darkened room and submit to a treatment which they are told will relax them (Lewis, 1966; Boblitt, 1969; Frankel, 1974).

The question of direct versus indirect effect theories becomes relevant when considering experimental procedures designed to test CET efficacy.

In the initial double-blind studies the placebo treatment conditions were noticeably different from active treatment conditions. As Hearst et al. (1974) stated:

> In these studies the active treatment group received electrical current to the skull and also experienced a 'tingling' peripheral sensation at the electrode site throughout each treatment session whereas the control group received neither electrical current nor peripheral stimulation. Such control is probably adequate if one assumes that the therapeutic modality is a direct cerebral effect of electrical current. ...if suggestion, setting, and peripheral stimulation are in fact, the critical therapeutic variables it is not surprising that patients who experience a tingling sensation throughout each session should do better than the others who experience no peripheral stimulation. (p. 463)

It is apparent that a control for peripheral stimulation will be critical for any study which aims to evaluate CET.

Summary

There are two conflicting theories of the mode of action of CET. The first is that the current has a direct

effect upon the brain cells. The second attributes any improvement to an indirect effect induced by the setting, apparatus, suggestion and peripheral stimulation. A controlled study must therefore take account of these factors.

Double-blind Studies

The majority of studies conducted in CET research failed to employ basic methodological principles needed in the investigation of a somatic therapy. The eight double-blind studies reported here are the exception, although even some of these were not entirely free of methodological flaws.

In four of these reports, significant differences between the effects of active versus placebo treatment were found (Straus, Elkind & Bodian, 1964; Rosenthal, 1972; Weiss, 1973; Feighner et al., 1973). In the other, and most recent, four works, no significant differences were found (Tomsovic et al., 1973; Hearst et al., 1974; Marshall et al., 1974; Moore, Mellor, Standage & Strong, 1975). These findings will now be examined in regard to the variety of methodological variables employed in these studies. Table 1 lists the studies, findings and methodological variables used.

An ideal double-blind procedure is one in which the subject, the operator administering CET and the individual assessing CET effects on the dependent variables are all completely unaware of which treatment, active or placebo, is being given. The necessity for an ideal double-blind procedure is outlined by Frankel (1974):

> The patient's response to CET treatment could also be affected by the daily verbal and non-verbal communications he receives from the CET operator while the operator is adjusting the electrodes and current levels. The communications are undoubtedly influenced, at least in a subtle way, by the operators knowledge of whether he is administering active or placebo CET. He too, like the patient, should ideally be blind... (p. 96)

Such procedures require an intermediary to operate the machine only, while another individual would interact only with subjects. An ideal double-blind procedure is probably best achieved with the use of specially built machines with hidden switches (Weiss, 1973; Frankel, 1974). Perhaps, because of the need for extra staff and equipment, only two of the eight double-blind studies were carried out under ideal double-blind conditions (Weiss, 1973; Hearst et al., 1974). In the former study, significant differences between active and placebo CET were found; in the latter study no significant differences were noted.

In many of the eight double-blind studies, placebo treatment conditions were also far from ideal. In four of these studies placebo treatment involved briefly turning on the CET machine at the beginning of a session so that subjects could feel an initial tingling, and then turning off the machine for the duration of the session (Straus et al., 1964; Weiss, 1973; Feighner et al., 1973; Tomsovic et al., 1973). Since subjects in active treatment presumably felt a tingling sensation throughout the session (Weiss asserts they did not in his study), and those in placebo treatment did not, the two conditions were not identical. In Rosenthal's

study, subjects in placebo treatment felt no tingling, but heard the same loud timer used in active treatment. Of these five studies only Feighner's employed a cross-over design, so that only subjects in his study were in a position to compare the two types. Of these five works, only one failed to produce significant results (Tomsovic et al., 1973).

In three of the most recent double-blind works, researchers were able to create identical active and placebo CET conditions (Hearst et al., 1974; Marshall et al., 1974; Moore et al., 1975), and in all of these no statistically significant differences found between active and placebo treatment. Two types of identical active and placebo CET conditions were devised. One method was to lower the current to just below the point at which tingling was perceived in active treatment. This would be done after subjects had been allowed to experience an initial tingling sensation. Thus an active treatment condition identical to placebo CET was achieved.

An alternative way of creating identical conditions was to have subjects experience a tingling sensation throughout both active and placebo CET (Marshall et al., 1974). This was done by placing a pair of electrodes, consisting of one positive and one negative element, on the closed eyelids. The usual placebo method of placing electrodes over the mastoid processes but disconnecting them from the machine was employed. Thus current flowed between the inch of skin separating the frontal electrodes, creating a tingling

TABLE 1

Eight double-blind studies and their main methodological variables

Authors	Significant difference active vs placebo	Target condition	Type of instruction	Cross-over	Ideal double-blind	Electrode location	Current parameters	Peripheral stimulation: active	Peripheral stimulation: placebo	Treatment duration
Straus et al. (1964)	YES (N=34)	insomnia	not reported	NO	NO	+ eyelids - mastoids	DC 30-40 Hz 1.8-2.0 msec	TT	TI	6-12 sessions 30 min. ea.
Rosenthal (1972)	YES (N=22)	anxiety depression insomnia	functional suggestion 2 types	NO	NO	- orbits + mastoids	DC 100 Hz 1 msec	TT loud noise	NT loud noise	5 sessions 30 min. ea.
Weiss (1973)	YES (N=10)	insomnia	functional	NO	YES	brow nape of neck	DC (DC bias) 45-240 Hz .4-1.2 msec	TT	TI TO	24 sessions 1-5 min. 2-10 min. ea. 21-15 min. ea.
Feighner et al. (1973)	YES (N=23)	anxiety depression insomnia	suggestion 2 types	YES	NO	eyelids mastoids	DC 100 Hz 1 msec	TT	TI TO	20 sessions 30 min. ea.
Tomsovic et al. (1973)	NO (N=43)	tension head/stomach pain insomnia	not reported	NO	NO	orbital mastoids	AC 100 Hz 2 msec	TT	TI TO	5 sessions 30 min. ea.
Hearst et al. (1974)	NO (N=28)	anxiety depression insomnia	suggestion	NO	YES	- brow + mastoids	AC, DC 100 Hz 2 msec	TI TBT	TI TO	5 sessions 30 min. ea.
Marshall et al. (1974)	NO (N=40)	depression	suggestion	NO	NO	+ eyelids - mastoids	DC (DC bias) 100 Hz 1 msec	TT	TT	5 sessions 30 min. ea.
Moore et al. (1975)	NO (N=10)	anxiety depression insomnia	functional	YES	NO	not reported	DC 100 Hz 1 msec	TI TBT	TI TO	10 sessions 30 min. ea.

Key: TT - tingling throughout; TI - tingling initially; TO - turned off; TBT - treatment below threshold.

sensation virtually indistinguishable from that experienced in active treatment.

To reiterate, no statistically significant differences were found in these three studies. The implications of these results may be very important, and can be related to the two theories of CET mode of action. Those studies in which subjects felt tingling in neither active nor placebo treatment (Hearst et al., 1974; Moore et al., 1975) possibly represented a test of the direct effect theory: the intervening variable of peripheral or rhythmic sensation was not present, therefore the direct effect alone was being tested. No statistically significant differences between active and placebo treatment were found. In the third study, in which subjects experienced tingling in both active and placebo treatment (Marshall et al., 1974), peripheral sensation was held constant. Although both active and placebo treatment groups improved, there was no significant difference between group improvement levels. Thus the indirect effect theory would appear to have been supported once more.

Summary

Eight double-blind studies, investigating mainly the target disorders of anxiety, insomnia and depression solved some of the methodological problems that plagued other studies. Of these, only three created placebo conditions which were identical to those of active treatment. No

significant differences between active and placebo treatment effects were noted in these studies. In four of the five less well-controlled studies, CET was found to have a significant effect.

Anxiety as a Target Disorder

As we have seen, the most common target disorders in North American CET research have been chronic anxiety, depression and insomnia. Rosenthal (1972b) stated that the best results with CET were obtained in patients suffering from anxiety neurosis:

> At the present time the clearest indication for electrosleep therapy are chronic tension states or chronic anxiety neurosis with or without accompanying reactive depression and chronic insomnia. The best results are probably seen when anxiety is not clearly related to an acute environmental stress but has persisted chronically with only partial remission over a long period of time. (p. 104)

According to the American Psychiatric Association's (1968) classification system, chronic anxiety or anxiety neurosis is defined as follows:

> This neurosis is characterized by anxious-over-concern extending to panic and frequently associated with somatic symptoms...anxiety may occur under any circumstances and is not restricted to specific situations or objects. This disorder must be distinguished from normal apprehension or fear which occurs in realistically dangerous situations. (DSM-II, 1968, p. 38)

(A). Psychological measures of anxiety

When speaking of anxiety from a psychological point of view, a distinction is made between an individual's

tendency to feel anxious in certain situations (trait anxiety) and the actual level of anxiety experienced at a given moment in time (state anxiety) (Cattell & Scheier, 1961; Spielberger, Gorsuch & Lushene, 1970). The latter authors describe the concept this way:

> State anxiety (A-State) is conceptualized as a transitory emotional state or condition of the human organism that is characterized by subjective, consciously perceived feelings of tension and apprehension, and heightened autonomic nervous system activity. A-States may vary in intensity and fluctuate over time. Trait anxiety (A-Trait) refers to relatively stable individual differences between people in the tendency to respond to situations perceived as threatening with elevations in A-State... In general, it would be expected that those who are high in A-Trait will exhibit A-State elevations more frequently than low A-Trait individuals... (p. 3).

The authors developed a brief self-report measure of state and trait anxiety named the State-Trait Anxiety Inventory (STAI). The form itself is entitled Self-Evaluation Questionnaire and contains a total of 40 items. The first 20 items comprise STAI form X-1. Instructions printed on the form are to answer items in terms of how one feels "right now, that is, at this moment". There are four statements from which one is to be chosen: "not at all", "somewhat", "moderately so", and "very much so". The remaining 20 items comprise STAI form X-2. Instructions printed on the form require subjects to answer items in terms of how they generally feel. Four statements from which to choose are: "almost never", "sometimes", "often", and "almost always". Evidence for the validity and reliability of the STAI, as well as its proven usefulness in research is reviewed (Spielberger

et al., 1970).

One personality test which has been useful in aiding the diagnosis of anxiety neurosis is the Eysenck Personality Inventory (EPI) (Eysenck & Eysenck, 1968). This inventory measures personality along the dimensions of extraversion-introversion (E) and neuroticism-stability (N). Each trait is measured by means of 24 questions which require a "yes" or "no" response. The inventory includes a response distortion scale (Lie) to detect a tendency to fake responses, and has two parallel forms for multiple administrations.

The N scale of this inventory taps the degree to which an individual tends to be emotionally labile and to over-react; it taps the predisposition to develop neurotic disorders under stress, or trait anxiety. High scores on N and low scores on E are most often found in anxiety neurotics.

Scheier and Cattell (1960) reasoned that elements of both trait and state anxiety may be contained in the same questionnaire item. The Eight Parallel-Form Anxiety Battery (EPFAB) is such a measure in that its parallel forms provide an anxiety score which expresses the differential weighting of a response to an item in the scale while taking into account state and trait factors. The use of parallel forms permits repeated measures to be taken at many closely spaced intervals for the same set of persons: there are a total of eight forms each consisting of seven subtests in which items have a demonstrated high loading on state anxiety. In the standardized sample, the forms were given twice daily with

no systematic effects being noted which were specifically due to length of interval. Construct validity (correlation with the anxiety factor) and inter-form reliability (average correlation with the other seven forms) were found to be best in forms F, D and B, and the authors recommend using these three for optimal stability of measurement. It has been demonstrated that repeated administrations of personality tests may lead to greater reliability in differentiating between individuals (Howard & Diesenhaus, 1965) or may not have any significant influence on test scores (Bendig & Bruder, 1962).

Each of the eight forms contains a total of 50 items contained in its seven subtests. The forms are designated by the letters A through H, and the subtests are entitled as follows: Questions, Annoyances, Skills, Do You Sometimes?, Comments, Checklist, and Embarrassing Circumstances. These subtests tap the following areas: questionnaire items, susceptibility to annoyance, lack of confidence in untried skills, readiness to confess common faults, emotionality of comments, anxiety-tension symptomatic self-checklist, susceptibility to embarrassment. The purpose of the last six subtests is not readily apparent to the subject.

Because of the parallel forms, the EPFAB is seen as an ideal daily measure of anxiety fluctuations. The intervals between testings have ranged from twice daily to more than a week and no effects on score from neither order of administration of forms nor the length of interval between test administration

were found (Bendig, 1962).

(B) Physiological correlates of anxiety

The physiological manifestations of anxiety as described by Freedman, Kaplan and Sadock (1972) include a faster heartbeat than normal not exceeding 100 beats per minute, rapid breathing, flushed neck and face, moist, cold palms, finger tremor and brisk tendon reflexes. Eaton and Peterson (1969) have added to this list muscular tension, excessive perspiration and pupillary dilatation.

Cattell et al. (1961) have found in their research a consistent "autonomic pattern of higher systolic blood pressure, higher heart rate, more rapid respiration" (p. 208) in state anxiety, but not to any appreciable degree in trait anxiety. They state moreover: "It is noteworthy that muscle tension and its consequences in the spinal region and particularly the muscles along the back of the neck and head have long been clinically associated with anxiety..." (p. 205). The relationship of muscle tension to anxiety has also been confirmed by Hamilton (1959) and Buss (1962) who found that the two major symptom groups of muscle tension and autonomic overactivity in two different psychiatric populations were related to anxiety.

Thus an increased heart rate, respiration and raised systolic blood pressure, as well as muscle tension, are seen as the major physiological correlates of state anxiety.

Summary

Anxiety is the chief characteristic of the neuroses (DSM-II, 1968), and as such deserves attention in regard to ways and means of controlling it. Anxiety has been ameliorated with CET application (Rosenthal, 1972b). A distinction has been made between trait anxiety, which refers to a person's proneness to suffer from somatic symptoms of anxiety given a stressful situation, and state anxiety, which refers to the actual level of physiological and psychological correlates of anxiety at a given moment in time. The EPI is a useful diagnostic aid in objectively establishing the existence of anxiety neurosis, the STAI is a reliable measure of both state and trait anxiety and the EPFAB appears to be an ideal tool for the research measurement of anxiety fluctuations.

Physiological Studies

In the evaluation of a somatic therapy, the measurement of physiological effects of treatment provides information about immediate organic changes. If these are found to correlate with subjective changes as measured by various psychological means, the researcher can draw certain conclusions about a given treatment's effectiveness. In CET research there have been many attempts to investigate the physiological process affected by treatment. The Soviet literature in particular is full of such studies and they have been reviewed extensively (Wageneder et al., 1967, 1969,

1970; Dodge, 1967; Iwanovsky et al., 1968; Iwanovsky, 1969). Most of the findings in the studies reviewed indicated there was an apparent direct effect of CET on a number of physiological variables. However, these studies were not reported in conventional journal format and provided few details regarding procedure, and generally no experimental controls appear to have been employed. We will now examine a representative sample of ten European and American studies of physiological effects of CET, as listed in Table 2.

In the studies under consideration, the observation of physiological changes included electroencephalogram variation (EEG), pulse and respiratory rates, blood pressure, gastric secretion, galvanic skin resistance, capillary reactivity and hormone fluctuations, as well as one in which actual current levels in the brain were measured. Two of these works failed to reveal significant results (Lechner, Geyer, Pfurtscheller, St. Schuy and Wageneder, 1966; Reigel, Dallmann, Christman, Hamilton, Luperku, Henschel, Larson & Sances, 1970), although in the latter work, significant effects were noted in measures obtained from monkeys. The former study employed a single-blind cross-over design, something which was done in only two of the other nine studies (Itil et al., 1971; Cox & Heath, 1975). Two studies in which monkey subjects were used to study gastric secretion levels as a result of CET obtained significant results (Reigel et al., 1970; Wilson et al., 1970). In the other eight studies both patient and normal populations were used.

TABLE 2

Ten physiological studies and their main methodological variables

Authors	Significant findings	Subjects	Placebo	Current parameters	Treatment duration	Physiological measures
Forster et al. (1963)	YES	6 patients 17 normals	NO	DC 20-40 Hz .2-.3 msec	1-11 sessions	m. spasticity pulse, respiration blood pressure EEG
Lechner et al. (1966)	NO	5 normals	YES single-blind cross-over	not reported	8 sessions 40-60 min.	EEG
Reigel et al. (1970)	NO YES	15 normals 7 monkeys	NO	15 Hz 80 Hz	25 sessions	gastric secretion pulse, respiration gastric secretion
Wolff (1970)	YES	20 normals	NO	12, 25, 50, 100, 200 Hz	1 sess/freq. 60 min.	pulse, respiration blood pressure
Wilson et al. (1970)	YES	6 monkeys	NO	15 Hz	not reported	gastric secretion
Grunner (1970)	YES	30 patients	YES	DC 14-16 Hz 200 Hz .5 msec	3 weeks	recalorification capillary reactivity GSR
Itil et al. (1971)	YES	10 patients	YES single-blind cross-over	AC (DC bias) 25-120 Hz .5-1 msec	4 sessions 60 min.	EEG
Rosenthal (1973)	YES	41 patients	NO	DC 100 Hz 1 msec	5 sessions 30 min.	serum thyroxine triiodothyronine (T_3) resin uptake
Dymond et al. (1974)	YES	patients N not reported	NO	DC 100 Hz 1 msec	not reported	intracerebral current levels
Cox et al. (1975)	YES	1 patient	YES single-blind cross-over	AC 100 Hz 1 msec	2 sessions 30 min.	EEG

Two investigators examined the effects of CET on certain autonomic nervous system (ANS) processes (Forster et al., 1963; Wolff, 1970). Along with taking measures of blood pressure, respiration and pulse rates, Forster also took note of CET effects on muscle spasticity using patients as well as normal subjects. The normal subjects received only one treatment session of which the duration was not specified. Patients underwent anywhere from one to eleven sessions. Along with EEG recordings, measures of blood pressure, pulse and respiratory rates were taken. Regardless of whether or not sleep actually occurred, there was a decline in the level of all measures during treatment. It is not clear whether these results were significant. However, the effects of CET on muscle spasticity were judged to be clinically noticeable in that an objective decrease was demonstrated. There were no follow-up data.

Although it gave evidence of a positive effect of CET on certain ANS processes, this study did not provide details about the statistical significance of those effects, nor did it indicate whether or not the changes were brief and transitory.

The study conducted by Wolff (1970) investigated the effects of different pulse frequencies on certain ANS measures. The procedure involved taking measures of blood pressure, pulse and respiratory rates before treatment. During treatment, pulse and respiratory rates were monitored every ten minutes during the 60 minutes of CET treatment. These

measures were also monitored three times after treatment with the last measure taken at 30 minutes after the CET machine was turned off. A second blood pressure tracing was done at the same time. In addition, subjects were asked to give their subjective impressions. It is not possible to draw any definite conclusions from this study since there was no control group, although baseline measures were taken. Moreover, although physiological changes were noted as a result of CET treatment, they were not tested for statistical significance.

While this study seems to confirm that physiological changes can result from CET treatment, it is unclear what these changes mean in terms of therapeutic value. Grunner (1970) has stated that it is difficult to evaluate and explain observed changes in regard to the psychophysiological significance. However, an objective psychological evaluation used along with physiological assessment would facilitate an evaluation of observed bodily function changes and possibly explain the therapeutic consequences of such changes. None of the studies under consideration attempted to evaluate psychological changes, if any, with the aid of psychological tools. Subjects were asked to express subjective feelings as a result of treatment, but these were neither quantified nor tested for statistical significance. It would appear that a merging of studies investigating psychological aspects and those evaluating physiological changes attributable to CET is needed in order to arrive at a clearer impression of

the therapeutic value of CET.

Perhaps one of the most important studies of those under consideration was that in which actual current levels in the brain were measured during CET application (Dymond, Coger & Serafetinides, 1975). This study represented the first attempt to estimate intracerebral current levels during CET and in fact provided support for the contention that CET could produce direct neurophysiological changes. Where these modifications lead is the next issue to be resolved, and perhaps the answer lies in part in a thorough combined examination of some psychological and related physiological variables during and after CET treatment.

Summary

Many of the physiological studies employed inadequate controls or none at all. Most of them obtained definite modifications in the physiological processes under scrutiny during or after CET treatment. Actual intracerebral CET current levels have been found to be of sufficient magnitude to suggest the possibility of direct neurophysiological changes.

Rationale for Present Study

The present study represented an attempt to investigate the effects of subjectively indistinguishable active and placebo CET on the physiological and psychological

manifestations of anxiety neurosis. The reasons for choosing this target disorder in the evaluation of CET effectiveness have already been discussed. The reasons for undertaking the study were as follows:

(1) There was no previous report of a study in which pulse and respiration rate, and systolic blood pressure (physiological correlates of anxiety) had been assessed as a result of and during actual active and placebo CET in which both conditions were virtually indistinguishable to the subject.

(2) There was no previous report of a study in which both physiological and psychological manifestations of a target disorder had been concurrently and systematically assessed as a result of active and placebo CET treatment, let alone one in which treatment conditions were phenomenologically identical. The rationale for the methodological parameters employed in this study was as follows:

(A) Current parameters

A direct current (DC) as opposed to an alternating (AC) one (in which the current flow regularly changes direction) was employed because it is thought that a constant current is essential for ensuring unidirectional cerebral cell reactions, an important factor in producing desired physiological modifications (Obrosow, 1959). Although AC has been used, DC is by far most frequently employed and adheres to the original electrosleep technique and theory.

The combination of 100 Hz, with a pulse duration of 2 msec and an amplitude of up to 1.5 ma was employed, these

being the most frequently used parameters in the double-blind studies. Wageneder et al. (1969) agreed that 100 Hz was thought to be the most effective frequency.

(B) Treatment duration

It was reported that exposure to CET for more than two hours per session resulted in morning dizziness and a degree of unsteadiness in walking which lasted for a few hours (Iwanovsky et al., 1968). In both the double-blind and physiological studies the exposure time varied from 15 to 60 minutes with an average duration of 30 minutes. The number of sessions varied from one to 24 with an average of ten. For these reasons a total of ten 30-minute treatment sessions was administered, half of which were placebo treatments.

(C) Electrode placement

In the present study, the active CET electrode placement was as follows:

(i) anode (positive electrode) on the brow

(ii) cathode (negative electrode) at the mastoid processes.

There is no firmly established rule about polarity, but a reverse electrode placement was more frequent in the literature (Iwanovsky et al., 1968). However, the direction of current flow appears to be an important factor according to one of the original users of CET, Giljarowskii, who stated:

> ...when the electrodes are arranged transcerebrally or oculo-occipitally the current enters the skull through the orbital fissure and the foramina

> in the orbits as well as through the thinner bones forming the walls and base; it leaves through the foramen magnum and the thin cellular bones of the mastoid process. This is the best method of bringing the current into the skull and making it penetrate into the brain substance. (Boblitt, 1969, p. 12)

Since current conventionally flows from the positive to the negative electrode, the anodes were anterior (but on the brow as opposed to directly on the orbits because of noted side effects) relative to the cathodes. Another reason for placing the anodes over the brow is that the only investigation which has demonstrated intra-cerebral current flow (Dymond et al., 1974) followed this procedure.

(D) Type of placebo

A placebo condition virtually indistinguishable from active treatment was employed using the method described by Marshall et al. (1974). This ensured that during both active and placebo CET, subjects experienced a tingling sensation throughout the entire session. This procedure, as opposed to one in which no tingling was felt in either condition, was thought to be superior for reasons stated by Frankel (1974):

> ...experimental designs, which call for neither the active nor the placebo patients to feel any current-related stimuli, are an important step in the right direction. Such techniques, however, involve the possibility that the amplitude levels of the current may be too low, they are therefore not as desirable as the protocols that call for the stimuli to be felt by both active and placebo groups. (p. 69)

(E) Design

The study employed a single-blind cross-over design. Due to staff and apparatus limitations, a double-blind design

was not feasible. However, the study had one double-blind element in that psychiatrists assessing the subjects were not given any information as to when each type of treatment was administered. The operator of the machine knew which treatment was being administered at all times and strove to remain as non-committal as possible. The single-blind limitation was overcome to some degree by virtue of the objective physiological measures recorded by a polygraph machine and by the use of self-report scales and questionnaires.

The overall structure of this investigation has included most of the recommendations for future studies made by Frankel (1974).

Summary

This study of CET effectiveness for improving anxiety neurosis represents an attempt to assess both physiological and psychological treatment effects in ideal active and placebo treatment conditions employing a single and partial double-blind cross-over design. The methodological parameters chosen were based on shortcomings of previous works and recommendations made in the literature for future works.

CHAPTER II

METHOD

The overall strategy was to measure changes in patients with anxiety neurosis, who were receiving both active and placebo CET. Each treatment was given for five consecutive days in counterbalanced order using a blind cross-over design.

Subjects

The subjects were consecutive patients seen by two staff psychiatrists at St. Clare's Mercy Hospital, St. John's, Newfoundland, who fulfilled the following criteria: (a) they had a primary diagnosis of anxiety neurosis as defined by the DSM-II (1968), (b) the condition had been present for at least three months, (c) they were free from any other psychiatric, medical or surgical disorder, (d) there was no obstacle to them attending daily for treatment over a two-week period, (e) there was no reason to change their medication throughout the period of the study, and they were informed of this and agreed to this condition.

Subjects were advised that a mild electrical treatment called electro-relaxation therapy might help their condition, and they gave consent to this before being referred to the experimenter. No subjects refused this offer and none dropped out during the course of the study.

There were ten subjects, four females and six males. Two of the males were in-patients during the two weeks of treatment, and the remainder were out-patients. Their ages ranged from 17 to 52 years with a mean of 30.8 years. No other treatment was given during the two weeks of treatment or the four-week follow-up period.

Apparatus

(A) CET treatment: A Neurotone 101 (1971), which was checked for proper and accurate functioning by an electrical engineer prior to the experiment was used to produce both active and placebo CET treatments. The machine was a portable, battery-operated solid state unit. The output was limited to the level of 1.5 milliamperes (ma) average current. The pulse setting used was a unidirectional square wave at a frequency of 100 cycles per second or Hertz (Hz), with a wave duration of 2 milliseconds (ms).

Felt pads which had been thoroughly soaked in a commercially prepared saline solution were attached to two pairs of electrodes. One pair, which consisted of two positive electrodes, was placed one inch apart on the brow just above the eyebrows. The second pair, consisting of two negative electrodes, was placed at the mastoid processes. The physical location of electrodes was identical for both active and placebo treatment. However, in placebo treatment, the electrode pair on the forehead consisted of one negative and one positive element. In the placebo treatment, the two

electrodes at the mastoid processes were disconnected from the CET machine, so that current was travelling only between the inch of skin surface separating the two forehead electrodes.

(B) Daily psychophysiological assessment: A four-channel Lafayette Datagraph was used to measure pulse, respiration rate and blood pressure, and to produce a visual record of each measure.

(i) Pulse rate - Pulse rate records were obtained by means of a photo-electric finger plethysmograph which operated by measuring the changes in blood volume by reflectance. It consisted of a long-life incandescent lamp and a hermetically sealed cadmium sulphite photo cell 1.5 centimeters (cm) square. This unit was applied to the subject's forefinger by means of an attached velcro fastener.

(ii) Respiration rate - Respiration rate, recorded concurrently with pulse rate, was obtained by means of pneumograph. This was a 12-inch long pneumatic flexible hose, approximately one inch in diameter. The hose was attached to subject's chest and kept in place by means of a thin chain around the torso. Breathing caused contraction and expansion of the hose and resultant pressure changes were converted to an electrical signal by a pressure transducer.

(iii) Systolic blood pressure - Systolic blood pressure, which is the highest pressure reached by the arteries, was measured by auscultation of the Korotkoff sounds. These sounds are produced by the pulsations of the artery under a

partially constricting blood pressure cuff, and should be recorded to the nearest 5 or 10 mm of mercury (Hg) because they change by at least 2 mm spontaneously from moment to moment (Constant, 1976). The Korotkoff sounds were obtained by means of an electronic sphygmomanometer which provided a tracing of the pressure in the cuff, with the sounds, produced by a microphone over the brachial artery, superimposed on it. The cuff was always placed on the right upper arm. The sphygmomanometer was calibrated such that 2 mm on the graph paper represented 10 mm Hg.

Both pulse and respiration rate tracings were done on three separate occasions of each treatment day: 1) pre-treatment, 2) mid-treatment, 3) post-treatment. Blood pressure tracings were made on two separate occasions of each treatment day after pulse and respiration rates had been obtained: 1) pre-treatment, 2) post-treatment. A mid-treatment blood pressure tracing was omitted because inflation of the arm cuff would have disturbed the subject.

A one-minute segment of the pulse and respiration rate tracings located just before the systolic blood pressure tracing was used to determine the pulse and respiration rate per minute. Each spike traced out by the plethysmograph and pneumograph was counted in this one-minute segment and the totals represented the pulse rate and respiration per minute.

Systolic blood pressure level was determined by locating on each sphygmomanometer tracing the first large

spike denoting maximum systolic pressure. This location was verified by a corresponding spike found on the plethysmograph tracing. The number of mm from baseline to the spike location were counted. Half mm were included. The average systolic pressure from two tracings was considered to be the true systolic level. Figure 1 represents a typical polygraph segment obtained during a treatment session.

(C) Daily psychological assessment: Two psychological measures of anxiety were used: pre- and post-treatment on each of the total of ten treatment days.

(i) IPAT Eight Parallel-Form Anxiety Battery (EPFAB) - In order to have psychological measures parallel the physiological measures, the EPFAB (1969) was employed. Those forms with the best construct validity and interform reliability were used (Forms F, D, and B). The order in which forms were given was randomized such that all were administered an approximately equal number of times (Chassan, 1967). The order of administration was counterbalanced.

The forms were administered according to manual instructions. Subjects were required to read instructions before proceeding to answer items.

Raw scores were converted into sten scores using the norms provided in the manual.

(ii) Muscle Tension-Anxiety Self-Rating Scale (TAS) - A form (Appendix A) using two lines each 10 cm long and representing levels from "none" to "severest" was devised as a daily measure of muscle tension and anxiety. Instruc-

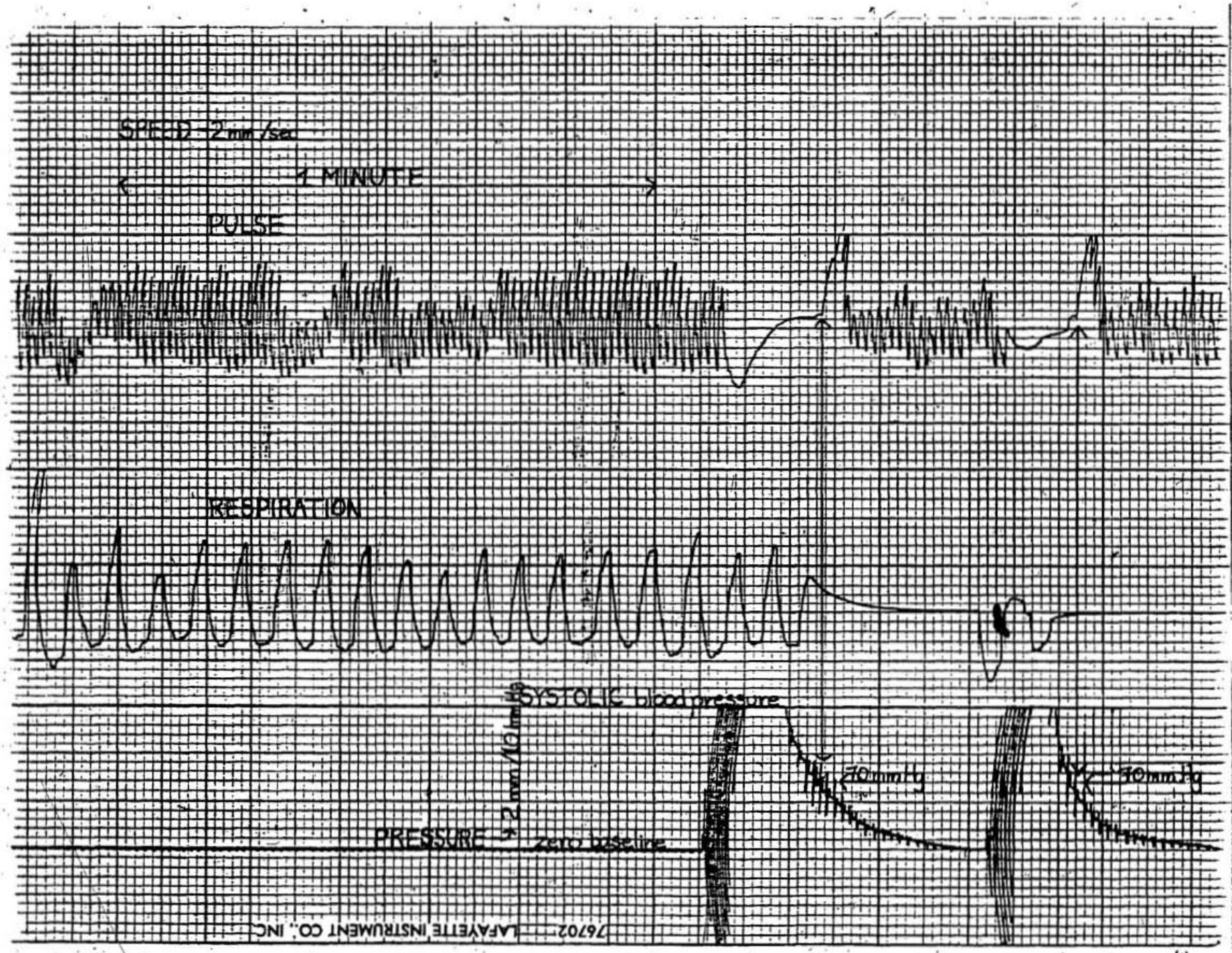


FIGURE 1. Polygraph tracing showing a typical one-minute segment of pulse and respiration rates followed by measurement of systolic blood pressure.

tions to subjects were to put a vertical pencil mark through the 10 cm line at a point which best described their present muscle tension and anxiety levels. Subjects were not allowed to see pre-treatment ratings while completing post-treatment ratings. The TAS yielded two separate scores of muscle tension (MT) and anxiety (A) which were used for data analysis.

(D) Weekly psychological assessment: A total of three psychological instruments were used to assess treatment effects on a weekly basis on Days 1, 8, 15, 22 and 43 of the experimental period for a total of five administrations each.

(i) Eysenck Personality Inventory (EPI) - Both forms A and B were used to obtain a weekly assessment of trait anxiety. The initial EPI administration also served the purpose of providing an objective verification of the psychiatrist diagnosis of anxiety neurosis.

Both forms A and B were alternated on each day of EPI administration in a counterbalanced fashion. Subjects 1, 3, 5, 7, and 9 were given order BABAB, and subjects 2, 4, 6, 8 and 10 were given order ABABA.

The EPI was administered according to instructions in the manual. Subjects were directed to read instructions printed on the form before answering items. Instructions were not amplified or altered in any way.

Raw scores on Form B were first converted into percentile scores. These percentile scores were then used

to find the equivalent raw score on Form A, so that scores from both forms were made parallel. EPI norms for American college students were used for this purpose. The resultant converted raw scores were used in data analysis.

(ii) State-Trait Anxiety Inventory (STAI) - The STAI is comprised of two self-report scales for measuring state and trait anxiety, with one scale printed on each side of the form. Both forms were given on each day of administration (Days 1, 8, 15, 22 and 43).

The form was administered according to guidelines set out in the manual. Subjects were instructed to read instructions printed on the form prior to answering items.

Raw scores were used in data analysis.

(iii) Complaint Checklist (CC) - A self-rating behaviour and symptom checklist (Appendix A) was individually drawn up for subjects on Day 1 pre-treatment. Subjects were asked to list in order of significance the behaviours or symptoms associated with their anxiety and which they hoped would be alleviated by the treatment. Subjects were then asked to rate each behaviour on a 1-point scale in terms of severity with zero representing "none", 5, "moderate", and 10, "severest". The checklist was filled out by the experimenter and subjects were not allowed to see previous ratings. Only the raw score on complaint "A" was used in data analysis.

(iv) Psychiatric Clinical Assessments (PCA) - A seven-point rating scale of anxiety, depression, and insomnia (Appendix A) was used by the two staff psychiatrists in the

weekly pre-treatment clinical assessment of subjects. Both the psychiatrist's blind clinical rating and the subject's subjective rating were recorded, so that a total of six ratings per assessment day resulted. The average of these six ratings was used as the score for data analysis.

Procedure

A cross-over design in which each subject was given both active and placebo treatments was employed. Half of the subjects were given active treatment in the first week, followed by placebo treatment in the second week while the reverse order was used for the remainder. The order of treatment was randomly determined for subject 1, and was alternated for each successive subject. Thus, subjects 1, 3, 5, 7, and 9 first received placebo treatment followed by active treatment, and subjects 2, 4, 6, 8, and 10 received the reverse order of treatment.

Treatment sessions were conducted in the afternoons Monday through Friday for two consecutive weeks for a total of ten treatment sessions, five active and five placebo. Length of actual treatment time per session was 30 minutes preceded by 15 minute rest period. There were three follow-up days on the first, second, and fifth Monday after the last Friday of treatment (Days 15, 22, 43).

Patients who met the psychiatrist's criteria for the study were referred to the experimenter and were asked again if they wanted to undergo a course of "electro-

relaxation" which might help to reduce their anxiety levels. They were not told that this treatment constituted part of an experimental study, nor that there were two types of treatment involved.

Patients were asked by the experimenter to consider the following conditions for undergoing electrorelaxation before making a final decision about participation:

(1) Patients would be required to come for treatment without fail at the same time every day Monday through Friday for two consecutive weeks, and the daily time commitment would be approximately one and one-half hours. Patients would also be expected to come on three follow-up days, which were the first, second, and fifth Monday after the last Friday of treatment.

(2) Patients would have to be able to read and understand the contents of the various psychological measures.

Patients who agreed to undergo the course of electrorelaxation treatment were given the following instructions by the experimenter: "(1) Electrorelaxation is a mild electrical therapy which is safe and painless and causes a slight tingling sensation at the electrode sites. (2) Before, during, and after treatment your pulse, respiration rate and blood pressure will be monitored by means of a polygraph machine. This will enable us to get an objective assessment of your physiological response to treatment. (3) The purpose of the psychological measures is to see what you think about yourself as a result of treatment.

(4) During treatment you must lie quietly on your back, try to relax and not move around too much nor lie on your side so that the machine attachments can function properly. This is important for maximum treatment benefit. Do not touch any of the apparatus." Subjects were instructed to present themselves at the experimenter's office on the psychiatry unit of the hospital at a mutually agreed upon time in the afternoon of the first Monday after consent for treatment had been given.

On the first day of treatment (Day 1) subjects were given the following pre-treatment psychiatric and psychological assessment:

(1) PCA (psychiatrist's office)

(2) CC (2 to 5 were given in the experimenter's office)

(3) EPI

(4) STAI

(5) TAS

(6) EPFAB

The PCA, CC, EPI, and STAI were given pre-treatment on Days 1, 8, 15, 22 and 43 of the experimental period. The TAS and EPFAB were administered pre- and post-treatment on Days 1-5, 8-12 of the experimental period.

After completion of pre-treatment psychiatric and psychological assessment, subjects were taken to the treatment room on the psychiatric unit. Subjects were asked to remove their shoes and any other clothing which might make them uncomfortable when lying down, as well as their eye glasses,

and to lie down on a hospital stretcher and make themselves as comfortable as possible. The polygraph apparatus was attached to subjects as described above, followed by CET electrode application as described above. The subject's legs were covered with a light blanket. Subjects were reminded of instructions to lie quietly on their backs, not to move around, not to roll on to their sides, nor to touch any of the attached apparatus. The room was darkened and subjects were left alone for the 15 minute pre-treatment resting period, after which the experimenter returned to turn on the polygraph machine and obtain a pre-treatment measure of pulse, respiration rate and blood pressure. Paper speed was set at .2 cm/sec. After an approximately three-minute recording of pulse and respiration rate had been obtained, two satisfactory blood pressure tracings were made. The polygraph machine was then turned off.

Following the pre-treatment recording of pulse, respiration rate and blood pressure, the CET machine was turned on. The procedure was the same for both active and placebo treatment conditions: The CET machine knob was turned on and the intensity slowly increased up to the point at which subjects were able to identify a tingling sensation on at least one of the four electrode sites. The intensity level at which this occurred varied from between .1 and .5 ma.

At this point in the procedure, subjects were again left alone for 15 minutes, after which time a mid-treatment polygraph record of pulse and respiration rate was obtained

by the experimenter. At the end of 30 minutes of treatment, the CET machine was turned off and the post-treatment polygraph recording of pulse, respiration rate and blood pressure was obtained in the manner described above.

Following the post-treatment physiological recordings, subjects returned to the experimenter's office and were given the post-treatment psychological assessment (TAS and EPFAB). This marked the end of a treatment session.

Table 3 provides a step-by-step guide, in order of sequence, for each treatment and follow-up day.

Hypotheses

The aim of this study was to look for an overall effect of the CET treatment procedure on anxiety neurosis, as well as for evidence of a differential effect between active and placebo treatment. Based on the review of the literature and the theoretical notions about CET, the following hypotheses were formulated:

Hypothesis I: The two-week treatment procedure will produce an overall statistically significant effect on weekly anxiety measures between those taken at baseline (Day 1) and those taken at the end of the two-week procedure (Day 15) and on the follow-up days (Day 22, 43).

Hypothesis II: The two types of treatment, active and placebo, will produce equal effects on both weekly and daily measures of anxiety.

TABLE 3

A step-by-step guide to the experimental procedure for each treatment day

Day 1,8.

Location	Step		
Psychiatrist's Office:	(1)	Psychiatrist's assessment	(a) PCA
Experimenter's Office:	(2)	Psychological assessment	(a) CC (b) EPI (c) STAI (d) TAS (e) EPFAB
Treatment Room:	(3)	CET and polygraph attached	
	(4)	15 minute rest period	
	(5)	Psychophysiological assessment	(a) pulse (b) respiration (c) blood pressure
	(6)	CET (active or placebo) begins	
	(7)	15 minute treatment	
	(8)	Psychophysiological assessment	(a) pulse (b) respiration
	(9)	15 minute treatment	
	(10)	CET (active or placebo) ends	
	(11)	Psychophysiological assessment	(a) pulse (b) respiration (c) blood pressure
	(12)	CET and polygraph detached	
Experimenter's Office:	(13)	Psychological assessment	(a) TAS (b) EPFAB

Day 2-5, 9-12.

Steps 2d, 2e, 3-13

Day 15, 22 (one-week follow-up), 43 (one-month follow-up)

Steps 1, 2a-c

Statistical Analysis

Both weekly and daily raw data are given in Appendix C. The raw data were transformed from raw scores into T-scores, with a mean of 50 and a standard deviation of 10, before entering them into a multi-factorial analysis of variance which was carried out by computer methods. The program used, capable of handling multi-factorial designs with repeated measures, was originally written in FORTRAN by W. Dunlap, Tulane University and converted to BASIC by J. Strawbridge, Psychology Department, Memorial University of Newfoundland.

Where appropriate, the Tukey test for all possible comparisons between pairs of means was used for post hoc analyses (Keppel, 1973, p. 138).

The Spearman rank-order correlation coefficient was employed in an additional post hoc analysis, in which personality characteristics and degree of response to the experimental procedure were analyzed (Welkowitz, Ewen & Cohen, 1971).

CHAPTER III

RESULTS

Reliability of Diagnosis

As a check on the reliability of the psychiatric diagnosis of anxiety neurosis in subjects, the EPI scores obtained on Day 1 (baseline) were examined. Analysis of N scores revealed the range for all subjects to be between the 87th and 99th percentiles, with the mean at the 96th percentile. The range for all subjects on E was between the 2nd and 85th percentile. Six of the 10 subjects ranged between the 2nd and 26th percentile, whereas three subjects ranged between the 34th and 62nd percentile (normal). One subject obtained an above-average score on E at the 85th percentile. The mean of E scores was at the 26th percentile (below average, indicating introversion). The mean percentile ranks on Spielberger's (1970) STAI were also high but not as extreme as the EPI N scores with group state anxiety at the 70th percentile and trait anxiety at the 75th percentile.

Status of the Hypotheses

As indicated by the statistically significant results reported in this study, the status of the two hypotheses formulated earlier is as follows:

Hypothesis I: The two-week treatment procedure will produce

an overall statistically significant effect on weekly anxiety measures between those taken at baseline (Day 1) and those taken at post-treatment (Day 8, 15) and on follow-up days (Day 22, 43). This hypothesis was confirmed. Hypothesis II: The two types of treatment; active and placebo, will produce equal effects on both daily and weekly measures of anxiety. This hypothesis was also confirmed.

Analysis of Weekly Data

Two main analyses of variance were performed on the weekly data. The first of these was an overall analysis employing the following variables in its design:

(1) Order
- (a) active - placebo (AP)
- (b) placebo - active (PA)

(2) Type of dependent variable
- (a) EPI - neuroticism-stability (N)
- (b) STAI - trait anxiety (TR)
- (c) STAI - state anxiety (ST)
- (d) Complaint Checklist (CC)
- (e) Psychiatric Clinical Assessment (PCA)

(3) Day
- Day 1 (baseline)
- Day 8
- Day 15
- Day 22 (one-week follow-up)
- Day 43 (one-month follow-up)

This 2 (order) x 5 (type of dependent variable) x 5 (day) analysis of variance yielded the statistically significant F-ratios presented in Table 4.

A summary of this analysis of variance is presented in Table 4.

TABLE 4

Summary of overall analysis of variance on weekly data

Source	SS	df	MS	F-ratio	p
Order (O)	19.088	1	19.088	.012	.91133
Error	12596.900	8	1574.610		
Type of dependent variable (DV)	.000	4	.000	.000	.99991
O x DV	1038.390	4	259.596	2.763	.04374*
Error	3006.600	32	93.956		
Day (D)	1383.230	4	345.807	4.667	.00468*
O x D	63.944	4	15.986	.216	.92621
Error	2371.320	32	74.104		
DV x D	967.703	16	60.482	2.570	.00203*
O x DV x D	540.245	16	33.765	1.435	.13553
Error	3012.550	128	23.536		
Total	25000.000	249			

Day, a within-subjects variable, produced a significant main effect ($F(4,32) = 4.667$, $p = .00468$). Figure 2 shows the decrease on pooled measures of anxiety as a function of treatment and follow-up days. Post hoc comparisons of pairs of means, presented in Table 5, revealed that there was a significant difference between the mean on Day 1 and, in order of magnitude, those obtained on Days 43, 22, 15 and 8.

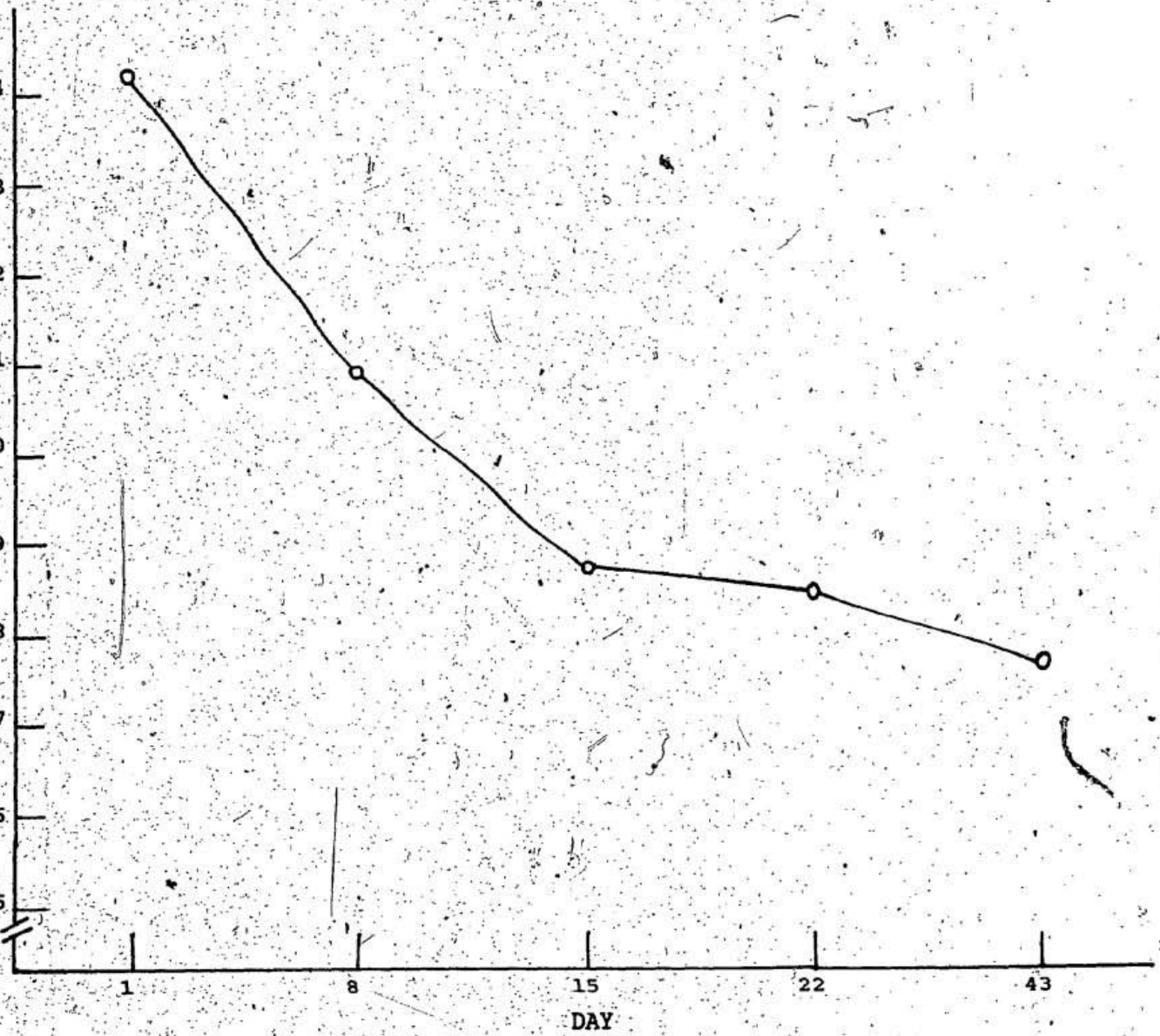


FIGURE 2. Weekly measures of anxiety combined as a function of day.

In addition, a significant difference was found between the mean on Day 8, and those obtained on Days 43, 22 and 15. Thus, the means obtained on Days 1 and 8 differed significantly from all the other days, with the greatest difference being noted between baseline (Day 1) and the one-month follow-up day.

TABLE 5

All possible comparisons between pairs of means on main effect of day (weekly data)

		DAY				
		43	22	15	8	1
DAY		2385.83	2424.30	2433.35	2547.20	2709.15
43	2385.83	-	38.47	47.52	161.37*	323.32*
22	2424.30	-	-	9.05	122.90*	284.85*
15	2433.35	-	-	-	113.85*	275.80*
8	2547.20	-	-	-	-	161.95*
1	2709.15	-	-	-	-	-

*Tukey's test, critical range, difference between means > 116.61, significant at .05 level.

A statistically significant two-way interaction was found between order and type of dependent variable ($F(4,32) = 2.736$, $p = .04374$). The means and standard deviations for this analysis are presented in raw data form in Table 6. Post hoc analysis of this interaction did not reveal a

significant difference between any of the pairs of means (Appendix B, Table 11).

TABLE 6

Means and standard deviations (raw data) for interaction between order and type of dependent variable (weekly data)

Type of dependent variable	ORDER AP $\bar{X}$	AP SD	PA $\bar{X}$	PA SD
N	18.40	2.96	16.24	5.15
ST	60.44	9.40	51.76	14.83
TR	48.12	18.63	47.76	20.21
CC	4.64	3.93	5.56	3.56
PCA	1.23	1.12	1.85	1.25

The main effect of day entered into a significant interaction with type of dependent variable ($F(16,128) = 2.57$, $p = .00203$), illustrated in Figures 3 and 4. As a follow-up, a separate analysis of variance was performed on each type of dependent variable. These analyses revealed that there was a significant decrease over days on scores obtained on ST ($F(4,32) = 2.755$, $p = .00417$), CC ($F(4,32) = 5.888$, $p = .00143$) and PCA ($F(4,32) = 10.27$, $p = .00007$), but not on N and TR. The means and standard deviations (in raw scores), as well as summary tables of the follow-up analysis of variance are presented in Appendix B, Tables 12 and 13.

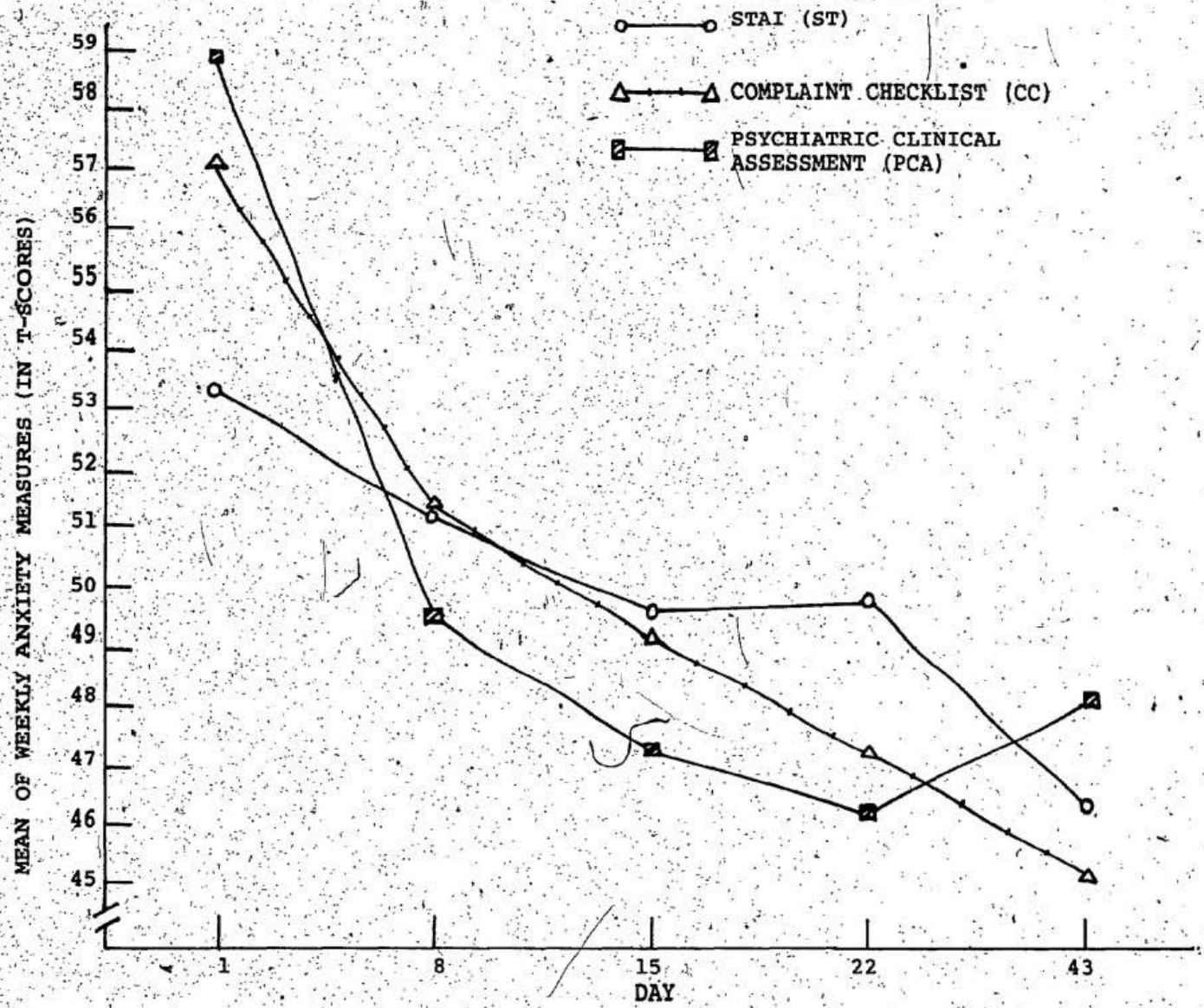


FIGURE 3. Weekly anxiety measures as a function of day (ST, CC and PCA).

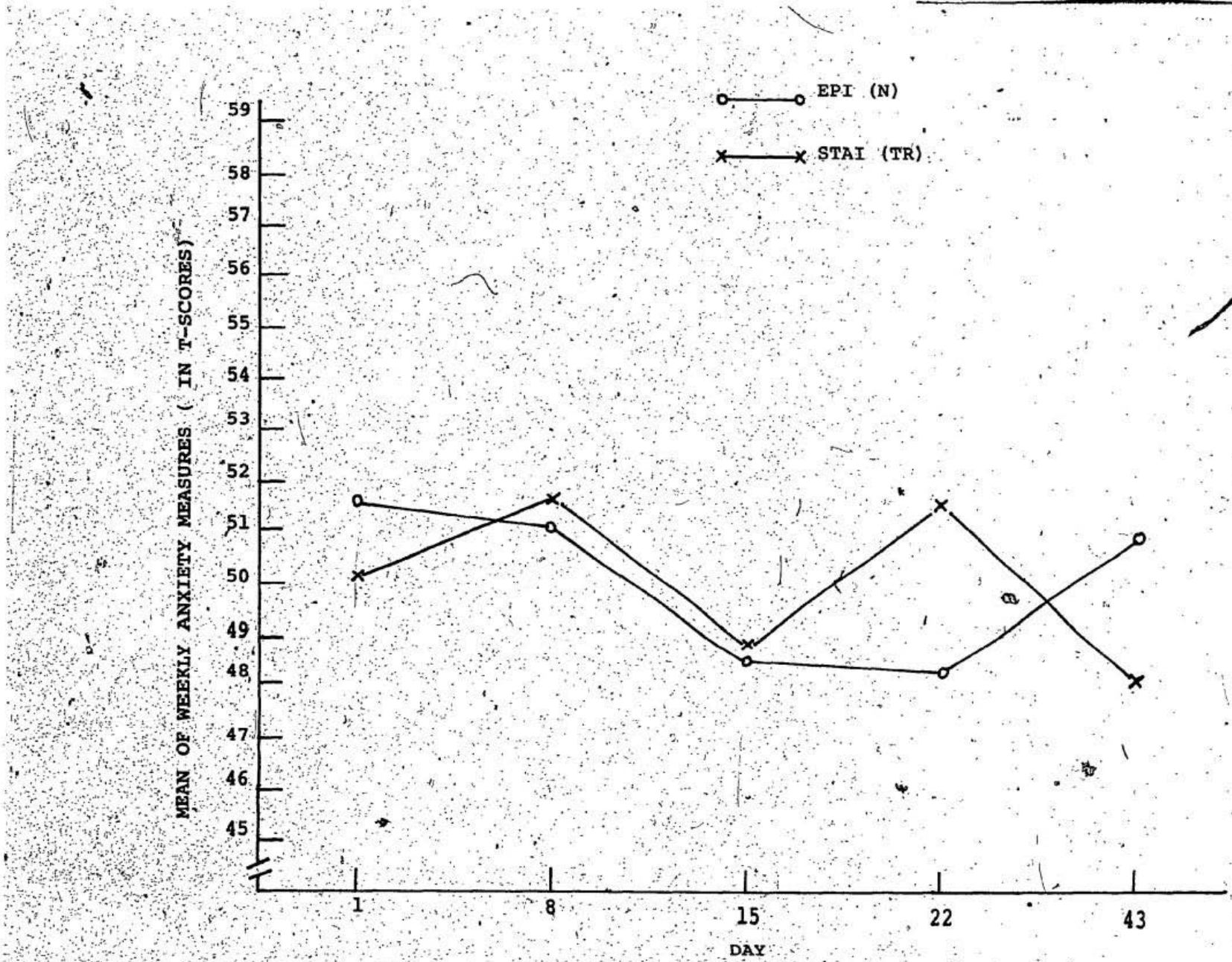


FIGURE 4. Weekly anxiety measures as a function of day (N and TR).

Post hoc comparisons of means on the results of ST, CC and PCA, are presented in Table 7 and show that for all three variables the difference between the means of Day 1 (baseline) and Day 43 (one-month follow-up) are statistically significant. The difference between the means of Day 43 and those of Days 8 and 15 are statistically significant for CC and PCA, but not for ST. In addition, the difference between the mean of Day 43 and Day 22 is statistically significant for PCA, but not for ST and CC. Thus, the greatest decreases between baseline (Day 1) and all the other days were noted on PCA. ST showed the least amount of change of the three types of measures under consideration, and scores on CC decreased almost as much as those on PCA.

A second analysis of variance was performed on the weekly data in order to look for a treatment effect. This involved examining scores on all five types of dependent variables (N, TR, ST, CC and PCA) at baseline (Day 1) and after one week of treatment (Day 8), thus giving a design in which there were five subjects per cell. This 2 (type of treatment (active or placebo)) x 2 (Day 1,8) x 5 (type of dependent variable) analysis of variance demonstrated that the main source of the variance was found in the two-way interaction between type of dependent variable and day ($F(4,32) = 3.893$, $p = .01099$). No main effect of treatment was revealed, nor was there a significant main effect of day, previously found in the overall weekly analysis, repeated. The summary of this analysis is presented in Table 8.

TABLE 7

All possible comparisons of means on interaction between type of dependent variable and day (weekly data, follow-up analysis of variance)

ST		DAY				
		1	8	15	22	43
DAY		463.50	495.00	496.60	511.30	533.30
1	463.50	-	31.50	33.10	47.80	69.80*
8	495.00	-	-	1.60	16.30	38.30
15	496.60	-	-	-	14.70	36.70
22	511.30	-	-	-	-	22.00
43	533.30	-	-	-	-	-

*Tukey's test, critical range = 63.06, p = .05

CC		DAY				
		1	8	12	22	43
DAY		451.35	472.97	491.89	513.51	570.27
1	451.35	-	21.62	40.54	62.16	118.92*
8	472.97	-	-	19.00	40.54	97.30*
15	491.89	-	-	-	21.62	78.38*
22	513.51	-	-	-	-	56.76
43	570.27	-	-	-	-	-

*Tukey's test, critical range = 76.89, p = .05

PCA		DAY				
		1	8	15	22	43
DAY		461.80	473.01	481.14	495.00	589.05
1	461.80	-	11.21	19.34	33.20	127.25*
8	473.01	-	-	8.13	21.99	116.04*
15	481.14	-	-	-	13.86	107.91*
22	495.00	-	-	-	-	94.05*
43	589.05	-	-	-	-	-

*Tukey's test, critical range = 65.55, p = .05

TABLE 8

Summary of analysis of variance on weekly data looking for treatment effect

Source	SS	df	MS	F-ratio	p
Treatment (T)	41.296	1	41.296	.111	.74463
Error	2964.730	8	370.591		
Type of dependent variable (DV)	197.833	4	49.458	.764	.55825
T x DV	150.822	4	37.706	.583	.68023
Error	2070.280	32	64.696		
Day (D)	262.287	1	262.287	2.614	.14235
T x D	1.607	1	1.607	.016	.89784
Error	802.685	8	100.336		
DV x D	378.669	4	94.667	3.893	.01099*
T x DV x D	136.133	4	34.033	1.400	.25567
Error	778.137	32	24.317		
Total	7784.480	99			

Analysis of Daily Data

Two main analyses of variance were performed on the daily data. In the overall analysis of variance of the daily data, the following variables were employed in the design:

(1) Order - (a) active - placebo (AP)
(b) placebo - active (PA)

(2) Type of treatment (a) active
(b) placebo

(3) Type of dependent variable
  (a) Pulse
  (b) Respiration
  (c) Systolic blood pressure
  (d) Muscle Tension (MT)
  (e) Anxiety (A)
  (f) Eight Parallel-Form Anxiety Battery (EPFAB)

(4) Day — Treatment days 1-5

(5) Occasion of measure
  (a) pre-treatment
  (b) post-treatment

This 2 (order) x 2 (type of treatment) x 6 (type of dependent variable) x 5 (day) x 2 (occasion of measure) analysis of variance yielded the statistically significant F-ratios found in the summary in Table 9.

Occasion of measure produced the only statistically significant main effect ($F(1,18) = 15.568$, $p = .00453$), showing that the pooled post-treatment measures ($\bar{X} = 49.536$) were significantly decreased from the pooled pre-treatment measures ($\bar{X} = 50.464$).

A statistically significant two-way interaction was found between type of dependent variable and day ($F(20,160) = 1.72$, $p = .03467$). These results are presented in Figures 5 and 6. A follow-up analysis of variance performed separately on each type of dependent variable failed to detect any significant main effect of 'day' for any single dependent variable. These results are given in Appendix B, Table 14. However, as suggested by Figures 5 and 6 the physiological measures, as opposed to the psychological ones, tended to increase over days.

TABLE 9

Summary of overall analysis of variance on daily data

Source	SS	df	MS	F-ratio	p
Order (O)	513.839	1	513.839	.152	.70717
Error	27122.100	8	3390.270		
Treatment (T)	23.275	1	23.275	.239	.64129
O x T	95.914	1	95.914	.986	.64839
Error	778.312	8	97.289		
Type of dependent variable (DV)	.000	5	.000	.000	.99998
O x DV	4809.680	5	961.935	.823	.54193
Error	46735.000	40	1168.380		
T x DV	421.556	5	84.311	.789	.56571
O x T x DV	683.434	5	136.687	1.278	.29161
Error	4276.510	40	106.913		
Day (D)	30.462	4	7.616	.151	.95861
O x D	332.067	4	83.017	1.647	.18576
T x D	74.713	4	18.678	.466	.76280
O x T x D	75.795	4	18.949	.472	.75804
Error	1283.800	32	40.119		
DV x D	1348.510	20	67.425	1.720	.03467*
O x DV x D	704.565	20	35.228	.899	.58958
Error	6272.650	160	39.204		
T x DV x D	604.652	20	30.233	.874	.62041
O x T x DV x D	640.799	20	32.040	.926	.55482
Error	5534.130	160	34.588		
Occasion of measure (OM)	258.404	1	258.404	15.568	.00453*
O x OM	50.222	1	50.222	3.026	.11784
Error	132.787	8	16.598		
T x OM	.041	1	.041	.001	.97223
O x T x OM	108.482	1	108.482	3.146	.11175
Error	275.892	8	34.487		
DV x OM	120.095	5	24.019	.479	.79133
O x DV x OM	113.582	5	22.717	.453	.80986
Error	2005.610	40	50.140		
T x DV x OM	165.605	5	33.121	1.440	.23039
O x T x DV x OM	266.651	5	53.330	2.319	.06052
Error	920.043	40	23.001		
D x OM	147.215	4	36.804	.938	.54367
O x D x OM	81.832	4	20.458	.521	.72355
Error	1256.130	32	39.254		
T x D x OM	56.480	4	14.120	.851	.50545
O x T x D x OM	74.602	4	18.651	1.124	.36283
Error	531.035	32	16.595		
O x D x OM	692.057	20	34.603	1.522	.08018
O x DV x D x OM	366.994	20	18.350	.807	.70323
Error	3638.000	160	22.738		
T x DV x D x OM	491.449	20	24.572	1.109	.34505
O x T x DV x D x OM	726.478	20	36.324	1.639	.04921*
Error	3545.850	160	22.162		
Total	120000.000	1199			

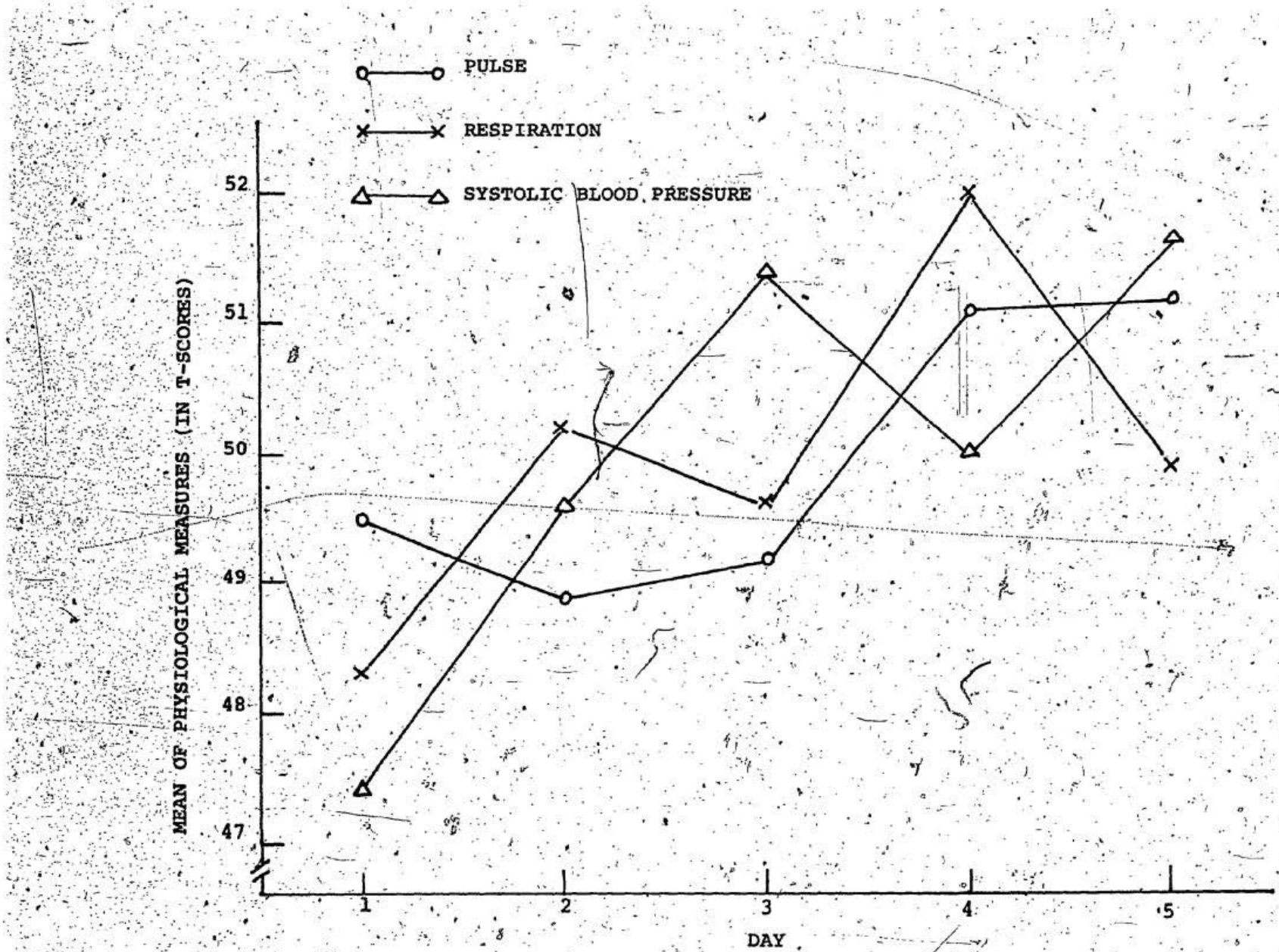


FIGURE 5. Physiological measures as a function of day (Pulse, Respiration and Systolic Blood Pressure).

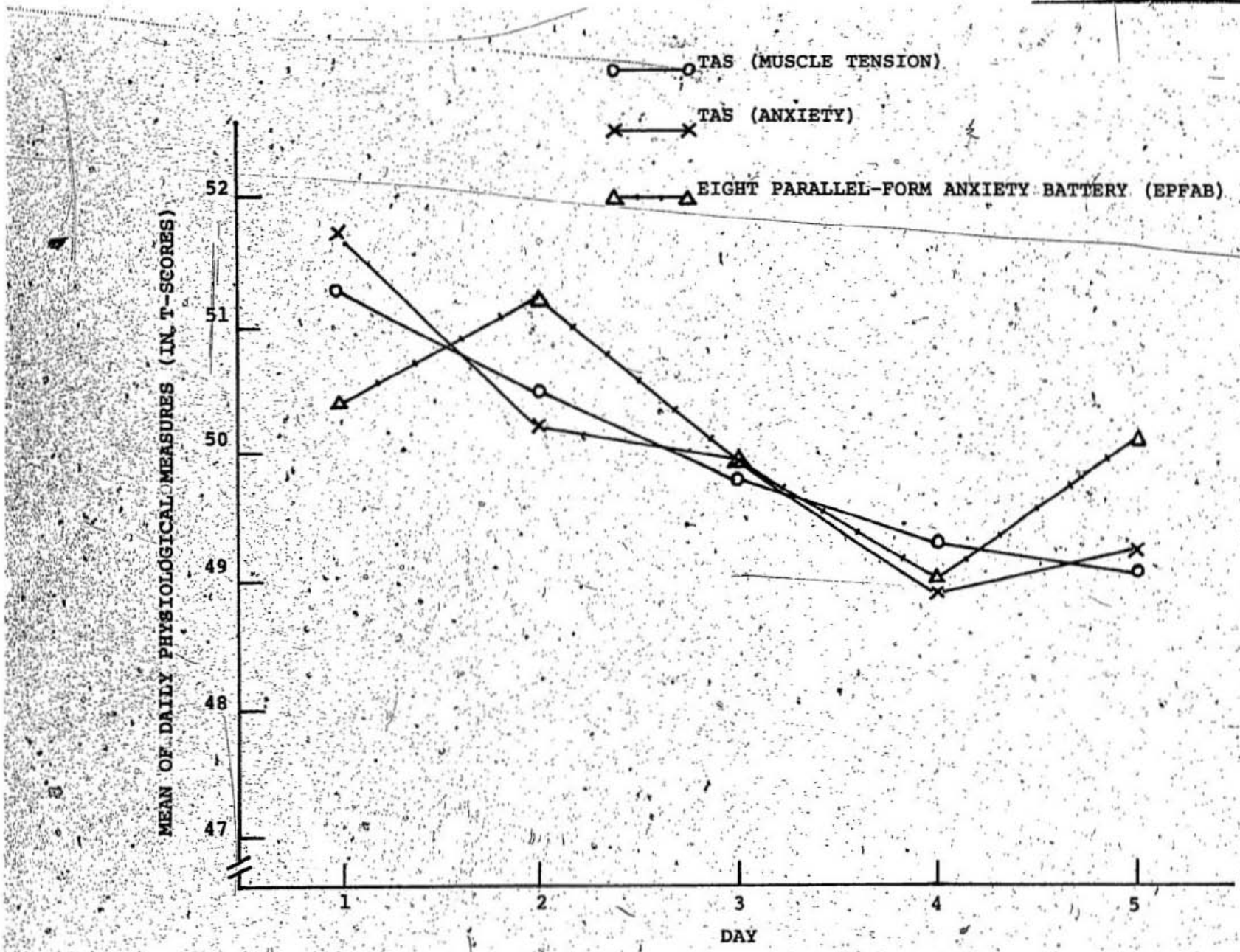


FIGURE 6. Daily physiological measures as a function of day (Muscle Tension, Anxiety and EPFAB).

A significant five-way interaction among order, type of treatment, type of dependent variable, day and occasion of measure was found ($F(20,160) = 1.639$, $p = .04921$). A follow-up analysis of variance performed separately for each order revealed that the four-way interactions among type of treatment, type of dependent variable, day, and occasion of measure was significant for order placebo-active (PA) ($F(20, 80) = 2.37763$, $p < .025$), but not for order active-placebo ($F < 1$).

A second main analysis of variance was performed on the daily data in which only the two dependent variables of pulse and respiration were examined, and in which the mid-treatment measure was included in the design. This analysis was undertaken to determine if there was any significant mid-treatment effect, as these were the only measures made when the actual treatment was in process. Thus, the occasion of measure variable in the analysis of variance design included a pre-treatment, mid-treatment and post-treatment score. The design was a 2 (order) x 2 (type of treatment) x 2 (type of dependent variable) x 5 (day) x 3 (occasion of measure). This analysis did not produce any statistically significant F-ratios (see Appendix B, Table 15).

Response to Treatment Procedure and EPI Personality Dimensions

Based on the findings confirming the hypotheses of an overall treatment effect and of equal effects between

active and placebo treatment, a post hoc decision was made to analyze the relationship between response to the overall treatment procedure and EPI personality dimensions.

Since a significant decrease had been demonstrated on scores from the weekly anxiety measures ST (state anxiety), CC (Complaint Checklist) and PCA (Psychiatric Clinical Assessment), scores from these three measures were used to obtain a response-to-treatment score (R), where

$$R = \frac{\text{Score Day 1} - \frac{\text{Scores Days 15 + 22 + 43}}{3}}{\text{Score Day 1}} \times 100.$$

As the parametric properties of this score appeared dubious, it was used to rank the subjects in order of responsiveness to treatment on ST, CC and PCA.

The Spearman rank-order correlations were calculated between these three measures. Results revealed they were significantly correlated with each other at the .01 level of significance (r_s (ST and CC) = .90, r_s (ST and PCA) = .75, r_s (CC and PCA) = .85 (one-tailed tests)).

The R scores on all these measures were then averaged to obtain an overall response rank (OR) for each subject. Correlation coefficients were then obtained between OR and the EPI extraversion ranking on Day 1. The resultant Spearman correlation coefficient of .71 was significant at the .05 level, indicating a high positive relationship between response to overall treatment and degree of extraversion. This suggests that those individuals who were more extra-

verted responded best to the overall treatment procedure. All subjects scored uniformly high on the EPI neuroticism (N) and the variance was too small to warrant calculating the correlation between N and OR.

The analysis of responders and EPI personality dimensions was taken one step further with a comparison of the overall response rate (OR) to fluctuations on a daily physiological measure under the two treatment conditions. Pulse rate was chosen for this purpose as it generated a larger variance than the respiration rate or systolic blood pressure. Day 5 of active and placebo treatment was chosen on the supposition that any changes would be maximal on the final day of treatment. An active and placebo pulse rate ranking (PRA and PRP) was obtained by means of the following formula:

$$\frac{\text{Day 5 pre-score}}{\text{Day 5 post-score}} \times 100.$$

These scores were then ranked and a correlation coefficient (r_s) between OR and PRA, and OR and PRP, was obtained. Neither of these two correlation coefficients, listed in Table 10, reached statistical significance, although opposing trends were noted. In active treatment the pulse rate of responders tended to increase, whereas there was a tendency for it to decrease during placebo treatment.

A final correlation was performed between extraversion and pulse rates at Day 5 of both active and placebo treatment. The results showed that extraverts revealed a significant

decrease on pulse rate at Day 5 of placebo treatment (r_s = -.694), and an increase, although not significant, on pulse rate at Day 5 of active treatment.

A summary of the results of these correlations is presented in Table 10.

A final correlation was calculated between extraversion (E) and PRA, as well as between E and PRP. The results yielded a significant negative correlation between E and PRP (r_s = -.694), and a positive, but not significant correlation between E and PRA. In other words, the more extraverted an individual may be, the more likely that pulse rate will decrease during placebo treatment, but tend to increase, although not significantly, during active treatment.

A summary of these correlations may be found in Table 10.

Summary

Weekly Data

Pooled measures of anxiety decreased over weeks, with the greatest decrease from baseline (Day 1) and after one week of treatment (Day 8) being found at the one-month follow-up (Day 43). Scores on measures of state anxiety (ST, CC and PCA) decreased significantly over weeks (with the greatest decrease from baseline being found at the one-month follow-up), but measures of trait anxiety (N and TR) did not decrease significantly from baseline. In contrast

to these findings of overall improvement on weekly measures of anxiety, no differential effect between active and placebo treatment was noted. There was a significant positive correlation between response to the overall treatment procedure and extraversion.

TABLE 10

Summary of Spearman rank-order correlation coefficients (r_s) between extraversion (EPI), overall response ranks (OR) and active and placebo pulse rates (PRA and PRP)

r_s (ST - CC)	= .90	$p < .01$
r_s (ST - PCA)	= .75	$p < .01$
r_s (CC - PCA)	= .85	$p < .01$
r_s (OR - E)	= .71	$p < .05$
r_s (TR - E)	= -.48	n.s.
r_s (OR - PRA)	= .38	n.s.
r_s (OR - PRP)	= -.55	n.s.
r_s (E - PRA)	= .42	n.s.
r_s (E - PRP)	= -.69	$p < .05$

Key:

ST (STAI, state anxiety)
CC (Complaint Checklist)
PCA (Psychiatric Clinical Assessment)
OR (Overall response rank)
E (EPI, extraversion-introversion)
TR (STAI, trait anxiety)
PRA (pulse rank, active treatment)
PRP (pulse rank, placebo treatment)

Daily Data

There was a significant decrease from pooled pre-treatment to post-treatment measures of anxiety. There was also a significant fluctuation over treatment days on daily measures of anxiety, but these were not significant for any single daily anxiety measure alone. Visual inspection of the physiological measures suggest that they increased rather than decreased over days. There was a significant correlation between extraversion and decrease in pulse rate at Day 5 of placebo treatment.

CHAPTER IV

DISCUSSION

Reliability of Diagnosis

In this study of CET efficacy, the diagnosis of anxiety neurosis was the main criterion for subject selection. An examination of baseline EPI scores revealed that, as a group, subjects obtained the type of profile typically found in individuals suffering from anxiety neurosis, with above-average N scores and below-average E scores. Above-average baseline anxiety levels as measured by STAI were also noted. Thus, the subjects' diagnosis of anxiety neurosis or chronic anxiety was supported by the above-average baseline anxiety levels tapped by the EPI and STAI.

The Overall Treatment Effect

Evidence for a significant overall treatment effect was found both in the weekly and daily data. In the weekly data, the significant main effect of day, as well as the significant two-way interaction between type of dependent variable and day provided confirmation of Hypothesis I. The decrease over days on all weekly measures of anxiety clearly illustrated the overall treatment effect. The greatest difference was found between Day 1 (baseline) and Day 43 (one-month follow-up). This would appear to imply that subjects continued to improve even after treatment had

been discontinued, making a strong case for the argument that non-specific factors play a significant part in the therapeutic effectiveness of CET. These factors might include such variables as suggestion, expectation of improvement and therapeutic rapport with one experimenter. Claridge (1970) has discussed such non-specific elements of drug treatment:

> "...an important influence on the placebo effect is the mystique and ceremony with which it is dispensed....How effective these instructions will be may depend on other subtle factors in the situation, such as the relative professional status of the drug-giver and drug-taker. Also important may be surroundings in which the experiment is carried out, including any impressive procedures to which the person is subjected" (p. 33).

Since the subjects were selected on the basis of having suffered from chronic anxiety for a period of at least three months, the possibility that the overall improvement was due to spontaneous remission appears to be unlikely. The two-way interaction between type of dependent variable and day revealed that there was a significant decrease in state anxiety as measured by ST, CC and PCA, indicating that levels of state anxiety decreased not only from a subjective point of view (the subject's) but also in the opinion of the psychiatrists. Levels of trait anxiety, considered to be indicative of the degree to which an individual is prone to experience periodic high levels of state anxiety, was unaffected by the overall treatment procedure (N and TR did not fluctuate significantly). In conclusion, subjects experienced less and less anxiety, and were judged by psychiatrists to have improved significantly over the six-

week span of the experimental period.

Further evidence for an overall treatment effect was found in the daily data, where a significant main effect of occasion of measure was revealed, along with a significant two-way interaction between type of dependent variable and day. The finding of the main effect of occasion of measure, in which the post-treatment levels were significantly decreased from pre-treatment levels, indicates that the experimental procedure served to affect the levels of anxiety, as tapped by the various daily measures, in the desired direction (decreasing). However, examination of the two-way interaction between type of dependent variable and day revealed that the physiological measures of anxiety tended to increase over days, whereas the opposite appeared to be true for the psychological measures. The post hoc analysis of these results did not identify any single significant contributor to the interaction, but visual inspection of the data in Figure 5 illustrates the slight upward trend for physiological measures in contrast to the downward trend for psychological measures evident in Figure 6. (A possible explanation for this particular finding will be considered later). The implications of these findings in the daily data are that while the overall procedure had an apparent immediate effect (pre-treatment versus post-treatment), it was not one which brought about any significant decrease in anxiety from day to day, at least in terms of what the daily anxiety measures tapped. However, the overall procedure was obviously

effective in reducing anxiety over weeks.

To summarize, while there was an immediate effect (pre-post), it did not serve to produce a detectable decrease in daily anxiety over a five-day treatment period; instead, a significant decrease in state anxiety gradually emerged over weeks, with the greatest reduction in anxiety being found between baseline (Day 1) and the one-month follow-up (Day 43).

Placebo Responders

From the evidence discussed in the preceding section it seems reasonable to suppose that an overall placebo response produced the improvement in anxiety neurosis in the subjects who participated in this study. The significant correlation found between overall response to treatment (OR) and extraversion (E) demonstrates that the degree of response to the non-specific effects of the overall treatment procedure in this study was related to the EPI E score. The higher the E score the greater was the overall response to treatment. However, this should be qualified by reiterating that all subjects in this study had a uniformly high neuroticism (N) score. Similarly, the response to the overall treatment procedure, as measured by the decline in pulse rate on Day 5 was related to the E score. This relationship, which was significant for the placebo treatment condition only, demonstrated that the higher the E score, the greater was the decline in pulse rate on Day 5.

The unexpected finding in this study of a significant relationship between EPI personality dimensions and placebo response prompted a search of the literature dealing specifically with the question of personality and response to placebo. In a comprehensive discourse on aspects of placebos, Claridge (1970) stated that there are three components to any situation involving drug therapy: the drug itself, the individual taking the drug and the person dispensing it, and it would appear that these elements are also present in an electrical treatment such as CET. Insofar as the personality of the individual taking the drug is concerned, Claridge maintained that placebo responders typically manifested anxiety and were generally neurotic, but were neither consistently extraverted nor introverted. He stated: "The truth is that extensive research has failed to identify any particular "placebo type" who will respond to placebos consistently under all conditions" (p. 37). Claridge conceded, however, that there was some evidence to suggest that certain personality characteristics were found more often in placebo reactors than non-reactors. He mentioned suggestibility and acquiesence as two of these characteristics. There is, however, more recent evidence which suggests that definite trends regarding the dimensions of introversion-extraversion are to be found in placebo responders. For example, Shipman, Greene and Laskin (1974) cited investigators who have described placebo responders as manifesting high levels of anxiety and being more dependent on outside

stimulation than on their own inner mental processes; that is, they were more "field-dependent" or extraverted. On the other hand, placebo non-responders were seen as relying less on external cues and being more introspective and hostile. In their own investigation, the authors found that normal subjects responded less to a placebo combined with strong suggestion than did psychoneurotics, who were defined as being "emotionally troubled people who are handicapped in life by their own personality defects" (p. 477). The authors also found that depressives with hypochondriasis and hysterics demonstrated a consistently favourable response to placebos, and that psychopathic and schizoid individuals generally did not respond to placebos.

Another investigation of personality dimensions and placebo response, carried out by Muller (1968) revealed that consistent placebo responders were out-going, enthusiastic, and verbally and socially skilled. Non-responders were found to be antagonistic and hostile. Similarly, Capone, Brahen and Weichert (1976) found that there was a significant relationship between hysteria and self-reported effects under not only placebo but also drug conditions.

In the studies under consideration, individuals with hysteria were found to be placebo responders, and people with such a condition generally obtain high scores on the EPI N and E scales (Eysenck, 1968). However, when individuals were psychopathic, a condition also associated with high levels of both E and N (Eysenck, 1968), they generally did

not respond to placebos. Since psychopathic behaviour is associated with belligerence and hostility (Freedman et al., 1972), the findings of non-response to placebos in individuals with such a disorder are consistent with those of Muller (1968).

Based on the findings in these reports and the results of this study, it would appear that there is sufficient evidence to show that some of the personality factors associated with placebo response, and possibly with response to active treatment, are high levels of trait anxiety and extraversion. However, this combination of high anxiety and extraversion is not related to placebo response in all psychiatric diagnoses. As Claridge (1970) stated in this regard:

> Relationships between personality traits and placebo reaction that are found in one situation may not appear, or may even be reversed, in another situation....The personality of the placebo reactor then, is only important insofar as it conditions his response to certain other features that may be present in the drug situation (p. 39, 42).

Similarly Shipman et al. (1974), echoed Claridge when they pointed out that personality dimensions were not the only factors affecting placebo response, which was described as "a complex phenomenon which is dependent on the interaction between personality characteristics, situational variables and mode of placebo presentation" (p. 482).

Further investigation of the therapeutic effectiveness of CET, or any other electrical or chemical treatment

would necessitate the careful control of the variables associated with response to treatment, whether under placebo or active conditions, beginning with the important one of personality dimensions.

Differential Effect of Active and Placebo Treatment

In the analysis of both the weekly and daily data, no specific treatment effect was detected, confirming Hypothesis II. In other words, the effects produced by active and placebo treatment were equal. Thus, we may conclude that whatever produced the significant overall treatment effect, it could not be attributed to the direct action of the current in active treatment. Therefore, a logical conclusion would appear to be that the components of suggestion, setting and cutaneous stimulation inherent in the experimental procedure combined to produce the improvement obtained in the subjects' condition. Thus, evidence which supports the indirect effect theory of CET mode of action was obtained in this study.

Order Effects

A puzzling finding was the significant interaction found in the analysis of weekly data between order and type of dependent variable. No significant single contributor to this interaction was found in post hoc analyses. Inspection of the data in Table 6 suggests that there may have been a difference, albeit not statistically significant, between

the two groups in regard to trait anxiety levels (note that order was a between-subject variable), and that this was a chance occurrence. This possibility is illustrated by the different N and TR levels for the two groups. An alternative possibility is that order AP may have been less effective in ameliorating that group's condition as opposed to the apparent beneficial effect order PA had for the second group. In fact, order PA seems to have been associated with better results not only on the weekly data, but also on the daily data, as we saw in the follow-up analysis of the five-way interaction, where only order PA was found to be significant for the four-way interaction among treatment, type of dependent variable, day and occasion of measure.

It should be kept in mind that order was an experimental control and not an experimental manipulation. As a result, the only conclusion that might reasonably be drawn from these data is that there may have been an effect in which subjects in group PA simply became accustomed to the whole procedure in the first week, and that in the second week, during which the PA group received active treatment, the administering of the actual current (which was the only differentiating factor between what happened to the PA and AP groups in the second week of treatment), contributed to the difference between the groups.

Direction of Current Flow

The overall improvement in anxiety levels, which was found to be statistically significant for both the weekly and daily data, would lead to the expectation of a decrease in not only the psychological measures, but also the physiological indices of anxiety. Visual inspection of the daily data in Figures 5 and 6 shows that in the two-way interaction between type of dependent variable and day, the psychological anxiety levels did, in fact, decrease, whereas the physiological indices show a clear increase. The follow-up analysis of variance on these data did not reveal any single significant contributor to the interaction, indicating that none of the six daily types of dependent variable, on their own, produced a main day effect. In other words, there was neither a significant decrease nor increase on any of the psychological and physiological variables.

In spite of the statistical insignificance of the fluctuation over days of both pulse and respiration rates, as well as systolic blood pressure levels, it is worth noting that Nias (1976) cited works, including his own, in which an "alerting effect" occurred in patients receiving constant-direct currents when forehead electrodes were positive (as was the case during active treatment in this study). An opposite, or "sedating effect" occurred when forehead electrodes were negative. One would expect an alerting effect to create an acceleration of autonomic responses. However, the possible existence of an alerting

effect does not satisfactorily explain the increase in physiological measures found in this study, for several reasons: (1) The studies cited by Nias differed from the present one in that they involved a procedure commonly known as polarization, in which constant, as opposed to pulsating, direct currents are used, and the second pair of electrodes is attached to the leg and not to the mastoid processes; and (2) no differential effect between active and placebo treatment was detected in this study, indicating that, had the alerting effect occurred, unequal effects between active and placebo treatment would have been noted.

Conclusions

This study of CET effectiveness on anxiety, in which a statistically significant overall effect was revealed, but in which no significant difference between active and placebo treatment was detected, is consistent with the findings of other studies in which the absence or presence of cutaneous stimulation during both active and placebo treatment was held constant (Hearst et al., 1974; Marshall et al., 1974; Moore et al., 1975). The results of this study provide evidence for supporting the indirect effect theory of CET mode of action, which holds that non-specific elements of the therapeutic procedure, which may include suggestion, setting and rhythmic cutaneous stimulation, and not the direct action of the current on the brain cells, are respon-

sible for any obtained improvement of a target disorder. There is also evidence that extraverts are more likely to respond to such a placebo effect than are introverts, given that both groups manifest high levels of trait anxiety. In this study, both subjects and psychiatrists noted an overall improvement in anxiety levels, although the physiological data did not reflect this decrease in anxiety levels. From the data obtained in this study, it is impossible to establish to what extent the elements of suggestion, setting and cutaneous stimulation each contributed to the overall improvement. The use of a control group, in which subjects would not have received any cutaneous stimulation at all, but simply have reposed in a quiet, semi-dark room, would have helped to answer this question.

Based on the results of this and other studies in which identical placebo and active treatment conditions were created, it is doubtful that the usefulness of CET can continue to be attributed to the healing force of the electrical current, and it is probable that a daily half-hour regimen of quiet, undisturbed repose in a comfortable setting would prove to be as effective as a course of CET.

BIBLIOGRAPHY

American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 2nd edition (DSM-II), (Washington, D.C., 1968).

Banshchikov, V.M. Present status of electrosleep in USSR. In Electrotherapeutic Sleep and Electroanesthesia, F.M. Wageneder, St. Schuy (eds.). (Amsterdam: Excerpta Medica Foundation, 1967).

Bendig, H.W. & Bruder, G. The effect of repeated testing on anxiety scale scores. Journal of Consulting Psychology, 1962, 26, 392.

Boblitt, W.E. Electrosleep as a sleep induction method. The Psychiatric Forum, 1969, Winter, 9-14.

Brand, J. Electrosleep therapy for migraine and headache. In Electrotherapeutic Sleep and Electroanesthesia, F.M. Wageneder, St. Schuy (eds.). (Amsterdam: Excerpta Medica Foundation, 1970).

Brown, C.C. Electroanesthesia and electrosleep. American Psychologist, 1975, March, 402-410.

Buss, A.H. Two anxiety factors in psychiatric patients. Journal of Abnormal and Social Psychology, 1962, 65, 426-427.

Capone, T., Brahen, L.S. & Wiechert, V. Personality factors and drug effects in a controlled study of cyclazoline. Journal of Clinical Psychology, 1976, 32 (1), 489-495.

Cattell, R.B. & Scheier, I.H. The Meaning and Measurement of Neuroticism and Anxiety. (New York: Ronald Press Co.,1961).

Chassan, J.B. Research Design in Clinical Psychology and Psychiatry. (New York: Appleton-Century-Crofts, 1967).

Chumakova, L.T. & Kirillova, Z.A. Electrosleep as an effective outpatient treatment for nervous and psychological disorders. In Innovative Medical Psychiatric Therapies, Suinn, R.M., Weigel, R.D. (eds.). (New York: University Press, 1976).

Claridge, G. Drugs and Human Behaviour. (Harmondsworth: Penguin Books, 1970).

Constant, J. Bedside Cardiology, 2nd edition. (Boston: Little, Brown & Company, 1976).

Cox, W.C. & Heath, R.G. Neurotone therapy: a preliminary report of its effect on electrical activity of forebrain structures. Diseases of the Nervous System, 1975, 36, 5, 245-247.

Dodge, C.H. Electrosleep, electroanesthesia, and electroneural diagnostics and therapeutics. Foreign Science Bulletin, 1967, 3 (3), 46-64.

Dymond, A.M., Coger, R.W., & Serafetinides, E.A. Intracerebral current levels in man during electrosleep therapy. Biological Psychiatry, 1975, 10 (1), 101-104.

Eaton, M.T., Peterson, M.H. Psychiatry, 2nd edition. (Flushing, N.Y.: Medical Examination Publishing Co., Inc., 1969).

Eysenck, H.J. The Handbook of Abnormal Psychology. (New York: Basic Books, 1961).

Eysenck, H.J. & Eysenck, S.B.G. Manual for the Eysenck Personality Inventory. (San Diego: Educational and Industrial Testing Services, 1968).

Feighner, J.P., Brown, S.L. & Olivier, J.E. Electrosleep therapy. Journal of Nervous and Mental Disease, 1973, 157 (2), 121-128.

Forster, S., Post, B.S. & Benton, J.G. Preliminary observations on electrosleep. Archives of Physical Medicine and Rehabilitation, 1963, 44, 481-489.

Frankel, B.L. Research on cerebral electrotherapy (electrosleep): some suggestions. American Journal of Psychiatry, 1974, 131 (1), 95-98.

Frankel, B.L., Buchbinder, R. & Snyder, F. Ineffectiveness of electrosleep in chronic primary insomnia. Archives of General Psychiatry, 1973, 29, 563-568.

Freedman, A.M., Kaplan, H.I. & Sadock, B.J. Modern Synopsis of a Comprehensive Textbook of Psychiatry. (Baltimore: Williams & Wilkins Co., 1972).

Grunner, O. The influence of electrical pulse currents of various frequencies, applied transcerebrally, on the production of electrosleep, and the evaluation of the direct and psychotherapeutic effects of electrosleep. In Electrotherapeutic Sleep and Electroanesthesia, F.M. Wageneder, St. Schuy (eds.). (Amsterdam: Excerpta Medica Foundation, 1970).

Hamilton, M. The assessment of anxiety states by rating. British Journal of Medical Psychology, 1959, 32, 50-59.

Hearst, E.D., Cloninger, C.R., Crews, E.L. & Cadoret, R.J. Electrosleep therapy: a double-blind trial. Archives of General Psychiatry, 1974, 30, 463-466.

Howard, K.I. & Diesenhaus, H. 16 PF item response patterns of repeated testing. Educational and Psychological Measurement, 1965, 25, 365-379.

Itil, T., Ganhon, P., Akpinar, S. & Hsu, W. Quantitative EEG analysis of electrosleep using frequency analyzer and digital computer methods. Electroencephalography and Clinical Neurophysiology, 1971, 31, 294.

Iwanovsky, A. Report on international progress in cerebral electrotherapy (electrosleep) and electroanesthesia. Foreign Science Bulletin, 1969, 5 (10), 15-40.

Iwanovsky, A. & Dodge, C.H. Electrosleep and electroanesthesia: theory and clinical experience. Foreign Science Bulletin, 1968, 4 (2), 1-64.

Keppel, G. Design and Analysis: A Researcher's Handbook. (Englewood Cliffs: Prentice Hall, 1973).

Koegler, R.R., Hicks, S.M. & Barger, J.H. Medical and psychiatric use of electrosleep: transcerebral electrotherapy. Diseases of the Nervous System, 1971, 32, 100-104.

Kalinowsky, L.B. & Hippius, H. Pharmacological, Convulsive and Other Somatic Treatments in Psychiatry. (New York: Grune & Stratton, 1969).

Lechner, H., Geyer, N., Pfurtscheller, G., St. Schuy & Wageneder, F.M. Electroencepalographic studies in electrosleep. In Electrotherapeutic Sleep and Electroanesthesia, F.M. Wageneder, St. Schuy (eds.). (Amsterdam: Excerpta Medica Foundation, 1967).

Lovell, G.D. & Morgan, J.J.B. Physiological and motor responses to a regular recurring sound: a study in monotony. Journal of Experimental Psychology, 1942, 30, 435-451.

Marshall, A.G. & Izard, C.C. Cerebral electrotherapeutic treatment of depressions. Journal of Consulting and Clinical Psychology, 1974, 42 (1), 93-97.

Moore, J.A., Mellor, C.S., Standage, K.F. & Strong, H. A double-blind study of electrosleep for anxiety and insomnia. Biological Psychiatry, 1975, 10 (1), 59-63.

Muller, B.P. Personality of placebo reactors and non-reactors. Diseases of the Nervous System, 1965, 26, 58-61.

Neurotone Model 101 Instruction Manual. (Garland: Neuro Systems, 1971).

Nias, D.K.B. Therapeutic effects of low-level direct electrical currents. Psychological Bulletin, 1976, 83 (5), 766-773.

Obrosow, A.E. Electrosleep therapy. In Therapeutic Electricity and Ultraviolet Radiation, E. Licht (ed.). (New Haven: Editor, 1959).

Oswald, I. Falling asleep open-eyed during intense rhythmic stimulation. British Medical Journal, 1960, 1, 1450-1451.

Reigel, D.H., Dallmann, D.E., Christman, N.T., Hamilton, L.H., Luperku, E.J., Henschel, E.O., Larson, S.J. & Sances, A. Jr. Physiological effects of electro-therapeutic currents in the primate and man. In Electrotherapeutic Sleep and Electroanesthesia, F.M. Wageneder, St. Schuy (eds.). (Amsterdam: Excerpta Medica Foundation, 1970).

Rosenthal, S.H. Alterations in serum thyroxine with cerebral electrotherapy (electrosleep). Archives of General Psychiatry, 1973, 28, 28-29.

Rosenthal, S.H. Electrosleep: A double-blind clinical study. Biological Psychiatry, 1972a, 4 (2), 179-185.

Rosenthal, S.H. Electrosleep therapy. In Current Psychi-atric Therapies, Vol. 12, J.H. Masserman (ed.). (New York: Grune & Stratton, 1972b).

Rosenthal, S.H. & Calvert, L.F. Electrosleep: personal subjective experiences. Biological Psychiatry, 1972, 4 (2), 187-190.

Rosenthal, S.H. & Wolfsohn, N.L. Electrosleep. Journal of Nervous and Mental Disease, 1970, 151 (2), 146-151.

Rush, S. & Driscoll, D.A. Current distribution in the brain from surface electrodes. Anesthesia and Analgesia, 1968, 47 (6), 717-123.

Scheier, I.H. & Cattell, R.B. Handbook and Test Kit for the IPAT 8-Parallel-Form Anxiety Battery. (Champaign: Institute for Personality and Ability Testing, 1973).

Shipman, W.G., Greene, C.S. & Laskin, D.M. Correlation of placebo responses and personality characteristics in myofascial pain-dysfunction (MFD) patients. Journal of Psychosomatic Research, 1974, 18, 475-483.

Smith, R.B. & O'Neill, L. Electrosleep in the management of alcoholism. Biological Psychiatry, 1975, 10 (6), 675-679.

Spielberger, C.D., Gorsuch, R.L. & Lushene, R.E. STAI Manual. (Palo Alto: Consulting Psychologists Press, 1970).

Straus, B., Elkind, A. & Bodian, C.A. Electrical induction of sleep. American Journal of Medical Science, 1964, 248, 514-520.

Tomsovic, M. & Edwards, R.V. Cerebral electrotherapy for tension-related symptoms in alcoholics. Quarterly Journal of Studies on Alcohol, 1973, 34, 1352-1355.

Van Poznak, A. Advances in electrosleep and electro-anesthesia during the past decade. Clinical Anesthesia, 1969, 3, 501-520.

Wageneder, F.M., Iwanovsky, A., Dodge, . & Christopher, H. Electrosleep (cerebral electrotherapy) and electro-anesthesia: the international effort at evaluation. Foreign Science Bulletin, 1969, 5 (4), 1-104.

Wageneder, F.M. & St. Schuy (eds.). Electrotherapeutic Sleep and Electroanesthesia. (Amsterdam: Excerpta Medica Foundation, 1967).

Wageneder, F.M. & St. Schuy (eds.). Electrotherapeutic Sleep and Electroanesthesia. (Amsterdam: Excerpta Medica Foundation, 1970).

Weinberg, A. Clinical observations in the use of electro-sleep. Journal of American Society of Psychosomatic Dentistry and Medicine, 1969, 16 (2), 35-39.

Weiss, M.F. The treatment of insomnia through the use of electrosleep: an EEG study. Journal of Nervous and Mental Diseases, 1973, 157 (2), 108-120.

Welkowitz, J., Ewen, R.B. & Cohen, J. Introductory Statistics for the Behavioural Sciences. (New York: Academic Press, 1971).

Wilson, A.S., Reigel, D., Unger, G.F., Larson, S.J. & Sances, A. Jr. Gastric secretion before and after electro-therapeutic sleep in executive monkeys. In Electro-therapeutic Sleep and Electroanesthesia, F.M. Wageneder, St. Schuy (eds.). (Amsterdam: Excerpta Medica Foundation, 1970).

Wolff, M.R. The effect of different pulse frequencies on electrosleep. In Electrotherapeutic Sleep and Electro-anesthesia, F.M. Wageneder, St. Schuy (eds.). (Amsterdam: Excerpta Medica Foundation, 1970).

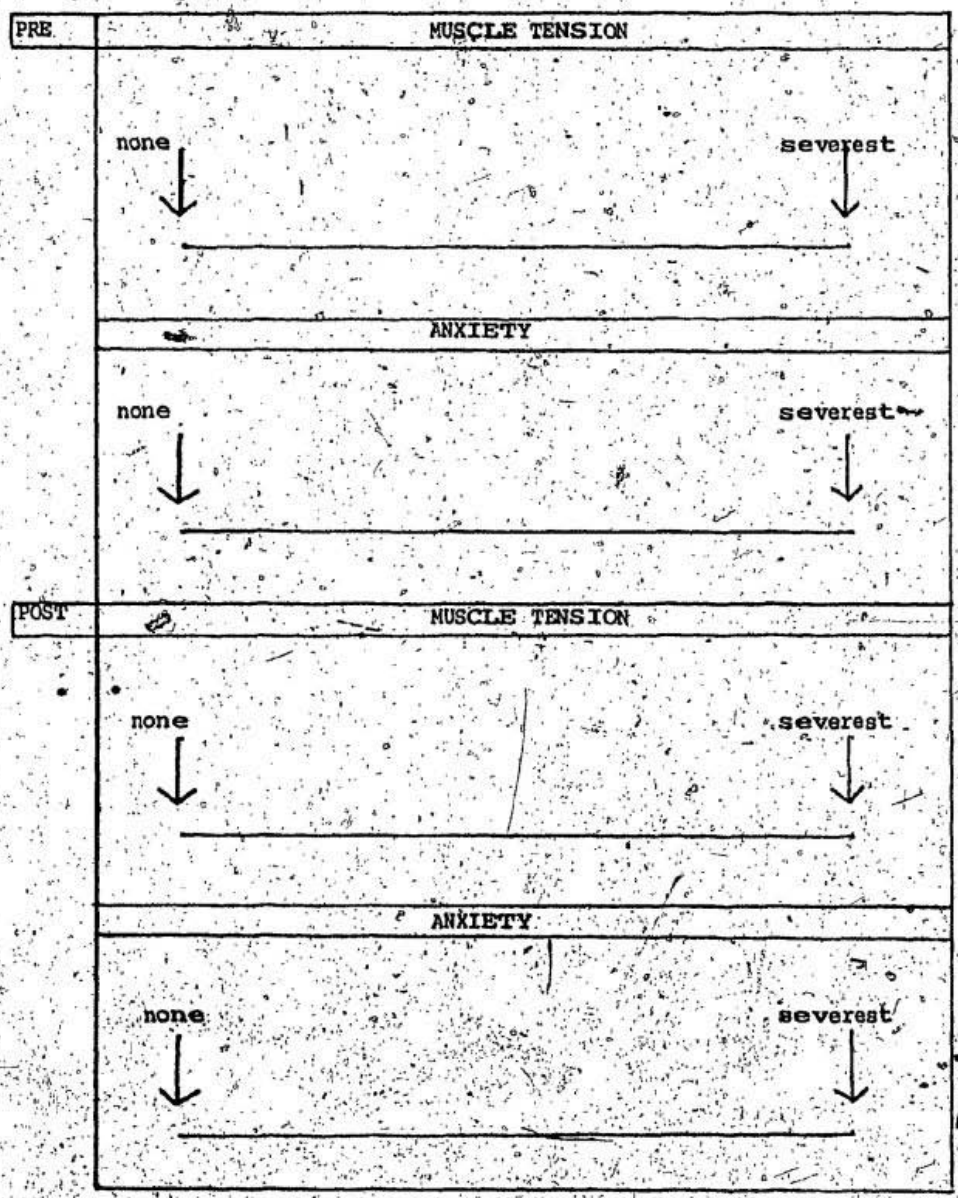
APPENDIX A

MUSCLE TENSION-ANXIETY SELF-RATING SCALE

NAME: _____

DAY: _____

INSTRUCTIONS: Put a vertical pencil mark through the line at the point which best describes your present level (at this moment) of muscle tension and anxiety.



COMPLAINT CHECKLIST

NAME:

COMPLAINT:

A.

B.

C.

D.

SEVERITY: (0-none, 5-moderate, 10-severest)

COMPLAINT	DAY 1	8	15	FOLLOW-UP 22 (1 WEEK)	43 (1 MONTH)
A.					
B.					
C.					
D.					

ELECTRO-RELAXATION THERAPY

CLINICAL ASSESSMENT

NAME: _____ AGE: _____ SEX: _____

DIAGNOSIS: _____ MEDICATION: _____

_____ _____

DURATION SYMPTOMS: _____

ANXIETY		nil ↓ 0	I	2	moderate ↓ 3	4	5	severe ↓ 6
Subjective - specify								
1	1							
2	2							
8	8							
22	22							
43	43							
Somatic - specify								
1	1							
2	2							
8	8							
22	22							
43	43							
DEPRESSION								
Experiential -specify								
1	1							
2	2							
8	8							
22	22							
43	43							
Expressive - specify								
1	1							
2	2							
8	8							
22	22							
43	43							
SLEEP								
Duration								
1	1							
2	2							
8	8							
22	22							
43	43							
Quality								
1	1							
2	2							
8	8							
22	22							
43	43							

APPENDIX B

TABLE 11

All possible comparisons between means on interaction between dependent variable and order (weekly data)

		TYPE OF DEPENDENT VARIABLE (DV)									
		PCA	TR	N	CC	ST	ST	CC	N	TR	PCA
	ORDER	AP	PA	PA	AP	PA	AP	PA	AP	AP	PA
DV		1185.80	1186.00	1186.53	1218.93	1247.65	1252.38	1281.08	1313.48	1314.00	1314.20
PCA	1185.80	-	.20	.73	33.13	61.85	66.58	95.28	127.68	128.20	128.40
TR	1186.00	-	--	.53	32.93	61.65	66.38	95.08	127.48	128.00	128.20
N	1186.53	-	-	-	32.40	61.12	65.85	94.55	126.95	127.47	127.67
CC	1218.93	-	-	-	-	28.72	33.45	62.15	94.55	95.07	95.27
ST	1247.65	-	-	-	-	-	4.73	33.43	65.83	66.83	67.03
ST	1252.38	-	-	-	--	-	-	28.70	61.10	61.62	61.82
CC	1281.08	-	-	-	-	-	-	-	32.40	32.92	33.12
N	1313.48	-	-	-	-	-	-	-	-	.52	.72
TR	1314.00	-	-	-	-	-	-	-	-	-	.20
PCA	1314.20	-	-	-	-	-	-	-	-	-	-

Tukey's test,
critical range = 147.73, p = .05

TABLE 12

Means and standard deviations of follow-up analysis of variance on interaction between type of dependent variable and day (weekly data)

	N		ST		TR		CC		PCA	
DAY	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD	$\bar{X}$	SD
1	18.0	1.83	54.3	14.89	55.50	9.82	7.70	2.45	2.61	.90
8	17.8	4.32	50.1	21.73	57.50	13.19	5.60	3.89	1.48	1.11
15	16.7	5.25	47.0	22.27	53.90	17.68	4.80	3.97	1.22	1.26
22	16.4	4.97	47.3	21.20	57.30	12.90	4.10	3.78	1.08	1.11
43	17.7	4.90	41.0	16.47	53.00	12.03	3.30	3.50	1.31	1.23

TABLE 13

Summary of follow-up analysis of variance on interaction between type of dependent variable and day (weekly data)

ST

Source	SS	df	MS	F-ratio	p
Order (O)	.447	1	.447	.001	.97518
Error	3905.640	8	488.205		
Day (D)	260.719	4	65.180	2.755	.04417*
O x D	76.166	4	19.041	.805	.53301
Error	757.023	32	23.657		
Total	4999.990	49			

CC

Source	SS	df	MS	F-ratio	p
Order (O)	77.283	1	77.283	.218	.65600
Error	2839.440	8	354.930		
Day (D)	828.340	4	207.085	5.888	.00143*
O x D	129.437	4	32.359	.920	.53399
Error	1125.490	32	35.172		
Total	4999.990	49			

PCA

Source	SS	df	MS	F-ratio	p
Order (O)	329.824	1	329.824	1.056	.33561
Error	2497.730	8	312.217		
Day (D)	1049.950	4	262.487	10.270	.00007*
O x D	304.613	4	76.153	2.980	.03323*
Error	817.887	32	25.559		
Total	5000.000	49			

(continued)

Table 13 (continued)

N

Source	SS	df	MS	F-ratio	p
Order (O)	322.255	1	322.255	1.082	.33007
Error	2383.520	8	297.940		
Day (D)	114.269	4	28.567	.433	.78581
O x D	67.855	4	16.964	.257	.90238
Error	2112.110	32	66.003		
Total	5000.010	49			

TR

Source	SS	df	MS	F-ratio	p
Order (O)	327.665	1	327.665	.659	.55486
Error	3977.180	8	497.147		
Day (D)	97.657	4	24.414	1.367	.26645
O x D	26.119	4	6.530	.366	.83220
Error	571.374	32	17.855		
Total	5000.000	49			

TABLE 14

Summary of follow-up analysis of variance on interaction between type of dependent variable and day (daily data)

Pulse

Source	SS	df	MS	F-ratio	p
Order (O)	485.143	1	485.143	.235	.64386
Error	16487.600	8	2060.950		
Treatment (T)	2.297	1	2.297	.043	.83473
O x T	100.052	1	100.052	1.869	.20720
Error	428.275	8	53.534		
Day (D)	188.608	4	47.152	2.275	.08206
O x D	68.048	4	17.012	.821	.52323
Error	663.102	32	20.722		
T x D	33.483	4	8.371	.334	.85361
O x T x D	30.971	4	7.743	.309	.87002
Error	802.430	32	25.076		
Occasion of Measure (OM)	135.005	1	135.005	11.950	.00863*
O x OM	9.810	1	9.810	.868	.61843
Error	90.383	8	11.298		
T x OM	.368	1	.368	.043	.83455
O x T x OM	5.880	1	5.880	.688	.56455
Error	68.375	8	8.547		
D x OM	35.422	4	8.856	1.988	.11937
O x D x OM	21.682	4	5.421	1.217	.32281
Error	142.569	32	4.455		
T x D x OM	46.897	4	11.724	2.632	.05173*
O x T x D x OM	11.066	4	2.767	.621	.65366
Error	142.540	32	4.454		
Total	20000.000	199			

(continued)

Table 14 (continued).

Respiration

Source	SS	df	MS	F-ratio	p
Order (O)	3144.090	1	3144.090	5.324	.04822*
Error	4724.230	8	590.528		
Treatment (T)	238.794	1	238.794	1.574	.24409
T x O	238.796	1	238.796	1.574	.24409
Error	1213.460	8	151.682		
Day (D)	280.510	4	70.127	.948	.54950
O x D	269.204	4	67.301	.910	.52842
T x O	2366.500	32	73.953		
T x D	66.277	4	16.569	.311	.86867
O x T x D	118.907	4	29.727	.558	.69782
Error	1705.670	32	53.302		
Occasion of Measure (OM)	1.754	1	1.754	.016	.89772
O x OM	15.790	1	15.790	.144	.71333
Error	874.274	8	109.284		
T x OM	85.966	1	85.966	.923	.63276
O x T x OM	296.494	1	296.494	3.183	.10995
Error	745.239	8	93.153		
D x OM	178.559	4	44.640	1.035	.40523
O x D x OM	117.738	4	29.435	.682	.61186
Error	1380.140	32	43.129		
T x D x OM	124.563	4	31.141	.662	.62565
O x T x D x OM	307.802	4	76.951	1.636	.18856
Error	1505.260	32	47.040		
Total	20000.000	199			

(continued)

Table 14 (continued)

Systolic blood pressure

Source	SS	df	MS	F-ratio	p
Order (O)	14.762	1	14.762	.013	.90982
Error	9426.550	8	1178.320		
Treatment (T)	6.561	1	6.561	.058	.80932
O x T	148.027	1	148.027	1.317	.28433
Error	899.233	8	112.404		
Day (D)	468.478	4	117.119	1.607	.19562
O x D	459.867	4	114.967	1.578	.20323
Error	2331.520	32	72.860		
T x D	416.812	4	104.203	1.693	.17500
O x T x D	373.758	4	93.439	1.519	.21942
Error	1969.050	32	61.533		
Occasion of Measure (OM)	49.616	1	49.616	.469	.51802
O x OM	104.971	1	104.971	.993	.65004
Error	845.924	8	105.740		
T x OM	14.762	1	14.762	.811	.60252
O x T x OM	3.690	1	3.690	.203	.66669
Error	145.567	8	18.196		
D x OM	70.323	4	17.581	.448	.77511
O x D x OM	10.866	4	2.717	.069	.98791
Error	1255.560	32	39.236		
T x D x OM	103.126	4	25.782	1.001	.42255
O x T x D x OM	56.792	4	14.198	.551	.70237
Error	824.215	32	25.757		
Total	20000.000	199			

(continued)

Table 14 (continued)

Muscle tension

Source	SS	df	MS	F-ratio	p
Order (O)	44.643	1	44.643	.022	.87957
Error	16045.800	8	2005.720		
Treatment (T)	20.393	1	20.393	.183	.68184
O x T	30.576	1	30.576	.274	.61907
Error	893.103	8	111.638		
Day (D)	129.526	4	32.382	1.209	.32614
O x D	8.562	4	2.141	.080	.98499
Error	857.401	32	26.794		
T x D	51.977	4	12.994	.524	.72135
O x T x D	106.078	4	26.520	1.070	.38805
Error	793.105	32	24.785		
Occasion of measure (OM)	68.034	1	68.034	5.602	.04391*
O x OM	9.531	1	9.531	.785	.59481
Error	97.160	8	12.145		
T x OM	.034	1	.034	.002	.96132
O x T x OM	17.972	1	17.972	1.260	.29446
Error	114.090	8	14.261		
D x OM	10.046	4	2.512	.047	.80391
O x D x OM	23.926	4	5.982	.969	.56064
Error	197.541	32	6.173		
T x D x OM	130.302	4	32.575	3.007	.03212*
O x T x D x OM	3.532	4	.883	.082	.98452
Error	346.719	32	10.835		
Total	20000.000	199			

(continued)

Table 14 (continued)

Anxiety

Source	SS	df	MS	F-ratio	p
Order (O)	45.781	1	45.781	.024	.87619
Error	15554.800	8	1944.350		
Treatment (T)	68.089	1	68.089	.612	.53827
O x T	153.198	1	153.198	1.377	.27420
Error	890.318	8	111.290		
Day (D)	185.846	4	46.462	1.995	.11819
O x D	52.298	4	13.075	.561	.69516
Error	745.137	32	23.286		
T x D	78.471	4	19.618	.811	.52902
O x T x D	54.531	4	13.633	.564	.69349
Error	773.656	32	24.177		
Occasion of measure (OM)	124.091	1	124.091	9.318	.01536*
O x OM	23.701	1	23.701	1.780	.21749
Error	106.534	8	13.317		
T x OM	10.356	1	10.356	2.116	.18190
O x T x OM	27.024	1	27.024	5.523	.04508*
Error	39.145	8	4.893		
D x OM	119.911	4	29.978	2.217	.08850
O x D x OM	33.902	4	8.476	.627	.64964
Error	432.628	32	13.520		
T x D x OM	7.262	4	1.815	.178	.94596
O x T x D x OM	146.949	4	36.737	3.602	.01547*
Error	326.334	32	10.198		
Total	20000.000	199			

(continued)

Table 14 (continued)

EPFAB

Score	SS	df	MS	F-ratio	p
Order (O)	1589.090	1	1589.090	1.094	.32736
Error	11618.200	8	1452.280		
Treatment (T)	108.699	1	108.699	1.191	.30765
O x T	108.698	1	108.698	1.191	.30765
Error	730.418	8	91.302		
Day (D)	126.000	4	31.500	1.094	.37669
O x D	178.654	4	44.664	1.551	.21042
Error	921.481	32	28.796		
T x D	32.346	4	8.086	.334	.85329
O x T x D	32.346	4	8.086	.334	.85329
Error	774.050	32	24.189		
Occasion of measure (OM)	.000	1	.000	.000	.99547
O x OM	.000	1	.000	.000	1.00000
Error	124.114	8	15.514		
T x OM	54.161	1	54.161	5.189	.05052*
O x T x OM	24.071	1	24.071	2.306	.16527
Error	83.500	8	10.438		
D x OM	425.010	4	106.253	2.289	.08067
O x D x OM	240.714	4	60.179	1.296	.29179
Error	1485.660	32	46.427		
T x D x OM	135.779	4	33.945	1.165	.34436
O x T x D x OM	274.005	4	68.736	2.360	.07352
Error	932.005	32	29.125		
Total	20000.000	199			

TABLE 15

Summary of analysis of variance on pulse and respiration data including the mid-treatment measure

Source	SS	df	MS	F-ratio	p
Order (O)	4330.880	1	4330.880	2.397	.15803
Error	14454.800	8	1806.850		
Treatment (T)	2.060	1	2.060	.031	.85757
O x T	21.294	1	21.294	.324	.58942
Error	252.099	8	65.637		
Type of dependent variable (DV)	0.000	1	0.000	0.000	.99551
O x DV	935.834	1	935.834	.452	.52561
Error	16562.200	8	2070.280		
T x DV	45.132	1	45.132	1.061	.33462
O x T x DV	151.029	1	151.029	3.550	.09403
Error	340.349	8	42.544		
Day (D)	254.557	4	63.639	.802	.53486
O x D	463.679	4	115.920	1.461	.23639
T x D	284.687	4	71.172	1.525	.21754
O x T x D	61.616	4	15.404	.330	.85607
Error	237.133	4	59.283	.617	.65642
O x DV x D	153.466	4	38.367	.399	.80911
Error	3074.280	32	96.071		
T x DV x D	105.647	4	26.412	.513	.72900
O x T x DV x D	157.519	4	39.380	.766	.55756
Error	1646.050	32	51.439		
Occasion of measure (OM)	100.084	2	50.042	1.288	.30287
O x M	2.883	2	1.441	.037	.96411
Error	621.486	16	38.843		
T x OM	218.686	2	109.343	1.479	.25681
O x T x OM	314.765	2	157.382	2.129	.15018

(continued)

Table 15 (continued)

Source	SS	df	MS	F-ratio	p
Error	1183.000	16	73.937		
DV x OM	73.580	2	36.790	.672	.52836
O x DV x OM	53.185	2	26.593	.486	.62881
Error	875.555	16	54.722		
T x DV x OM	159.188	2	79.594	.811	.53472
O x T x DV x OM	270.795	2	135.398	1.380	.27964
Error	1569.910	16	98.120		
D x OM	193.780	8	24.223	1.029	.42434
O x D x OM	103.138	8	12.892	.548	.81694
Error	1506.630	64	23.541		
T x D x OM	198.337	8	24.792	1.364	.22883
O x T x D x OM	231.217	8	28.902	1.590	.14505
Error	1163.240	64	18.176		
DV x D x OM	134.354	8	16.794	.757	.64257
O x DV x D x OM	207.040	8	25.880	1.167	.33242
Error	1419.340	64	22.177		
T x DV x D x OM	184.994	8	23.124	1.202	.31176
O x T x DV x D x OM	143.510	8	17.939	.932	.50290
Error	1231.420	64	19.241		
Total	60000.000	599			

APPENDIX C

ACTIVE TREATMENT (DAILY MEASURES)

ORDER			PULSE			RESPIRATION			SYSTOLIC BLOOD PRESSURE		MT		A		EPFAB	
S#	Sex/Age	Day	Pre	Mid	Post	Pre	Mid	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
2	AP	1	72	76	75	18	19	18	90	100	.1	5.1	10	1.7	8	4
	(F,52)	2	82	71	78	19	18	17	100	100	2.7	2.6	3.4	2.9	5	4
		3	72	79	75	18	22	19	110	100	3.0	1.8	2.2	2.5	4	5
		4	84	85	83	20	29	22	80	100	.1	.1	.1	.2	3	2
		5	85	89	91	16	10	18	100	90	.7	.7	.6	.6	4	4
4	AP	1	87	87	84	18	18	20	85	85	9.-	8.4	8.6	8.1	7	10
	(F,32)	2	84	81	81	20	19	20	90	90	8.6	8.3	8.7	7.9	8	7
		3	85	88	85	15	18	17	100	95	9.6	9.2	9.7	9.5	7	7
		4	82	77	81	20	19	19	100	100	9.6	8.6	9.6	8.5	6	8
		5	82	75	76	20	20	19	95	95	9.7	9.0	9.7	9.0	8	7
6	AP	1	75	72	70	22	23	21	10	80	1.4	.5	.3	.3	8	7
	(F,36)	2	69	70	66	21	19	20	100	100	.3	.3	1.9	.3	7	7
		3	65	64	70	20	13	20	100	100	2.0	.3	.4	.3	7	6
		4	76	67	72	23	19	21	105	90	.2	.3	.3	.3	9	5
		5	65	70	71	20	20	21	100	100	.4	.5	.4	.6	5	9
8	AP	1	73	71	67	18	17	17	110	105	.7	.8	8.4	.7	7	7
	(M,31)	2	79	78	74	18	19	19	110	110	2.1	2.3	3.5	8.0	7	9
		3	78	79	78	18	18	18	110	110	5.0	1.2	8.3	8.8	9	10
		4	81	80	76	18	21	18	110	100	4.1	3.5	8.4	2.4	9	7
		5	82	71	74	21	20	20	100	100	3.2	2.4	7.5	6.5	7	7
10	AP	1	90	78	84	9	29	20	110	110	.4	.3	.4	.4	5	2
	(M,31)	2	79	73	72	15	27	18	110	100	3.0	1.1	.3	.2	5	3
		3	78	70	68	13	27	24	105	95	.2	.2	2.4	.2	2	5
		4	76	65	67	12	27	24	110	100	.1	.7	0	.8	3	4
		5	72	70	71	16	15	11	110	100	.1	.2	.2	2.8	2	4

(continued)

S#	ORDER Sex/Age	Day	PULSE Pre	PULSE Mid	PULSE Post	RESPIRATION Pre	RESPIRATION Mid	RESPIRATION Post	SYSTOLIC BLOOD PRESSURE Pre	SYSTOLIC BLOOD PRESSURE Post	MT Pre	MT Post	A Pre	A Post	EPFAB Pre	EPFAB Post
1	PA	8	66	68	67	12	12	12	70	70	.1	.1	.8	0	5	7
	(M,26)	9	70	64	61	13	12	13	70	90	0	0	0	0	10	4
		10	71	71	72	13	13	15	80	80	0	0	0	0	5	7
		11	80	76	80	14	15	15	80	80	0	0	0	0	5	7
		12	79	81	83	15	16	16	90	90	0	0	0	0	9	8
3	PA	8	58	57	58	19	19	51	100	105	1.2	.2	.7	.2	6	6
	(M,25)	9	57	57	59	22	22	20	90	90	.6	.2	.8	.2	6	5
		10	64	63	63	15	15	19	105	95	.2	.3	.4	.2	4	2
		11	64	64	61	23	23	20	125	115	.2	.2	.1	0	3	5
		12	68	63	64	17	22	24	105	105	.3	.1	.1	.1	5	4
5	PA	8	98	97	93	17	17	15	80	75	7.-	6.3	8.4	9.4	10	9
	(M.19)	9	95	95	90	16	20	17	80	90	8.7	8.4	9.1	9.1	9	10
		10	93	87	86	17	15	19	90	80	9.-	7.7	9.2	9.5	10	9
		11	89	93	83	22	23	16	85	90	10.-	9.-	10.0	9.3	10	10
		12	95	90	89	18	17	19	90	100	8.6	9.4	9.0	9.7	10	10
7	PA	8	92	85	83	19	17	15	100	105	5.5	6.8	.2	4.4	8	4
	(F,39)	9	84	75	73	18	16	16	100	110	4.0	.9	.6	.3	7	7
		10	75	78	83	15	13	16	120	120	4.0	0	0	0	6	5
		11	93	90	93	16	14	15	110	110	.5	.4	0	.1	5	6
		12	88	92	87	19	15	15	105	110	0	0	0	0	6	5
9	PA	8	44	48	43	18	18	16	110	95	3.0	2.7	4.3	4.4	8	7
	(M,17)	9	40	40	39	16	14	12	110	100	2.9	2.9	4.3	4.1	8	9
		10	43	46	43	19	15	16	100	110	3.2	3.2	3.8	3.9	9	7
		11	48	46	42	16	16	15	110	120	3.4	3.8	4.2	3.8	7	9
		12	37	39	39	17	14	14	100	90	3.5	3.3	3.9	3.8	9	8

PLACEBO TREATMENT, DAILY MEASURES

S#	Day	PULSE Pre	PULSE Mid	PULSE Post	RESPIRATION Pre	RESPIRATION Mid	RESPIRATION Post	SYSTOLIC BLOOD PRESSURE Pre	SYSTOLIC BLOOD PRESSURE Post	MT Pre	MT Post	A Pre	A Post	EPFAB Pre	EPFAB Post
2	8	82	81	79	19	21	19	90	100	.9	.1	1.7	.2	2	2
	9	80	78	80	20	17	17	100	90	.1	.2	.1	.2	3	3
	10	88	84	86	21	20	18	100	100	.1	.1	.2	.1	1	3
	11	89	74	85	21	17	18	90	90	.1	.2	.1	.1	2	1
	12	77	75	72	19	17	17	70	100	.2	.2	.2	.3	2	1
4	8	80	76	74	20	20	17	90	90	9.8	9.2	9.8	9.2	8	7
	9	72	69	75	20	20	18	90	90	8.7	8.8	8.6	8.5	8	8
	10	73	78	68	20	19	19	110	105	8.8	8.5	9.-	8.5	8	7
	11	85	70	70	15	17	18	90	100	9.8	8.9	9.8	8.9	7	8
	12	75	81	72	19	20	19	105	100	9.6	9.-	9.7	8.9	7	9
6	8	70	60	64	22	18	20	90	90	3.6	1.5	.2	.2	6	8
	9	78	74	72	23	21	20	100	100	1.5	2.2	.3	.4	9	5
	10	72	67	68	20	21	20	110	100	.4	.4	.5	.6	5	8
	11	74	77	78	19	20	21	110	90	.5	.2	.5	.3	8	5
	12	79	78	73	23	22	23	95	90	.4	.3	.4	.3	4	6
8	8	79	74	70	20	17	18	110	110	7.1	7.1	8.9	6.5	7	8
	9	74	80	83	20	18	19	90	105	3.1	2.6	3.3	2.9	7	8
	10	75	78	77	20	19	22	100	110	1.3	1.-	2.2	.9	6	8
	11	81	81	76	19	19	18	105	90	6.1	1.6	5.5	1.3	9	7
	12	81	71	76	20	18	21	105	105	2.-	.2	1.6	.3	8	8
10	8	73	67	62	12	14	22	100	80	.3	.3	.3	.1	3	4
	9	66	62	63	30	27	26	105	90	.2	.2	.1	.2	3	2
	10	59	55	61	25	24	17	100	95	1.7	1.8	1.8	1.8	3	4
	11	67	64	64	29	17	23	105	90	.6	.1	.7	0	3	4
	12	76	73	75	17	15	16	105	95	4.9	.4	2.4	.2	4	1

(continued)

S#	Day	PULSE			RESPIRATION			SYSTOLIC BLOOD PRESSURE		MT		A		EPFAB	
		Pre	Mid	Post	Pre	Mid	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
1	1	65	64	64	13	12	13	80	90	2.3	0	4.1	0	10	6
	2	71	63	68	14	13	13	80	80	.1	0	1.9	0	9	5
	3	65	58	69	14	11	12	80	70	0	0	0	0	5	8
	4	62	76	78	14	13	13	80	80	0	0	0	0	4	5
	5	80	74	80	14	13	11	100	100	0	0	0	0	5	9
3	1	64	61	60	17	20	21	100	105	1.2	.2	.7	.2	6	6
	2	65	62	62	14	18	18	105	100	3.4	2.7	4.4	3.5	6	4
	3	63	60	61	15	20	24	100	95	2.3	2.2	2.5	2.2	4	7
	4	56	58	59	18	19	22	110	110	2.4	1.8	2.2	1.8	6	8
	5	73	65	66	23	23	21	110	105	1.6	1.2	1.7	.9	3	8
5	1	89	88	86	17	18	17	85	95	6.3	5.-	7.4	5.7	9	8
	2	90	88	92	17	19	18	85	100	6.-	4.4	6.8	5.6	9	10
	3	96	99	95	21	17	17	100	110	4.9	4.2	6.4	6.8	8	9
	4	104	102	104	18	18	18	95	90	5.1	4.7	7.5	6.1	8	10
	5	99	95	94	18	19	17	90	100	5.2	4.3	7.-	7.1	10	10
7	1	92	85	86	16	16	15	110	125	2.9	4.6	8.7	3.9	8	6
	2	84	81	79	16	14	16	110	120	2.-	5.1	2.4	5.2	9	7
	3	93	94	89	16	18	18	100	105	.4	5.4	.7	.3	5	8
	4	91	78	83	17	13	16	110	110	0	.4	.1	0	6	5
	5	93	82	84	18	16	15	115	125	2.4	.4	.8	.3	6	7
9	1	48	58	48	18	17	18	110	90	2.2	2.5	4.3	3.5	5	4
	2	46	52	48	17	15	17	100	100	2.4	2.7	3.6	3.9	6	7
	3	44	51	49	16	19	14	90	105	2.4	2.4	3.9	3.1	7	7
	4	42	43	43	19	17	21	90	75	2.2	2.7	3.6	3.7	8	8
	5	43	43	44	17	16	15	115	100	2.9	2.9	3.8	3.5	8	8

WEEKLY MEASURES

S#	Day	EPI E	EPI N	EPI L	STAI ST	STAI TR	CC A	CC B	CC C	CC D	PCA
2	1 b	13	18	2	58	46	9				3.16
	8 a	12	9	3	23	42	8				.33
	15 b	14	14	1	20	41	5				.33
	wk. a	13	16	1	31	47	0				0
	mo. b	10	18	1	46	48	0				.16
4	1	4	19	0	65	73	9	10			2.50
	8	7	24	1	76	74	9	10			2.66
	15	2	20	0	78	79	10	10			1.50
	wk.	8	22	1	73	76	9	9			1.16
	mo.	4	19	0	77	76	10	10			2.50
6	1	10	19	7	55	58	5				1.50
	8	13	21	4	42	59	0				1.0
	15	10	18	4	36	50	0				.16
	wk.	10	17	4	43	59	3				0
	mo.	10	20	4	36	46	0				0
8	1	7	19	1	65	61	8	5			3.83
	8	11	22	2	67	67	10	7	8		1.83
	15	4	20	0	65	64	8	4	8		.50
	wk.	10	20	2	54	60	3	3	3		.50
	mo.	4	19	0	42	55	3	1	1		.50
10	1	6	16	0	33	61	2	0	10	9	2.83
	8	3	18	4	36	57	3	1	9	6	1.16
	15	4	19	0	32	58	1	1	10	3	1.50
	wk.	3	15	4	24	58	0	0	0	0	.83
	mo.	8	18	0	26	53	1	1	5	1	.33

(continued)

			EPI			STAI		CC				PCA
S#	Day		E	N	L	ST	TR	A	B	C	D	
1	1	a	9	19	2	32	41	8	8	8		1.50
	8	b	9	16	0	33	54	4	4	8		1.0
	15	a	11	16	1	29	39	4	3	1		.50
	wk.	b	13	19	0	32	47	8	8	8		1.16
	mo.	a	10	19	1	32	42	6	8	8		1.66
3	1		11	16	3	38	44	10	10	4		1.66
	8		14	19	1	28	33	0	2	0		0
	15		18	3	2	20	20	0	0	0		0
	wk.		20	4	2	23	34	0	0	0		.16
	mo.		19	4	1	20	34	0	0	0		.16
5	1		3	15	3	75	63	10				2.16
	8		4	14	0	77	68	10				2.16
	15		5	17	1	76	69	9				3.50
	wk.		2	19	0	76	66	9				2.66
	mo.		3	19	0	50	57	6				3.16
7	1		16	21	1	64	55	8	10	10		3.16
	8		13	19	2	42	50	4	8	10		1.0
	15		15	21	1	55	49	2	3	10		.83
	wk.		13	14	1	41	52	2	0	2		1.0
	mo.		15	20	1	29	52	1	2	1		1.83
9	1		6	18	3	58	53	8	7	10		3.83
	8		6	16	3	77	71	8	10	7		3.66
	15		8	19	0	59	70	9	10	8		3.33
	wk.		6	18	3	76	74	7	8	10		3.33
	mo.		7	21	2	52	67	6	5	9		2.83