THE USE OF FLOTATION REST IN THE TREATMENT OF PERSISTENT PSYCHOPHYSIOLOGICAL INSOMNIA

by

ELIZABETH JEAN BALLARD

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Department of Psychology

The University of British Columbia
1956 Main Mall
Vancouver, Canada
V6T 1Y3

Date April 17, 1989.

Abstract

Although relaxation techniques have been successful in treating sleep onset insomnia, research evidence of efficacy has traditionally focused on self-reported improvement measured during the course of treatment. Few studies have collected objective measures or long-term followup data. The present study used subjective and objective measures in evaluating the long-term effectiveness of the flotation form of the Restricted Environmental Stimulation Technique (REST) as a treatment for persistent, psychophysiological sleep onset insomnia. It was expected that treatment would reduce both measures of sleep latency over the long term.

Flotation REST is a behavioural intervention that involves the use of a flotation tank. The tank is similar to a large, enclosed (dark and quiet) bathtub. A door on the side allows easy entry and exit. The floater lies suspended in a supine position with the face and ventral portion of the body out of the aqueous solution of $MgSO_4$. The solution has a specific gravity of 1.27 and a temperature of 34.2^{0}C.

Thirty-six volunteers (20 women and 16 men) with psychophysiological insomnia were randomly assigned to one of four conditions: flotation REST only, flotation REST with autogenic relaxation, autogenic relaxation only, or delayed treatment control. Each treatment group received four 2-hour treatment sessions within a fourteen-day period. During each session, 45 minutes were spent in treatment. The remainder of the two hours was devoted to tasks related to the study.

Sleep latency was measured before treatment and one, four, and twelve weeks after treatment. Self-report latency was recorded at home in a daily sleep log. Objective latency data were collected via the Somtrak sleep

assessment device (SAD machine), which was used at home. The SAD machine consists of a tone generator and timing device connected to a cassette recorder, all fitted inside a briefcase. During use, a one-second tone goes off every ten minutes, and simultaneously the tape begins to record for ten seconds. If the person hears the tone, a response of "I'm awake" is given and recorded. Paper and pencil measures of affect and arousal were collected during treatment and whenever sleep latency was measured.

The results indicated that flotation REST (either by itself, or in combination with autogenic relaxation) was effective in reducing both subjective and objective sleep latency over the long term. Both flotation REST conditions were equally effective as the autogenic relaxation condition in reducing sleep latency after twelve weeks.

Reductions in tension were correlated with reductions in subjective sleep latency and reductions in anger were correlated with reductions in objective sleep latency, all over the long term.

People who reached their peak activity level late in the day took objectively longer to get to sleep than people whose peak activity level occurred earlier.

It was concluded that flotation REST (on its own and in combination with autogenic relaxation) improved both subjective and objective sleep latency up to twelve weeks after treatment.

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Introduction

Overview

This study deals with the evaluation of a behavioural technique, flotation REST, as a treatment for persistent psychophysiological insomnia. After an examination of differences between good sleepers and insomniacs, a description of the subtypes of insomnia (particularly persistent psychophysiological insomnia), and a discussion of current treatment techniques, a case will be made for the potential usefulness of flotation REST in treating this problem. The chapter will end with a summary of the major questions that guided this research.

Differences Between Good Sleepers and Insomniacs

Most people obtain enough sleep to allow them to feel alert and enthusiastic all day. But for somewhere between 33% and 42% of adults, or approximately one out of every three, this is not the case (Bixler, Kales, Soldatos, Kales & Healy, 1979; Mellinger, Balter, & Uhlenhuth, 1985). For these people, getting to sleep or staying asleep is a major difficulty and their daytime functioning suffers. Insomnia, which can be described as the chronic inability to obtain adequate sleep due to delayed sleep onset, to multiple night arousals and/or to early morning awakening, is one of the more common sleep complaints reported to physicians and assessed at sleep disorders centres (Kales & Kales, 1984; Kales, Soldatos, & Kales, 1987). What distinguishes good sleepers from insomniacs? There are at least three dimensions along which good and poor sleepers can differ: physiological, cognitive, and psychological. Empirical research efforts have attempted to document reliable differences; however, they have not been uniformly successful.

Physiological differences between good and poor sleepers. Insomniacs differ consistently from good sleepers on a number of objectively measured variables. For example, insomniacs take longer to fall asleep, get less sleep overall and spend more time awake after their initial sleep onset than do normal sleepers (e.g., Coates, Killen, George, Marchini, Silverman, Hamilton et al., 1982; Monroe, 1967; Zorick, Kribbs, Roehrs, & Roth, 1984). However, according to subjective data, poor sleepers show a tendency to overestimate their latency to sleep onset and to underestimate their total sleep time and their number of night awakenings (Bixler, Kales, Leo, & Slye, 1973; Borkovec & Weerts, 1976; Carskadon, Dement, Mitler, Guilleminault, Zarcone, & Spiegel, 1976). The discrepancy between subjective estimations and objective measurements of sleep in insomniacs is not discouraging given that there are verified differences between insomniacs and good sleepers on sleep latency and total sleep time. Also, as Van Oot, Lane and Borkovec (1983) suggest, the discrepancy may reflect a confounding of the type of insomniacs being examined. If sleepers who have subjective insomnia only (that is, although the patient reports disturbed sleep, there is no objective evidence of disruption) participated in the studies, their scores would contribute to the disparity between subjective and objective measures of sleep performance. Also, insomniacs experience a great deal of variability in their sleep performance from night to night (Carskadon, Mitler, & Dement, 1974; Coates, et al., 1982; Roth, Kramer, & Lutz, 1976; Williams, Hursch, & Karacan, 1972). This variability may lead to a generalized perception of poor sleep, so that on the very few nights when objective measures of sleep are taken for research purposes, sleep performance will be subjectively evaluated as poor, because

it is always seen that way, although the corresponding objective measure that night may show normal sleep.

In a landmark study that gave rise to the hyperphysiological arousal hypothesis of insomnia, Monroe (1967) found that poor sleepers had elevated autonomic activity prior to and during sleep, on a number of physiological variables. Further studies compared autonomic arousal in insomniacs and good sleepers but the findings are inconsistent. For example, Monroe's (1967) work showed, among other signs of elevated autonomic activity, a higher rectal temperature in poor sleepers than in good ones. But a few years later, Johns, Gay, Masterton, and Bruce (1971) failed to replicate this finding. Johns and his colleagues did, however, report higher levels of adrenocortical activity in poor sleepers, reflecting increased psychological stress. These results were not supported later when both Frankel, Buchbinder, Coursey, and Snyder in 1973 and Adam, Tomeny, and Oswald in 1986, failed to find differences between good and poor sleepers in any of the cortisol measures. However, Adam and her colleagues did find significantly higher temperatures in her insomniacs than in the good sleepers, thus supporting Monroe's (1967) earlier claims. But this group disputed the claims by Monroe (1967) and Frankel, Coursey, Buchbinder, and Snyder (1976) that insomniacs have decreased amounts of REM or of slow wave sleep. Adam et al. (1986) found no differences in either type of sleep among insomniacs and good sleepers.

In treatment studies where physiological arousal has been decreased during therapy, no correlations with improvements in sleep performance have been found (Borkovec & Fowles, 1973; Borkovec, Grayson, O'Brien, & Weerts, 1979; Coursey, Frankel, Gaarder, & Mott, 1980; Freedman & Papsdorf, 1976; Hauri, 1981; Haynes, Sides, & Lockwood, 1977; Lick &

Heffler, 1977; Nicassio, Boylan, & McCabe, 1982). As yet, the exact nature of the relation between increased physiological arousal and sleep disruption remains unclear.

The question arises, does heightened physiological arousal contribute to either the development or maintenance of insomnia in some people, or is there some other mechanism underlying the problem? A possible alternative has been suggested by the work of Jordan, Hauri, and Phelps (1976). They proposed that the sensorimotor rhythm (SMR) belongs to a sleep-specific physiological system which may be associated with certain types of insomnia. The SMR is a 12- to 14-cps brainwave rhythm similar to sleep spindles commonly found in non-rapid eye movement sleep and it originates in the sensorimotor cortex. Humans have strong sleep spindles (or SMR), but their waking rhythm is low. Jordan et al. (1976) found significantly less SMR in insomniacs during wakefulness than in good sleepers. Preliminary research by Feinstein, Sterman, and Macdonald (1974) suggested that sleep may be improved if operant conditioning is used to train insomniacs to increase their waking SMR. Hauri (1981) further demonstrated, and Hauri, Percy, Hellekson, Hartmann, and Russ (1982) later replicated the finding, that SMR biofeedback led to therapeutic improvements in sleep, but only for low anxious, psychophysiological insomniacs. (For a description of psychophysiological insomnia see pp. 10 - 11). It was ineffective for high anxious insomniacs. Hauri (1986) suggests that for the subset of poor sleepers who are non-anxious, persistent psychophysiological insomniacs, who develop conditioned reinforcers of disturbed sleep during stress, a neurological problem with sensorimotor rhythm may be at the root of the insomnia. This means that for at least one

subset of insomniacs heightened physiological arousal is not the underlying mechanism.

Physiological arousal per se may not be the mechanism underlying insomnia. The findings of Adam and her colleagues (1986) indicate that physiological arousal mediated by anxiety may be the mechanism. These authors suggest that because anxiety has been shown to raise metabolic rates (Landis, 1925, cited in Adam et al., 1986) and body temperature (Dabbs & Moorer, 1975, cited in Adam et al., 1986) perhaps the increased anxiety found in poor sleepers (e.g., Coursey et al., 1975) may cause the higher body temperatures that they found in their insomniacs. To the extent that temperature and metabolic rates influence sleep performance, physiological arousal mediated by psychological variables related to anxiety may indeed be an indirect factor in initiating or maintaining insomnia.

Cognitive arousal in the insomniac. In addition to some physiological arousal, the insomniac may experience elevated cognitive arousal. Borkovec (1982) summarized evidence pointing out the importance of presleep mental activity as a factor in sleep onset insomnia. Findings from a number of studies showed that cognitive arousal, in the form of obsessive thoughts in bed (Shealy, 1979), worries in general (Roth et al., 1976), and worries about sleep in particular (Ascher & Efran, 1978; Kales et al., 1976; Van Egeren, Haynes, Franzea, & Hamilton, 1983) are common to many insomniacs.

Mitchell (1979) found that reductions in presleep tension and presleep disruptive cognitions were associated with significant improvements in subjective sleep onset latency. These results support earlier findings that cognitive control procedures which reduce presleep tension produce improvements in self-reported sleep onset latency over and

above the improvements due to progressive muscle relaxation (Mitchell & White, 1977). Another study (Van Egeren et al., 1983) found evidence that negative presleep cognitions concerning sleep and physical sensations (e.g., thoughts about not falling asleep, noises in bedroom) were significantly associated with subjective sleep onset latency.

Lichstein and Rosenthal (1980) showed that insomniacs themselves attributed their sleep problem more frequently to cognitive than somatic arousal. This suggests that insomniacs have a particular style of cognitive activity. This style may lead them to have increased central nervous system arousal, which in turn delays sleep onset.

Psychological differences between good and poor sleepers. In trying to understand the causes for poor sleep, researchers have examined psychological variables to see if there are differences between insomniacs and good sleepers. The Minnesota Multiphasic Personality Inventory (MMPI) has been used widely by sleep researchers to measure psychopathology in insomniacs. For the most part, insomniacs and poor sleepers tend to show a slight elevation on the depression scale, and a profile indicating psychological stress (e.g., Carskadon et al., 1976; Coursey, Buchsbaum, & Frankel, 1975; Johns et al., 1971; Kales, Caldwell, Preston, Healy, & Kales, 1976; Kales, Kales, & Soldatos, 1982; Monroe, 1967; Roth et al., 1976). Although MMPI abnormalities are frequently associated with insomnia patients, at least one group of researchers (Zorick et al. 1984), has suggested that these abnormalities may be the result of having a chronic condition, rather than the cause of it.

Insomniacs, compared to good sleepers, tend to score higher on measures of anxiety (e.g., Adam et al., 1986; Coursey et al., 1975; Haynes, Woodward, Moran, & Alexander, 1974), mild depression (e.g., Coursey et

al., 1975), and medical complaints, as measured by the Cornell Medical Index (the CMI symptoms checked usually reflect tension-somatic complaints, such as tension headaches, cold hands and feet) (e.g., Hauri & Fisher, 1986; Johns et al., 1971; Monroe, 1967).

Because insomnia can result from a number of causes, some researchers have concerned themselves with trying to identify psychological variables associated with specific subtypes of insomnia. For example, Hauri and Fisher (1986) compared persistent psychophysiological insomnia patients (PPI) with both normal sleepers and insomniacs with dysthymic disorders. PPI refers to a subtype of insomnia that is not associated with medical, psychiatric, or environmental causes. Patients with dysthymic disorders suffer depression. The researchers found that the scores of PPI patients on the MMPI were very close to those of normal sleepers. The major differences were: (a) on a subset of the *Hy* (hysteria scale) scores which, when more closely examined, showed that the PPI patients checked more items associated with somatized tension than did normal sleepers, and (b) on the Welsh R scale, measuring denial and repression, with the PPI patients scoring significantly higher than the normal sleepers.

With respect to mood, as measured by the Profile of Mood States (McNair, Lorr, & Droppleman, 1971), PPI patients were more fatigued than normal sleepers, but reported no other mood differences. On scales of thrill seeking and excitement, and on susceptibility to boredom as measured by Zuckerman's Sensation Seeking Scale (Zuckerman, 1974), PPI patients were significantly lower than normals.

The conclusion reached by Hauri and Fisher (1986) was that PPI patients present a relatively normal psychological profile, except that they

are more guarded and defensive, more fatigued in the daytime, have more tension-related somatic symptoms and are less sensation-seeking than normal sleepers.

The results of the comparison of persistent psychophysiological patients with dysthymic insomniacs showed that psychophysiological patients have less psychopathology than depressed insomniacs. Specifically, the psychophysiological patients were less neurotic, less depressed, and less anxious. This study provides empirical evidence for the accuracy of the Association of Sleep Disorders Centers' (1979) proposed differential diagnosis for persistent psychophysiological insomnia. PPI and depressed patients experience similar sleep problems, but the PPI patients are free of underlying psychiatric disorders.

Taken together, these studies imply that insomniacs in general tend to be more depressed, to have more somatic complaints and to be more anxious than good sleepers. However, when compared to insomniac patients with a psychiatric condition, the persistent psychophysiological insomniac has less of a psychopathological profile than does the insomniac with an affective disorder.

In summary, what are the major differences between insomniacs and good sleepers? Insomniacs tend to take longer to get to sleep and to spend more time awake after sleep onset than normal sleepers. These differences may be due to central nervous system (CNS) arousal mediated by physiological arousal, in the form of increased body tension, reflected in part by higher temperature. Evidence of psychological differences suggest that CNS arousal may be related to heightened cognitive activity in the form of worry, and anxious or obsessive thoughts. Insomniacs are reported to be

more anxious than good sleepers and to experience more mild depression as well as being more concerned with somatic complaints.

Good sleepers differ from poor sleepers along at least three dimensions: physiological, cognitive, and psychological. What does this say about insomnia? How can it be categorized so that differential diagnoses may lead to the most effective treatment programmes?

Categorizing Insomnia

Insomnia is a symptom. In fact, insomnia can be thought of as symptomatic of a constellation of heterogeneous sleep disorders. The Diagnostic Classification of Sleep and Arousal Disorders, published by the Association of Sleep Disorders Centers (1979), devotes an entire category, called Disorders in Initiating and Maintaining Sleep (DIMS), to insomnia, and identifies nine distinct subtypes. They are: 1. Psychophysiological DIMS (transient and persistent); 2. DIMS associated with psychiatric disorders; 3. DIMS associated with use of drugs and alcohol; 4. DIMS associated with sleep-induced respiratory impairment; 5. DIMS associated with sleep-related (nocturnal) myoclonus and "restless legs"; 6. DIMS associated with other medical, toxic, and environmental conditions; 7. Childhood-onset DIMS; 8. DIMS associated with other DIMS conditions; and finally 9. No DIMS abnormality (this refers to Subjective Insomnia where the patient reports sleep difficulty but no objective evidence of it can be found). These subtypes reflect an attempt to differentiate insomnia on the basis of alleged origin.

Sleep disorders clinics see hundreds of patients yearly. About 35% of them suffer DIMS associated with psychiatric disturbances (Coleman, Roffwarg, Kennedy, Guilleminault, Cinque, Cohn, et al., 1982). Because insomnia is often viewed as a symptom of an underlying disorder,

whenever that disorder can be identified, it becomes the central focus of therapy (Van Oot et al., 1983; Zorick et al., 1984). Thus, in insomnia associated with psychiatric conditions, such as clinically diagnosed affective disorders, personality disorders, and generalized anxiety, the primary concern is treatment of the psychopathology. The treatment of the sleeping problem is secondary. In insomnia associated with sleep-induced respiratory impairments, such as sleep apnea, where the patient stops breathing for many short periods throughout the night, medical interventions for the breathing problem are required.

The second largest category of patients treated for Disorders of Initiating and Maintaining Sleep at the sleep disorders clinics is the persistent psychophysiological insomnia group, which represents almost 15% of the patients assessed, (Coleman, et al., 1982). For these patients, treatment of the sleep disorder is of primary importance.

Patients who suffer persistent psychophysiological insomnia are characterized as having chronic[1], somatized tension-anxiety and negative conditioning to their normal sleep environment (ASDC,1979). The tension and anxiety are generally experienced as "restlessness, motor tension, . . . automatic hyperactivity, apprehensive expectation, ruminative thoughts, hypervigilance, and excessive visual scanning" (ASDC, 1979, p. 24). The psychological factors contributing to the sleep disturbance are generally neither obvious nor clearly related to recent events.

Either internal or external factors can become "conditioned reinforcers" contributing to the sleeplessness of persistent

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[1] When a transient insomnia, related to other causes, such as stress, has persisted for at least three weeks after resolution of the temporary cause, it becomes chronic; however, the definition is vague in the specification of what constitutes a temporary cause and whether one can always be identified.

psychophysiological insomnia. Internal conditioned factors include the "vicious circle syndrome" where fear of not being able to fall asleep builds with each unsuccessful attempt to sleep. When one consciously makes an effort to fall asleep, the central nervous system becomes aroused, which then delays sleep onset until the arousal subsides. This factor is likely to be present in persistent psychophysiological insomnia patients who report regularly falling asleep when not trying, such as while watching television, reading, or listening to music, but who find themselves awakening completely when deliberately trying to go to sleep.

External conditioned factors may result from the association, during a temporary bout of insomnia, between sleeplessness and routines or environments normally related to going to sleep. For example, during transient insomnia related to acute stress, the bedroom, the bed or presleep routines such as bathing and undressing may become associated with the inability to sleep, so that after repeated associations the preparations for bed or the bed itself become conditioned reinforcers for sleeplessness. When the acute cause of the sleep disruption is gone, the conditioned response to the presleep rituals or to the bedroom remains, leading to persistent psychophysiological insomnia. One indication of negative conditioning is when sleep is frequently found to be better away from home than at home.

Persistent, psychophysiological insomnia is very similar to the DSM-III-R diagnosis of primary insomnia (APA, 1987). The major difference between the two is the absence of a negative conditioning component in primary insomnia.

To summarize, insomnia is often symptomatic of an underlying disorder. According to one nosology, there are at least nine subclasses of insomnia. These subclasses are characterized by the presumed etiology of

the sleep disruption. Other than Disorders of Initiating and Maintaining Sleep associated with psychiatric conditions, the largest single type of insomnia treated at Sleep Disorders Centres is persistent psychophysiological insomnia. Although the true causes of the various DIMS subtypes are still unclear, identification of underlying pathologies assists in treatment decisions. Sleep disorders professionals consider treatment of the underlying condition of chief concern when faced with insomnia associated with psychiatric or medical conditions, or sleep-related physiological abnormalities. For persistent psychophysiological insomnia the primary focus of treatment is the insomnia itself.

Treatments for Disorders of Initiating and Maintaining Sleep

In deciding what treatment to use in the symptomatic relief of insomnia, professionals base their judgments on beliefs about the nature of insomnia. Those who think that the heightened CNS activity is due to physiological hyperarousal will focus on treatments that reduce autonomic arousal. Others who give more weight to a cognitive explanation for the increased CNS activity will favour treatments aimed at reducing disruptive, presleep cognitive arousal. Many professionals will, of course, structure their therapy so as to incorporate both somatic and cognitive treatment components.

Differential diagnoses concerning which subtype of insomnia that the patient suffers have greatly facilitated treatment. In cases where medical or psychiatric conditions underlie the sleep disorder, treatment for these conditions frequently leads to improved sleep. In these cases, symptomatic treatment of the insomnia problem may or may not form part of the complete therapeutic package. However, for at least two of the DIMS categories, persistent psychophysiological insomnia and no DIMS

abnormality (also called Subjective Insomnia), no obvious medical or psychiatric condition is present and treatment of the symptom is of most importance.

Treatment for insomnia falls into two major categories: pharmaceutical and behavioural. Traditionally, the greater emphasis has been on drug treatment. However, with growing concern over the risks and long-term ineffectiveness of drugs in treating insomnia, behavioural therapies have become a more accepted alternative.

Drug therapy. Drug treatments have shown some success in alleviating symptoms of insomnia. Hypnotics can decrease sleep latency, decrease the number of night awakenings, and increase the total sleep time in insomnia patients. However, they have also caused serious problems of their own. Studies of drug-induced sleep have raised issues of dependence, tolerance and withdrawal, rebound insomnia and anxiety, of abnormalities in nighttime sleep patterns, and more recently have focused on their impact on daytime performance, of a number of activities, ranging from monotonous tasks to driving a car.

The most common class of nonbarbiturate hypnotic prescribed is benzodiazepine. Long term and even short term use of these hypnotics can lead to the development of drug tolerance, which means that the initial dosage no longer produces the same effect on sleep (File, 1985; Kales & Kales, 1973; Lader, 1983; Smith & Wesson, 1983). This situation can tempt the insomniac to increase the dosage in order to gain relief. This path leads to dependence, because should the patient stop taking the medication, the immediate result is usually a worsening in sleep. A withdrawal syndrome characterized by decreased sleep and increased daytime anxiety can be seen after chronic (more than two weeks) use, even when the drug is withdrawn

gradually. This rebound insomnia, therefore, often results in people continuing to rely on their medication (Marks, 1983). Ribordy and Denney (1977) suggest that attributional effects support the pharmacokinetic effects of drug withdrawal, making it more difficult to terminate drug use. The insomniac is likely to attribute what little sleep has been achieved to the effects of the medication, not to his or her own ability to sleep (e.g., Storms & Nisbett, 1970). Thus when trying to withdraw from drug use, the decrement in sleep performance associated with rebound insomnia effects confirms the belief that it was the drug that had led to sleep. It has been proposed that dependence on soporifics may be caused by a combination of the rebound insomnia and the attributional effects (e.g., Freemon, 1972; Kales & Kales, 1973; Oswald, 1968).

With some short half-life benzodiazepine hypnotics, such as triazolam, early morning insomnia (that is, awakening before the night is over) daytime anxiety, memory impairment and even amnesia have occurred -- sometimes even with normal dosages (Bixler, Scharf, Soldatos, Mitsky, & Kales, 1979; Kales, Soldatos, Bixler, & Kales, 1983a, 1983b; Lister, 1985; Scharf, Khosla, Brocker, & Goff, 1984). Although short courses of drug therapy (lasting up to six weeks) have been recommended, chronic insomniacs usually suffer from a return of their sleep difficulties following drug discontinuation (Gillin & Mendelson, 1981; Smith & Wesson, 1983).

Hypnotics alter the nature of nighttime sleep. Benzodiazepines have been shown to decrease the amount of slow wave sleep (that is, Stages 3 and 4 sleep) and of REM (rapid eye movement sleep) even when taken as directed (Roth, Zorick, Wittig, & Roehrs, 1982).

Hypnotics have also been associated with a number of detrimental effects on human performance. Roehrs, Kribbs, Zorick, and Roth (1986), for

example, discuss the residual effects of repeated administrations of benzodiazepines on simple and complex visual reaction time tasks, on a divided-attention task, on digit symbol substitution tests and on an auditory vigilance task. They found flurazepam significantly increased reaction times and reduced symbol substitution accuracy early in on in the course of drug therapy. However, in the late phase, performance after the drug did not differ from performance after placebo. Thus the detrimental effects of some benzodiazepines vary over the course of treatment.

Some sleeping medications have been tied to impaired driving performance. O'Hanlon and Volkerts (1986) showed that three drugs, secobarbital 200 mg, flurazepam 30 mg and loprazolam 2 mg, each had a significant detrimental effect on driving performance. This effect was said to be greater than the effect of a blood alcohol content of 1.0 mg/ml, which is accepted as an intoxicating level. These authors claim that a 1.0 mg/ml blood alcohol content is associated with approximately a seven times greater risk of involvement in a fatal traffic accident (e.g., Borkenstein, Crowther, Shumate, Zeil, & Zylman, 1964; O'Hanlon, 1980, both cited in O'Hanlon & Volkerts, 1986). The effect of hypnotics on driving demonstrates that the medicated insomniac is not the only one who is at risk from drug therapy.

A recent survey (Mellinger et al., 1985) reported that 3.1% of the respondents said that they had used over-the-counter (OTC) drugs in the previous year to help promote sleep. Use of OTC, prescription drugs, or both was reported by 7.1%. Thus some people are using both prescription and nonprescription drugs to treat their sleep problem.

The same survey (Mellinger et al., 1985) indicates a decline in the prescribing of hypnotics. The annual number of prescriptions for hypnotic

drugs filled in American drug stores decreased from 42 million in 1972, to 21 million in 1982. The prevalence of use of prescription hypnotics among adults aged 18 to 74 years also decreased, going down from 3.5% in 1970 to 2.4% in 1979 (Mellinger et al., 1985). Even though presciption and use of hypnotics is decreasing, drug therapy remains a common treatment for insomnia.

There has been a change in both the class of drug being prescribed and the rate of prescribing. While prescriptions for nonbarbiturate hypnotics are increasing, they are not increasing as quickly as prescribing of short-acting barbiturates is declining (Mellinger et al., 1985). This also indicates a general trend towards decreased reliance on the pharmaceutical treatment of insomnia.

Because of their negative side-effects, hypnotics have restricted applicability. They are best suited for brief interventions in the treatment of transient, situational forms of insomnia, such as insomnia related to jet lag, acute stress, or environmental change (Roth et al., 1982). Hypnotics are not particularly well suited to the treatment of chronic insomnias.

Behavioural treatments. Based on the physiological, cognitive, and psychological variables thought to differentiate the normal sleeper and the insomniac, behavioural treatments aimed at improving the sleep performance of insomniacs have been developed and tested.

Behavioural interventions have focused in one way or another on ameliorating central nervous system arousal. The techniques most commonly used to treat insomnia include relaxation training, biofeedback, stimulus control, and paradoxical intention. More recently sleep restriction therapy has been tried, but very little research on its effectiveness is available. The appropriateness of these interventions for treating

insomnias associated with psychiatric conditions or with some medical conditions is uncertain. Behavioural treatment for the sleep disruption could be beneficial as an adjunct to treatment given for the underlying psychiatric or medical conditions. However, it would seem that the behavioural techniques are best suited to the treatment of persistent psychophysiological insomnia and of subjective insomnia (i.e., the no DIMS abnormality subtype).

Many of the studies evaluating the effectiveness of the behavioural techniques were performed prior to the publication of the ASDC (1979) nosology of insomnias. Thus, the people treated in these studies comprised a mixed group of insomniacs. Possibly the two subtypes of insomnia most amenable to behavioural treatments are psychophysiological and subjective insomnia. Although sleep onset insomnia was the most frequently studied, the severity of the problem varied from study to study. Further, the samples drawn for experimentation came more often from a college population than from the community at large, thus limiting the generalizability of the findings. The results of the earlier studies, therefore, must be viewed with caution, because the effectiveness of a treatment may interact with the severity of the condition (Carr-Kaffashan & Woolfolk, 1979) and the specific subtype of insomnia (Hauri & Fisher, 1986), and may be of limited generalizability.

Relaxation therapies. Briefly, what are these treatment procedures? A factor common to most of the behavioural therapies is anxiety reduction. Relaxation therapies cover a range of techniques with focus on somatic and cognitive components. The most commonly used relaxation technique is progressive muscle relaxation originated by Jacobson (1938). However, most studies use the abbreviated Jacobson technique suggested by

Bernstein and Borkovec (1973). Progressive muscle relaxation involves the systematic tensing and releasing of various muscle groups throughout the body. Borkovec and his colleagues have conducted a series of studies to evaluate the crucial components in this treatment (e.g., Borkovec & Hennings,1978; Borkovec, Kaloupek, & Slama, 1975; Borkovec & Weerts, 1976; Steinmark & Borkovec, 1974). Borkovec (1982) concludes that the active ingredients of progressive muscle relaxation involve muscle-tension release and focused attention on internal sensations.

Another relaxation therapy used to treat insomnia is autogenic training. It is a cognitively induced relaxation procedure developed by Schultz and Luthe (1959). The object is to bring about a self-induced condition of physical and mental calmness, by reducing arousal created by mental activity that is incompatible with sleep. This calm is induced by auto-suggestions of sensations of warmth and heaviness in the arms and legs, hands and feet. According to Luthe (1963) the three main psychophysiological principles on which autogenic training is based are: "(a) reduction of exteroceptive and proprioceptive afferent stimulation; (b) mental repetition of psychophysiologically adapted verbal formulas; and (c) mental activity conceived as 'passive concentration'." (p. 176). In the traditional form, the training involves six short verbal "formulas" including feelings of heaviness in the limbs, abdominal and peripheral warmth, cardiac and respiratory regularity and coolness of the forehead (Pikoff, 1984). Kahn, Baker, and Weiss (1968) were the first to use autogenic training to treat insomnia. Although there were methodological inadequacies, such as the use of a single group design and a lack of specific sleep latency measures, the authors found that a self-reported estimate of improvement after treatment showed a majority felt that their sleep had

improved significantly. Nicassio and Bootzin (1974) found autogenic relaxation training to be as effective in treating insomnia as progressive relaxation. However, both self-reported and objective sleep improvement were found for only some of the insomniacs receiving autogenic training in the 1980 study by Coursey and his colleagues.

Biofeedback relaxation treatments consist of a set of techniques that apply learning procedures to allow patients to monitor and change physiological activities such as heart rate, blood pressure and muscle tension. Two types of biofeedback have been used to treat insomnia: electromyographic (EMG) and somatasensory (SMR, sensorimotor rhythm). However, SMR training is not a relaxation therapy, nor is the sensory motor rhythm associated with sensations of relaxation.

The basic procedure for eliciting a relaxation response involves several steps. First, electrodes are placed on the skin (usually on the forehead or jaw) to measure the level of tension in the striate muscles beneath the electrodes. Next, the electrical activity in the muscle group is processed by the biofeedback apparatus and converted into auditory or visual signals. The participant receives the signals and then alters his or her behaviour to produce a signal reflecting reduced muscle tension and presumably reduced arousal.

Feedback of electroencephalographic (EEG) activity from the sensorimotor cortex is used in SMR feedback. By increasing SMR activity (recall that SMR refers to a 12- to 14-cps brainwave pattern which when found during sleep is referred to as a sleep spindle) during wakefulness, sleep was improved in non-anxious insomniacs (Hauri, 1981; Hauri et al., 1982; Hauri & Fisher, 1986).

Active components in relaxation and biofeedback treatments. Several researchers have suggested that the success of not only progressive muscle relaxation, but also of other forms of relaxation therapies such as autogenic training, biofeedback, and meditation may involve (a) the interruption or termination of cognitive activity that is incompatible with sleep, and (b) the redirecting of attention away from intrusive, sleep-incompatible cognitions, (e.g., Borkovec, 1982; Woolfolk, Carr-Kaffashan & McNulty, 1976). Focusing on monotonous or repetitive stimuli has been associated with reduced physiological arousal (Woolfolk, 1975) and with sleep onset (e.g., Bohlin, 1971; 1973). Relaxation exercises and biofeedback training have two positive effects on the cognitively aroused insomniac. First, by focusing on one of the techniques (such as, tensing and releasing muscle groups; mentally repeating autogenic formulae; or concentrating on recreating behaviour that brought about correct signals during biofeedback training) attention is diverted from the sleep-incompatible cognitions presumed to be disrupting sleep. Second, the monotonous stimulation of the repetitive format of these behavioural exercises is itself sleep-inducing.

In reviews of behavioural treatments of insomnia (Borkovec, 1982; Van Oot et al., 1983), relaxation training was found consistently to show outcome results significantly superior to placebo and no-treatment conditions. For relaxation-treated insomniacs, self-reported sleep latencies averaged reductions of 45%, while objective sleep measures (EEG-defined sleep) showed improvements ranging from 59% to 71%.

The mechanism whereby relaxation therapy reduces sleep latency is not entirely clear. While the initial use of relaxation therapy was aimed at reducing the autonomic hyperarousal presumed to be preventing sleep, several studies have found no relation between reduced physiological

activity during treatment and outcome improvement in either self-report diaries or objective measures of sleep (e.g., Borkovec, Grayson, O'Brien, & Weerts, 1979; Coursey et al., 1980; Hauri, 1981). Borkovec's (1982) cognitive-hyperarousal theory of insomnia is, perhaps, closer to the mark than the alternative physiological arousal theory. The suggestion that the active ingredient within the relaxation procedures is the learning to focus attention on pleasant, monotonous, internal sensations that distract insomniacs from worrisome, distressing, and cognitively arousing thoughts and images has intuitive appeal and empirical support (e.g., Borkovec & Henning, 1978; Mitchell & White, 1977).

Stimulus control treatment. Another behavioural approach, the stimulus control treatment, addresses the negative conditioning component of insomnia. The bed is assumed to have lost its power to act as a discriminant stimulus for sleep. Based on a case study, Bootzin (1972) suggested that, for insomniacs, sleep-incompatible behaviours become associated with the bed, bedroom or even presleep rituals. Thus instead of becoming calmer and sleepier when getting ready for bed or getting into bed, the insomniac becomes more alert. Treatment for this conditioned response involves systematically changing the person's sleep-incompatible response to the bed and associated stimuli. The idea is to re-establish the bed as a discriminative stimulus for sleep. Instructions for "reconditioning" the bed include: (a) go to bed only when sleepy; (b) get out of bed if not asleep in 10 minutes, go to another room and do something else, return to bed only when sleepy, follow this routine all night if necessary; (c) get out of bed at the same time each morning, regardless of the amount of sleep obtained the night before; (d) do not take naps during the day.

Research has demonstrated the effectiveness of stimulus control in treating sleep onset insomnia, but it has relied solely on self-reported measures of sleep latencies (e.g., Haynes, Price, & Simons, 1975; Lacks, Bertelson, Gans, & Kunkel, 1983; Ladouceur & Gros-Louis, 1986; Turner & Ascher, 1979a, 1979b, 1982; Zwart & Lisman, 1979). One study combining the stimulus control technique with a passive relaxation exercise (Shealy, 1979) found stimulus control to augment the effects of passive relaxation, bringing about improved sleep latency more rapidly than relaxation alone. Although stimulus control has consistently been significantly better than no treatment or placebo treatments, it is not reliably different from other behavioural treatments such as progressive relaxation and paradoxical intention. It remains to be seen whether stimulus control can produce objectively measured improvements that are better than those found with relaxation treatments.

In several studies related to the stimulus control paradigm, questions concerning its construct validity have arisen. Contrary to a stimulus control paradigm prediction, the frequencies of sleep-incompatible behaviours linked to the bed and bedroom did not differ between insomniacs and normal sleepers (Haynes, Adams, West, Kamens, & Safranek, 1982; Haynes, Follingstad, & McGowan, 1974). Haynes et al. (1982) also found that neither individual sleep-incompatible behaviours (such as reading, studying, smoking) nor the total duration spent doing a number of sleep-incompatible behaviours were reliably related to self-reported sleep onset latency. In a study looking for the active components in the stimulus control procedure, Zwart and Lisman (1979) demonstrated that contrary to predictions based on the stimulus control paradigm, improvement was not due to the bed becoming a discriminant stimulus for

sleep. This conclusion resulted from the finding that insomniacs who were not asleep within 10 minutes of turning out the lights and who had been instructed to remain in bed, sit up and engage in some activity (e.g., reading or watching television) improved their sleep onset latency to the same degree as insomniacs following the stimulus control instructions of getting out of bed if not asleep within 10 minutes. The authors suggest that stimulus control may be a misnomer for this procedure. The simplest explanation for the reported success of the stimulus control procedure, they feel, is not that the bed has regained its power as a discriminant stimulus for sleep, but rather that the procedure calls for a contingent disruption of the sleep-incompatible behaviours or cognitions that occur prior to sleep onset.

Paradoxical intention. This is a behavioural technique with a twist. Its procedure requires insomniacs to attempt to stay awake while in bed, rather than trying so hard to fall asleep. This cognitive self-control strategy was designed to overcome performance anxiety thought to be interfering with the insomniac's ability to go to sleep. The idea is to break the worry habit by focusing on doing the feared behaviour: not falling asleep.

Experimental support for the effectiveness of this technique is weak. Three studies, using self-report data alone, have shown the efficacy of paradoxical intention. Reports from several other studies show conflicting results. For example, in one study, paradoxical intention was significantly better than stimulus control, sleep information, and no treatment (Ladouceur & Gros-Louis, 1986), yet in two others it was equally effective as stimulus control and progressive relaxation, and more effective than placebo and no treatment (Turner & Ascher, 1979a, 1979b). Espie and Lindsay (1985) reported findings based on a total of six insomniacs. Three

of them rapidly improved their sleep onset latency using the paradoxical intention procedure, but the other three got worse.

In a comparison among paradoxical intention, placebo and no treatment control groups, Ascher and Turner (1979) reported a 53.5% sleep improvement for the paradoxical intention group, which was a significantly better result than for either of the control groups. Turner and Ascher (1982) tested therapist effects in the use of paradoxical intention, stimulus control, progressive relaxation, and waiting list control. They obtained the experienced therapist data from an earlier study (Turner & Ascher, 1979a). With experienced therapists, improvement in all three treatment groups was reliably better than in the control group. Statistically there was no significant difference among the three treatments. However, with therapists-in-training, the paradoxical intention group performed less well than even the waiting-list controls.

One recent study (Lacks et al., 1983) compared paradoxical intention with stimulus control, progressive relaxation, and a placebo treatment. Clinically, stimulus control produced a 55% improvement in subjective sleep latency, while the improvements for the other groups ranged from a low of 17% for paradoxical intention to 23% for the placebo treatment.

Based on these findings, one cannot yet say that paradoxical intention is a consistently successful treatment technique for sleep onset insomnia. These findings do suggest that cognitive arousal, in the form of presleep performance anxiety, plays a role in delayed sleep onset.

Sleep restriction therapy. A very recently developed behavioural technique, sleep restriction therapy (Spielman, Saskin, & Thorpy, 1984), is dramatically different in focus from any of the preceding techniques. Instead of addressing physiological or cognitive arousal, sleep restriction

therapy focuses on a different factor believed to be perpetuating insomnia: it treats the problem of excessive time spent in bed. As its name implies, the procedure requires the patient to limit the amount of time spent in bed. Thus, initial treatment restricts the total time in bed to the minimum amount of time the patient estimated he or she actually sleeps. As sleep efficiency increases, so does the allowable amount of time spent in bed. Unlike the previously described techniques, this one does not focus on sleep onset insomnia. Its focus is on sleep maintenance problems.

The two studies (Spielman et al., 1984; 1987) published on the use of sleep restriction indicate that it has a stabilizing and consolidating effect on sleep. However, the authors admit that the patients find it difficult to comply with the procedures. More evidence, both subjective and objective, is required before conclusions can be drawn about the efficacy of this treatment.

Methodological Concerns

Many of the studies evaluating the subjective effectiveness of behavioural treatments used a technique developed by Steinmark and Borkovec (1974). These researchers devised a pragmatic procedure to control for improvement due to therapeutic demand or placebo effects. This technique requires testing for improvements in sleep latency twice during therapy. The first assessment occurs under Counterdemand conditions. Until this assessment period, the participants are told that because the therapist does not expect any improvements in sleep latency they should not be discouraged by lack of improvement. Measurements of sleep latency taken during the last week of this Counterdemand condition are assumed to demonstrate treatment effectiveness unconfounded with therapeutic demand or placebo effects.

During the final week of therapy, the Positive Demand condition is implemented. Participants are now told to expect dramatic improvements in sleep latency. Improvements in placebo treated groups are expected to appear during this period, while no further significant improvements are expected in the active treatment groups.

The improvements in subjective sleep latency that studies using the Counterdemand - Positive Demand strategy reported occurred during the course of therapy. Only in a small subset of studies were evaluations of long-term therapeutic effectiveness made. When such evaluations were reported, the results were frequently based on data collected on only those participants in the study who could be reached for a telephone interview sometime after the end of treatment. Participants were asked to estimate their current, typical latency to sleep onset (e.g., Borkovec et al., 1979; Freedman & Papsdorf, 1976; Haynes et al., 1977; Haynes & Price, 1975; Shealy, 1979). Means based on daily reports of sleep latency were infrequently collected. Because of the questionable reliability of one-shot estimates of sleep latency, the claims of long-term effectiveness based on such data need to be viewed with some caution.

To avoid the issue of therapeutic demand effects several researchers used both subjective and objective measures of sleep latency.

Summary

The empirical evidence shows that behavioural treatments are almost uniformly better than placebo in the immediate improvement of sleep onset latency. However, many of the studies tested only the immediate effects and lacked long-term evaluations. Of the studies reporting assessments taken after the end of therapy, few used objective means to measure improvement. Almost all relied on self-reported data.

The methods used to collect the long-term effectiveness data varied from averaging daily estimates taken over 14 days, to telephone interviews during which previous participants were asked to estimate how long, on average, it was now taking them to fall asleep. The length of time between end of treatment and the followup evaluation varied from several weeks to 11 or 12 months Differential participant drop-out affected the reliability of the data collected.

One goal of the behavioural techniques is anxiety reduction which is achieved by means of physical relaxation and cognitive distraction or refocusing, as well as by contingent disruption of sleep-incompatible behaviours and cognitions. The mechanisms underlying their effectiveness may be a reduction in sympathetic arousal and a decrease in subjective apprehension.

Although chronic insomnia is resistant to drug therapy (Kales et al., 1983) behavioural techniques have shown themselves to be relatively successful treatments for psychophysiological and/or subjective insomnia. With psychophysiological insomnia being the most common of the nonmedical and nonpsychiatric insomnias, new behavioural techniques that have the potential for treating it successfully are worthy of attention. Recent research indicates that a unique behavioural technique, flotation REST, may have the necessary prerequisites to qualify as a possible treatment for psychophysiological insomnia.

<u>An Alternative Behavioural Treatment for Persistent Psychophysiological Insomnia</u>

Psychophysiological insomnia is often diagnosed by exclusion. When medical (e.g., arthritis, sleep apnea), psychiatric (e.g. affective disorders), and environmental (e.g., allergies) factors have been ruled out, the

diagnosis of psychophysiological insomnia is considered. To qualify for this classification, there must be objective evidence of sleep disturbance. As well, two other factors are also likely to be present. First, and most importantly, there will be evidence of chronic, somatized tension-anxiety. This may take the form of excessive physical restlessness and cognitive hyperactivity. The second factor, often following from the prolonged association of the somatized tension-anxiety with the attempt to go to sleep, is negative conditioning associated with the bed. This manifests itself as an annoying alertness occurring when trying to go to sleep.

Behavioural treatments that disrupt the cognitive ruminations and focus attention on sleep-inducing stimuli are likely to be successful in easing the somatized tension-anxiety and in reducing the conditioned arousal. It appears that the common element in the relaxation techniques is their ability to perform these two functions. There is evidence that flotation REST can evoke a deep physical and mental relaxation, and can do so with little effort required from the floater. The nature of the environment used for floating is such that external distractions are kept to a minimum. Thus any relaxation technique taught in such an environment is likely to be concentrated on more closely, and perhaps learned more rapidly. Before explaining further how flotation REST might be used to advantage in the treatment of psychophysiological insomnia, it is necessary to describe the technique and to summarize the existing research using it.

Flotation REST. REST is an acronym which stands for Restricted Environmental Stimulation Technique. There are two methodological variants of REST: chamber and flotation. Chamber REST involves confinement to a bed in a room that provides reduced stimulation (darkness and silence). In flotation REST, an individual floats supine in a quiet and

darkened tank in a saturated aqueous solution of $MgSO_4$ which is maintained at 34.2 degrees Celsius.

Since 1977, growing public interest in flotation REST has called into question the previously held negative image of immersion REST (Suedfeld, Ballard, & Murphy, 1983; Zuckerman, 1969). The technique, used since 1977, differs from earlier forms in that one is no longer immersed upright, for an unspecified period of time, in a water tank while wearing a breathing mask. Instead, one now reclines with the face and ventral surface of the body above the water and breathes normally. Sessions are time limited, usually lasting from 45 minutes to 2 hours. The more comfortable and considerably less frightening position, and the time-limited procedure, have eliminated the unpleasant reactions reported in earlier studies.

Suedfeld et al. (1983) assessed the effects of flotation in a heterogeneous group of paying customers at a commercial flotation facility. Floaters, whether novices or experienced, males or females, scored higher than the standardized mean on arousal seeking and on private body consciousness (i.e., a tendency to focus on internal sensations). They also showed a significant decrease in subjective stress during and following their float. The most common feelings reported after the session were: calm, still, at rest, alert, and acquiescent. The flotation environment itself was rated as sleepy, exciting, pleasant and relaxing rather than arousing, gloomy, unpleasant or distressing, respectively. Consequently we see that as little as one hour of flotation REST is experienced as generally relaxing and pleasant.

Within the last six years, systematic examinations of the effects of flotation REST have begun. Turner and Fine (1983) used four 35-minute

exposures to tape-recorded relaxation phrases to examine the effect of relaxation induction on plasma cortisol, ACTH and LH. Half of their participants heard the tape while floating and half heard the phrases while reclining in a dimly lit room. A significant decrease in plasma cortisol occurred in the flotation REST group only. Neither of the other measures showed significant changes, although ACTH levels had a greater downward trend in the REST group. Thus, repeated, brief flotation REST-assisted relaxation procedures can produce a relaxed state which is associated with specific decreases in pituitary-adrenal axis activity.

A more rigorous replication and extension of the Turner and Fine (1983) study by Turner, Fine, McGrady, and Higgins (1987) looked at the effects of flotation REST compared to biofeedback-assisted relaxation training on normotensive and hypertensive patients. The results showed that both REST-assisted and biofeedback-assisted relaxation produced significant decreases in blood pressure. However, only REST-assisted relaxation was associated with consistent decreases in adrenal hormones. If one accepts the hypothesis that decreases in plasma levels of aldosterone, renin and cortisol, and in urinary cortisol, contribute to the relaxation response, then this leads to the suggestion that REST-mediated relaxation may be deeper than biofeedback-assisted relaxation.

In pilot work with three essential hypertensives, Fine and Turner (1982) gave each twenty 40-minute flotation REST sessions without relaxation messages. Each participant maintained clinically significant decreases in systolic and diastolic blood pressure at one month post-treatment. The authors speculated that, for a floater, relaxation is passively induced by the environment. They compared this passive experiencing of relaxation to the passive concentration required in

autogenic training and suggested that the advantage of REST is its ability to induce the sensation of relaxation effortlessly.

Jacobs, Heilbronner, and Stanley (1984) examined the effects of short-term flotation REST plus a relaxation, breathing and visual imagery programme on blood pressure, and on subjective and objective measures of relaxation. They found that ten 45-minute sessions of flotation REST, combined with the guided relaxation programme, produced a significantly greater reduction in systolic and diastolic blood pressure in a normotensive population than did the same programme administered in a normal bedroom environment. The REST group also reported greater subjective relaxation than the control group on a self-report relaxation questionnaire.

The findings showed that practicing a relaxation exercise in the flotation REST environment elicits a greater level of relaxation than practicing the same exercise in a normal sensory environment. The authors were surprised to discover that the dark, quiet flotation tank environment, which produced significant reductions in physiological and subjective arousal, did not elicit a great deal of sleep. The floaters reported falling asleep on 9% of their sessions while the controls reported sleeping on 27% of their trials. This is consistent with the Suedfeld et al. (1983) findings that floating produces a relaxed but alert state of consciousness.

These studies demonstrate that flotation REST is effective in assisting individuals to experience deep relaxation, psychological calmness and decreases in blood pressure. Further, it appears that flotation REST has a synergistic effect when combined with other behavioural techniques. But how does it happen? Jacobs et al. (1984) have suggested that the flotation REST environment strengthens the concentration and attentional abilities of the floater, thus increasing the physiological alterations known to result

from the relaxation techniques that require focused attention (e.g., progressive relaxation, Borkovec, 1982). Turner et al. (1987)'s work supports the notion that REST-assisted relaxation is more potent in reducing adrenal hormone activity than is biofeedback-assisted relaxation.

Although the amount of literature on the effects of flotation REST is small, the early findings suggest that this technique has components common to the relaxation therapies that might qualify it as a potentially successful tool for treating psychophysiological insomnia. It induces both subjective (self-reported) and objective relaxation (as reflected in decreased in blood pressure and adrenal hormones). If flotation REST were combined with autogenic relaxation (which is more compatible with the passive relaxation elicited with flotation than the tension-release technique of progressive muscle relaxation) and used to treat delayed sleep onset in psychophysiological insomnia, one might expect to find significant improvements in sleep.

Primary Research Questions and Hypotheses

The intent of this study was to examine the long-term effects of flotation REST combined with autogenic relaxation training on sleep onset latency. To do this systematically, four comparison groups were included: a flotation REST alone condition, to test for effects of the environment itself; an autogenic relaxation training condition, to evaluate an alternative behavioural technique; and a delayed treatment control condition, to evaluate the effects of time.

Question 1: Is flotation REST effective in decreasing sleep latency over the long term in people with persistent psychophysiological insomnia?

Hypothesis 1: Both REST only and REST and relaxation will significantly decrease sleep latency in persistent psychophysiological insomniacs as long as twelve weeks after treatment.

Question 2: Is flotation REST as effective in reducing sleep latency in persistent psychophysiological insomniacs as autogenic relaxation?

Hypothesis 2: Flotation REST and flotation REST combined with autogenic relaxation training will not differ from autogenic relaxation training in decreasing sleep latency in persistent psychophysiological insomniacs as long as twelve weeks after treatment.

Secondary Research Questions

Cognitive, affective, and physiological arousal have all been proposed as mechanisms maintaining sleep onset insomnia. The current study is based on the proposition that thoughts, feelings, and physiological activity have an interactive impact on sleep latency. Whether the effect is to lengthen or shorten latency depends upon the affective tone and the level of arousal associated with the cognition. Therefore, measures of affect and arousal are included in this study to assess the relation between affective state, arousal and sleep latency.

Question 1: Does treatment have a long-term effect on affect and arousal?

This question gives rise to another, do long-term changes in affect (mood) and arousal correlate with long-term changes in sleep latency? It is expected that reductions in negative affect and arousal will be related to reductions in sleep latency over the long term.

Question 2: Do affect and arousal decrease during treatment?

This question focuses on the immediate impact of treatment on affect and arousal. The expectation is that negative affect and arousal will

decrease immediately after treatment. Consequently the question is raised, "Do changes in affect or arousal occurring during treatment correlate with long-term changes in sleep latency?"

Question 3: Are affect and arousal in persistent psychophysiological insomniacs related to sleep latency prior to treatment?

This question addresses the idea that heightened affect and arousal are found in people who are suffering insomnia. It is expected that persistent, psychophysiological insomniacs would have slightly, but not excessively, elevated levels of negative affect, negative arousal, and anxiety.

Question 4: What relation does circadian rhythm have to sleep latency?

This issue is not directly related to the effects of treatment. Rather, it looks at a possible individual difference among psychophysiological insomniacs that might contribute to the degree of their difficulty in getting to sleep. It does not necessarily imply that there is a circadian rhythm disorder underlying the problem. It is expected that people who feel that their peak activity time occurs in the evening will have more difficulty in getting to sleep than people whose peak period is earlier in the day.

Method

Participants

Recruitment. The 36 persistent psychophysiological insomnia sufferers in this sample were recruited from the community (34 volunteers) and from the Sleep Disorders Clinic at Shaughnessy Hospital (2). Recruitment took place over a 22-month period. During that time newspaper stories, paid newspaper advertisements, radio interviews, free Public Service Television announcements and privately printed brochures publicized the criteria to meet in order to qualify for the free treatment. The criteria were: healthy adults (i) between the ages of 19 and 45 years; (ii) who were receiving neither medication nor therapy for either a medical or psychiatric disorder; (iii) who estimated regularly taking an hour or more to fall asleep; and (iv) who did not work shifts. Individuals meeting these criteria were encouraged to call the REST Laboratory at the University of British Columbia to get more information about the study. Although a sixty minute estimated latency was specified, it was understood that many volunteers would have less than that length of sleep latency. The criterion was set in order to discourage calls from people with mild insomnia. The age limit was specified to avoid potential hormonal confounds in menopausal or postmenopausal women.

The volunteers from the Sleep Disorders Clinic had been referred to the Clinic by their family physician because of their sleep disruptions. Clinic personnel diagnosed these patients as persistent psychophysiological insomniacs (ASDC, 1979). Although six volunteers were referred to the study from the Clinic, only two completed all phases of the study. Of the remaining four, one experienced a spontaneous recovery during Baseline data collection, one withdrew after the 1-week Posttest assessment because

she felt that the evaluation was not helping her, one chose to continue taking medication rather than take behavioural treatment of any kind, and one required medication for nightmares, which disqualified her from the study. All Clinic volunteers were reached through the REST Laboratory and received the same information as to the nature of the study as did volunteers from the general public.

Screening Procedure

Level one - telephone interview. More than 380 respondents were screened over the telephone. The experimenter used a prepared telephone script to guide the conversation (Appendix A). Part 1 of the script introduced the study and asked questions concerning initial criteria for inclusion in the programme. Part 2a dealt with those who were eligible to continue the screening process and Part 2b with those who were not. Of the respondents, 236 people, for one reason or another, did not meet the criteria. For example, some were over age, worked shift work, were taking medication or therapy for a physical or psychiatric condition, or their sleep latency was too short. The disqualified callers who wanted help were referred to the Shaughnessy Sleep Disorders Clinic. The remaining 144, who passed the telephone screening, were given Part 3 of the telephone script which indicated the length of participation and outlined the steps involved in the study. Volunteers referred from the Sleep Disorders Clinic received Parts 1 and 3 of the telephone script.

Level two - assessment forms and clinical interview. The 144 people who met the initial criteria received a package of questionnaires to be completed at home. Of these, 57 were judged appropriate by the Clinic and 36 took part in the study. Reasons for the exclusion of the remaining 21 are discussed in Appendix B. The package included a cover letter explaining

what to do and whom to call when done, a map of the University of British Columbia campus, an 11-page Sleep Assessment booklet plus answering guide (Appendix C) and a 90-item, revised, Symptom Check List (SCL-90-R) (Derogatis, 1983). The two questionnaires were used at the Sleep Disorders Clinic to help assess the severity, duration and type of sleep disorder. The cover letter instructed the volunteer to make an appointment with Dr. Fleming at the Shaughnessy Sleep Disorders Clinic as soon as the forms had been completed. During the next step, Dr. Fleming administered the Millon Clinical Multiaxial Inventory (Millon, 1983), analyzed it and the two forms from home, and conducted a brief clinical interview with the volunteer. He then determined the individual's eligibility for inclusion in the study.

The Millon Clinical Multiaxial Inventory was used as a screening device to assist in making clinical judgments concerning primary behaviour disorders. Insomnia sufferers who seemed to require professional attention for something other than their sleeping problems were excluded from the study because they could not be given an unconditional diagnosis of persistent psychophysiological insomnia. These people were offered assistance, if they wanted it, at the Sleep Disorders Clinic. The experimenter phoned the qualifying volunteers and made an appointment for their Initial Interview.

The final sample. There were 20 women and 16 men in the final sample. Their mean age was 33.3 years and ranged from 24 to 44 years. The mean duration of insomnia was 15.1 years. Prior to treatment, sleep onset latency means and standard deviations for the sample were subjectively estimated as 67.1 (32.35) minutes and objectively measured as 50.0 (27.60) minutes.

The Relaxation Environments and Autogenic Relaxation Exercise Tapes

The REST flotation tank. The flotation tank[2] resembles a large, enclosed, fibreglass bathtub. A sliding door in the top half allows easy opening from both inside and outside the tank. The inner dimensions are approximately 2.4 m long by 1.2 m wide by 1.2 m high. The tank is filled to a depth of 25 to 30 cm with a solution of water and $MgSO_4$ with a specific gravity of about 1.26 and a temperature of 34.2 degrees Celsius.

A waterproof intercom on the wall outside the tank allows auditory monitoring of the floater and voice communication with the experimenter. Speakers for playing tape-recorded material are housed in the shell of the tank.

To use the tank a participant has to be free of any contagious or infectious disease. Further, the floater must not have open sores or large cuts. Small cuts or abrasions are covered with vaseline. Prior to entering the tank, the floater showers and shampoos, then inserts earplugs to keep out water and external sounds. After stepping into the tank and closing the door, the floater sits down, lays back and floats with the face and ventral portion of the body out of the water. Because the water temperature is close to normal skin temperature, the air-water boundary is blurred and the floater feels as if suspended in space. Music is used to signal the end of a float. In the UBC REST facility, floats vary in duration from 45 up to 120 minutes, depending on the nature of the study. Before getting out of the tank, the floater generally stretches both arms and legs. After the session, the floater again showers and shampoos to remove the salt solution.

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[2] The Float-to-Relax company in Denver, Colorado, is gratefully acknowledged for their donation of the flotation tank used in this research.

Instructions on how to float comfortably (for example, avoid splashing the solution on the face, and try a number of arm positions to find personal favourites) are given prior to the first shower on the first day of treatment.

The REST chamber. The REST chamber is a quiet, dimly lit room manufactured by the Industrial Acoustics Corporation. The walls and ceiling are sound absorbent. To add to the restful ambience, the floor has been carpeted and a large landscape poster hung. A speaker and intercom are attached to the wall just behind the head of the bed. Fresh linens, pillows, blankets and comforter are used on the bed. During the autogenic relaxation treatment sessions in this room, the door was closed and the participant made himself or herself comfortable by removing shoes, eyeglasses, and large pieces of jewellery, and loosening tight clothing before reclining on the bed.

The autogenic relaxation exercise tapes. Autogenic relaxation training is considered a psychophysiological self-control therapy (Pikoff, 1984). Its goal is complete physical and mental relaxation. Three principles underlie the training: (i) reduction of exteroceptive and proprioceptive afferent stimulation; (ii) mental repetition of psychophysiologically adapted verbal formulas; and (iii) mental activity conceived as "passive concentration" (Luthe, 1959).

Budzynski's (1974) script of the autogenic relaxation exercise, in which the key phrases focus on the arms and legs feeling heavy and warm, was used. Two copies of the exercise were taped: one, following Budzynski's script, for use in the REST Chamber and the other, amended slightly by removing references to the bed, for use in the REST tank. Both

versions were recorded in the same voice[3] . Each exercise lasted 30 minutes.

The Treatment and Control Conditions

There were four conditions in the study, three treatment groups and one control, with nine people in each. Each set of four treatments took place within a two-week period, usually at the rate of two per week. Each visit lasted between one and one-half to two hours.

REST only (R). People in this group floated four times, for 45 minutes each time, in the REST flotation tank. This condition was included to provide an evaluation of the effect of the flotation REST environment itself on sleep latency.

REST and autogenic relaxation (RA). People in this group received four 45-minute floats, during each of which they followed a 30-minute, pre-recorded, guided autogenic relaxation exercise. The purpose of this condition was to see if the effects of a known treatment technique would enhance the relaxation effects of REST to produce an improvement in sleep latency.

Autogenic relaxation (A). People in this group received four 45-minute treatments, during each of which they followed a 30-minute, pre-recorded, guided autogenic relaxation exercise while reclining comfortably on a bed in the REST chamber. This condition served as a comparison group in the evaluation of the effectiveness of the two REST groups.

Delayed treatment control (C). People assigned to this group completed two periods of sleep monitoring, separated by a 12-week waiting

[3] Dr. Fred Valle of the Psychology Department at the University of British Columbia kindly recorded both of the autogenic relaxation tapes.

period. Treatment was given following the second monitoring phase. This condition controlled for the effect of elapsed time on sleep latency.

Dependent Variables

Sleep, affect, and arousal data were gathered over the course of the study. At the Initial Interview, one personality variable was assessed (see Figure 1 for a schedule of data collection).

Sleep variables. Although the primary focus of this study was on sleep latency, subjective and objective data were collected on two sleep variables: sleep latency (amount of time it took to fall asleep after lights out) and total sleep time (amount of time spent asleep at night).

Sleep data were collected at four different times for the three treatment groups: Baseline, 1-week and 4-weeks Posttests, and at Followup, 12 weeks after treatment; and twice for the control group: Baseline and again 12 weeks later, at Followup.

Affect and arousal measures. Four different paper and pencil questionnaires were used to measure affect and arousal during the study.

The Beck Depression Inventory (BDI) (Beck, 1978) is a 23-item questionnaire which reflects the extent of depression experienced. The BDI was used to evaluate the degree of depression (a negative affective state frequently associated with insomnia) present before treatment, and change in depression after treatment. It was administered during the Initial Interview, at Baseline, both Posttests, and at Followup.

The Profile of Mood States (POMS) (McNair, Lorr, & Droppleman, 1971) assesses transient, fluctuating affective states. Six moods or affective states are measured by this 65-item form: tension-anxiety, depression-dejection, anger-hostility, vigour-activity, fatigue-inertia, and confusion-bewilderment. Tension, depression, anger, fatigue, and confusion all

	PHASES					
MEASURES	**Initial Interview**	**Baseline**	**Treatment**	**1-Week Posttest**	**4-Week Posttest**	**Followup**
1. Subjective Sleep Variables						
- Sleep Log -						
i. Latency						
ii. Total Sleep Time		14		7	7	7
2. Objective Sleep Variables						
- SAD Recordings -						
i. Latency						
ii. Total Sleep Time		4		3	3	3
3. Beck Depression Inventory (BDI)	1	1		1	1	1
4. Profile of Mood States (POMS)	1	4	4 4 pre post	3	3	3
5. Self-Report Arousal Scale (SAS)	1	4	4 4 pre post	3	3	3
6. Blood pressure and pulse	1		4 4 pre post			
7. State-Trait Anxiety Inventory (STAI)	1					
8. Morningness-Eveningness Questionnaire (MEQ)	1					

Figure 1. Schedule for data collection.

reflect negative mood states. Vigour is the only positive mood measured by the POMS. These moods were measured at Baseline to see whether sleep latency was related to higher scores. The POMS was administered immediately before and after each treatment session to assess changes in mood due to treatment. Data were also collected at both Posttests and at Followup to see if moods changed over time.

The Self-Report Arousal Scale (SAS) (Kjellberg & Bohlin, 1974) assesses phenomenological arousal or activation. Its six subscales are: sleep-wakefulness, energy, stress, euphoria, irritation, and concentration. Wakefulness and energy reflect phenomenological physiological arousal, stress and irritation reflect negative arousal associated with the interpretation of a bodily state, euphoria reflects positive arousal, and concentration refers to current ability to focus attention. The SAS was administered at the same times as the POMS questionnaire.

The Spielberger State-Trait Anxiety Inventory (STAI- Form Y) (Spielberger, 1983) measures two constructs: anxiety as an unpleasant emotional state and anxiety as a relatively stable personality trait. This inventory was administered only once, to assess the initial levels of state and trait anxiety in order to see how anxiety related to Baseline sleep variables and to improvements in sleep performance.

Blood pressure and pulse readings were taken as objective measures of physiological arousal. However, these arousal measures had to be dropped from analyses due to a foreseeable, but overlooked methodological problem. Early in the course of data collection it became obvious that for the flotation REST groups any observable differences in the pretreatment to posttreatment measurements of blood pressure and pulse would be highly confounded with the actions of showering, dressing and returning to the

REST chamber for assessment. To maintain a consistent procedure across participants, these physiological data were collected for all people although they were not used in the evaluation of treatment effects on objectively-defined physiological arousal. These measures were intended to test the relation between objective physiological arousal and sleep latency.

Although these data were not used, the procedure for collecting them is described. The measures were obtained at the Initial Interview, and again before and after each treatment session. Blood pressure was measured three times at each assessment period. The first measurement was for calibration only. The mean systolic and diastolic pressures were calculated using the second and third readings.

All readings were taken while the person sat comfortably in a chair, in the REST chamber. Before Baseline and pre-treatment measures were obtained, each person spent at least ten minutes sitting in the chamber completing forms and talking. The posttreatment measures were taken as soon as possible after the treatment session ended.

Personality variable. A measure of individual differences was administered at the Initial Interview:

The Morningness-Eveningness Questionnaire (MEQ) (Horne & Ostberg, 1976) measures individual differences in circadian rhythms. The total MEQ score falls into one of five categories: Definitely a Morning Type; Moderate Morning Type; Mixed Type; Moderate Evening Type; Definitely an Evening Type. These types referred to the time of day when an individual felt at the peak of personal alertness.

Sleep Monitoring Instruments

Subjective measurement. Because insomnia is a subjective complaint, self-reports of sleep performance are a valid dimension in the

evaluation of treatment. The sleep log provided a convenient format for recording self-reported sleep performance.

The sleep log (Pollak, 1979). Up to 14 nights of sleep can be recorded on one sleep log form. The log consists of two key parts: a graph and a set of questions about sleep. The purpose of the graph is to provide a visual record of sleep patterns. The times that a person went to bed, fell asleep, woke up during the night and finally got out of bed are all marked. In the question section, the person records such things as how long it took to fall asleep, whether hypnotics or alcohol was used at bedtime, how many night awakenings occurred, and total amount of sleep obtained.

Objective measurement. Although insomnia is thought of as a subjective complaint, and therefore self-reports of sleep performance are relevant, several studies have questioned the ability and, indeed, the motivation of individuals to estimate their own sleep performance accurately (e.g., Carskadon et al., 1976; Kales & Kales, 1974). Because there is some question as to the validity of self-report sleep data, objective measures of sleep performance are often used as evidence of an initial sleep disorder and to corroborate estimates of improved sleep performance. Participants could have been asked to spend time in the Shaughnessy sleep lab, instrumented with EEG, EMG and EOG electrodes, in order to provide an all-night recording of sleep performance. However, aside from the additional cost in time and inconvenience to the participant, sleep performance away from home was not the central focus of the study. An alternative method of objectively assessing sleep performance while the sleeper was in his or her regular environment was sought. The solution

chosen was the SOMTRAK Sleep Assessment Device, which quickly became dubbed the SAD machine[4].

The SOMTRAK or sleep assessment device (SAD). The SAD machine, consisting of a timing device and an adjustable tone generator attached to a cassette tape recorder and fitted inside an attache case, was sent home with the participants four different times during the study. The first time that the machine was taken home a detailed demonstration was given, demonstrating the steps described in the printed instructions that accompany the machine. Before going to bed on each night of use, when ready to try going to sleep, the participant recorded the time and date, then pushed the "start timing" button. The controls were set so that the first 1-second tone would go off 10 minutes after starting time and then every 10 minutes throughout the night. Immediately prior to the tone, the tape recorder would begin recording for a 10-second period. If the person was awake and heard the tone, a response of "Hello" or "I'm awake" was given during the 10-second recording time. For three people, the interval between tones was changed from 10 to 20 minutes because they became annoyed by the shorter interval. They felt they could not get to sleep before the next beep came.

The volume and pitch of the tone were adjusted by the user. Because auditory threshold levels increase during sleep and therefore the ability to detect signals decreases (Rechtschaffen, Hauri, & Zeitlin, 1966), the sound of the tone was set loud enough to be heard in a quiet room, but not so loud that it would awaken a light sleeper. Auditory awakening thresholds during sleep range from 15 dB to over 100 dB above background room noise

[4] Six ST-100 SOMTRAK units were purchased from Farrall Instruments, Inc., Grand Island, NE.

level (Ogilvie & Wilkinson, 1984). This leaves a great deal of latitude in setting the volume for the tone at an appropriate level.

The SAD machine provides an inexpensive and a much less intrusive alternative to sleep lab polysomnography. Tests of the clinical validity of SAD recordings indicate no significant differences between EEG- and SAD-generated data in comparisons of sleep latency, total sleep time, duration of awakenings during the night, and sleep efficiency percentage (Lichstein, Nickel, Hoelscher, & Kelley, 1982). The continuous monitoring of the EEG, however, reveals significantly more night awakenings than does the time-sampling method of the SAD recording. Thus some data may be lost through the use of the SAD machine. Nevertheless, in this study the ability to examine the participants' sleep in their own beds far offset the anticipated loss of data.

Timetable

There were three major phases in the study: Baseline, Treatment and Posttreatment Assessments (see Figure 2). The pretreatment measurement period was called Baseline, the immediate posttreatment evaluation was called the 1-Week Posttest, the short-term evaluation was called the 4-Weeks Posttest and the long-term evaluation, done at 12 weeks after the last treatment, was called the Followup. Below is an outline of the phases.

Baseline Phase

Initial interview. This interview took place after the volunteer qualified for inclusion in the study. It took approximately an hour and a quarter to complete. Volunteers were given a copy of their basic rights and privileges as research participants, and were asked to sign two copies of a consent form, one for the experimenter and one for their own files.

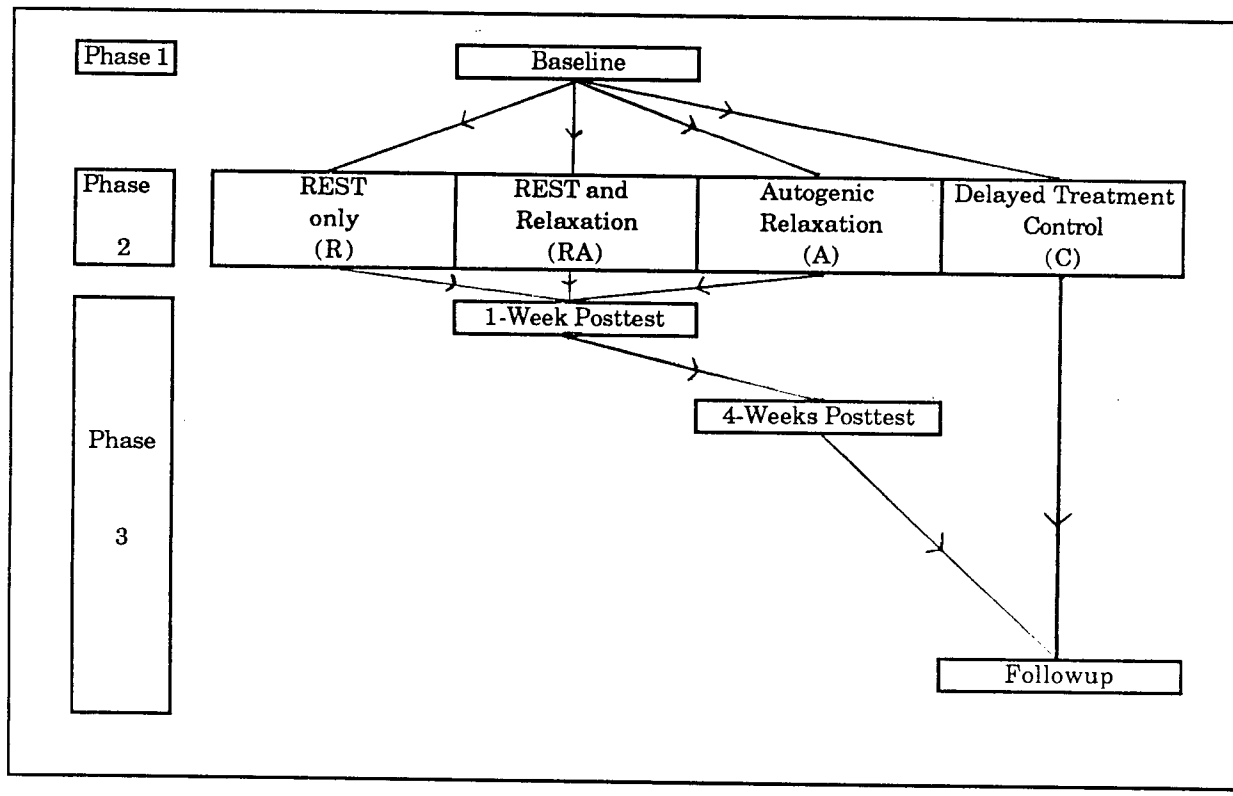

Figure 2. Overview of study.

(Appendix D). During the interview several questionnaires (Beck Depression Inventory, State-Trait Anxiety Inventory, Morningness-Eveningness Questionnaire) were administered.

After completing the tests, the participants scheduled the dates for their Baseline monitoring phase. They were instructed on how to fill in the daily sleep log. Once this was done, they were shown the REST chamber and flotation tank. They were then given a brief explanation of the operation of the Sleep Assessment Device (SAD) and reminded of the $50 refundable deposit, mentioned in the consent form, which was designed to encourage the careful use and return of the machine. Three baseline blood pressure and pulse recordings were taken. These and all future measures were taken in the REST chamber.

Baseline sleep monitoring. All participants recorded their sleep behaviour in a sleep log for 14 nights. On the last four of these nights they also used the SAD machine to monitor their sleep. For each of the four SAD monitoring nights, the Profile of Mood States and the Self-Report Arousal Scale forms were completed prior to turning the lights out. The Beck Depression Inventory was filled out on the last night of sleep monitoring. The sleep log, SAD machine and questionnaires were returned to the REST Laboratory at the end of the monitoring period.

The participants were then randomly assigned to one of the four conditions. They had been told during the initial screening that they would have no choice as to which treatment they received, and all had agreed to accept whichever treatment they were assigned.

The blocked assignment method was not used because of the time it took to recruit qualified volunteers. Participants qualified one at a time over a period of 22 months. To wait until all 36 people had qualified before

assigning them in blocks of four to their conditions was not feasible. Therefore, random assignment with replacement was used until each condition was filled.

The participants who were assigned to the delayed treatment control condition were told that their treatment would begin thirteen weeks later and that because of the long time between their baseline and their treatment it would be necessary to re-evaluate their sleep. Thus a seven-night sleep log monitoring period was required just before treatment began, along with a three-night SAD evaluation plus the same paper and pencil questionnaires already completed during the Baseline phase. Dates for this second baseline were scheduled, as were tentative treatment days.

Treatment Phase

Four treatment sessions were given over a 14-day period, with at least one day separating the sessions. Each session occurred in the morning so as to discourage reliance upon the immediate relaxation effect of the therapies for sleep induction. All treatments took place in the REST Laboratory at the University of British Columbia, where the flotation tank and the REST chamber are located.

Before and after each treatment session, approximately 15 minutes were spent in collecting Profile of Moods States and Self-Report Arousal Scale data, and blood pressure and pulse measurements.

Before the participant began the first treatment session, a deep breathing exercise was demonstrated. It consisted of a four-by-four cycle: breathe in to the count of four, hold for four, breathe out to the count of four, and hold for four, then repeat three times, for a total of four cycles. This breathing pattern was used at the beginning and end of each treatment and

before each of the homework practice sessions (discussed below) to help focus attention on internal sensations.

Rationale. All participants were told that the two goals of the treatment programme were: one, to help them develop a Personal Standard of relaxation, and two, to help them to enhance their ability to concentrate, so that they could focus on their Standard. Good concentration skills would help them focus attention on that Standard whenever cognitive or physical hyperarousal threatened to delay their sleep onset. The treatment sessions were aimed at achieving goal number one, and at providing a distraction-free environment in which concentration could be practised easily. Homework assignments in cognitive focusing were given between each treatment to further assist with goal number two.

On the first day of treatment, treatment-specific consent forms were read and signed by each participant (see Appendix D). Everyone was told general information about normal sleep, about two major hypotheses concerning the mechanisms underlying insomnia (i.e., cognitive hyperarousal and physiological hyperarousal), and how relaxation therapies work at reducing cognitive and physiological arousal. The two key principles of relaxation treatment for insomnia were described as (a) distraction from any mental activity that is incompatible with sleep and (b) attention to and experience of sleep-conducive sensations. Each person was also told how his or her particular treatment was believed to implement these two principles.

The autogenic relaxation participants were told that the autogenic exercise would (a) assist in distracting attention from overly arousing mental activity (e.g., worrying about what you said at work today; or going over and over what you plan to do at the meeting tomorrow; or just fearing

that you won't be able to get to sleep tonight), and would (b) lead to pleasant sensations of deep physical relaxation. The mental and physical sensations were then to be remembered as their own Personal Standard of relaxation. At bedtime they were to practise the exercise and recall their Standard of relaxation. In so doing they would be applying the two principles of relaxation therapy, (i.e., distraction from bothersome, overly arousing, mental activity, and intentional focus on pleasant, physiologically relaxing sensations) to help decrease their sleep latency.

People in the REST only condition were told that the flotation environment passively elicits sensations of deep physical and mental relaxation. These sensations were to be remembered as their own Personal Standard of relaxation. At bedtime the Standard was to be recalled so it could serve as (a) a distraction from bothersome, overly arousing, mental activity, and as (b) a pleasant, physiologically relaxing sensation, thus applying the principles of relaxation therapy in order to help decrease sleep latency.

The REST and autogenic relaxation group was told that the REST environment passively elicits sensations of deep physical and mental relaxation and that the autogenic exercise would (a) assist in distracting attention from overly arousing mental activity and would (b) lead to pleasant sensations of deep physical relaxation. The mental and physical sensations were to be remembered as their own Personal Standard of relaxation. At bedtime, the exercise was to be practised and their Standard of relaxation was to be recalled, so as to apply the principles of relaxation therapy.

Homework. To aid in the development of good concentration skills cognitive focusing exercises were assigned for homework. Cognitive

focusing requires that attention be placed on one thing for a particular length of time and that distractions be conscientiously dismissed or ignored during that period. These distractions can be internal, as in irrelevant thoughts, or external, such as irrelevant noises. Each time the mind wonders away from the object of focus, the divergence is to be acknowledged and the mind is to be refocused on the object.

Cognitive focusing homework was assigned for the period between each pair of treatment sessions. Between sessions 1 and 2, the participants were asked to practice cognitive focusing using a real object. The same object was to be used each time. The participants were to look closely, as if seeing the object, such as a pencil, for the first time. They were to say to themselves everything that they could see. For example, "I see the imprint of the manufacturer's label, and some teeth marks on the end. The eraser appears extremely worn on one side." Participants were instructed to practice focusing four times daily for two minutes each time. The timing of their practice sessions was left up to them, with the provision that they should try to distribute them evenly throughout the day and evening.

In the interval between treatments 2 and 3, the four daily, 2-minute practices were distributed such that the real object was observed on the first and third practice and was imagined on the second and fourth practice. This new form of practice was introduced so that the participants would begin to shift their cognitive focusing from physical objects to mental images. Finally, during the third interval (that is, between treatments 3 and 4), the mental image was the object of focus at all four practices.

This progression in the cognitive focusing exercise from concentrating on an object that is physically present to concentrating on a

mental representation of that object was used to make the development of good concentration skills as easy as possible.

Relaxation practice. In addition to the cognitive focusing described above, every night during the Treatment phase the participants were instructed to try to recall the sensations they experienced during their treatment sessions and to rehearse their relaxation exercise. The exercise depended upon which treatment the individual received. People receiving autogenic relaxation, whether in the chamber or in the tank, focused on key phrases from the exercise. For example, they said over to themselves such things as "My arms and legs are heavy and warm. Warmth is flowing into my hands and feet." People in the REST only condition were asked to focus on the physical sensations of relaxation that they recalled passively experiencing during their floats. The idea was to practice paying attention to pleasant, monotonous, relaxing experiences and sensations, which were in and of themselves conducive to sleep because they were physiologically soothing. The focused attention also served to distract attention from sleep-incompatible thoughts and images.

Before the questionnaires were administered in sessions 2, 3 and 4, therapeutic progress was discussed. Any problems related to the homework and any questions about the treatment procedure were dealt with in this 15 to 20 minute period. While non-sleep related problems were acknowledged, active psychotherapy was avoided. After each treatment and its accompanying posttreatment measures had been administered, a brief inquiry was made into the degree of relaxation experienced and relevant thoughts about the sensations. This subjective response to the experience was elicited to help the participant develop the images and sensations of relaxation that would make up his or her Personal Standard

of relaxation. Finally, the homework assignment was reviewed. The discussion and review took about ten to fifteen minutes.

While a recent study by Lacks and Rotert (1986) indicated that poor sleep hygiene practice is not a central cause of insomnia, the researchers suggested that sleep hygiene information be included as part of a behavioural treatment package so as to avoid exacerbation and chronicity of the insomnia. By decreasing the tendency of poor sleep leading to poor hygiene practice, which in turn leads to poorer sleep, hygiene education can supplement behavioural treatment. Therefore, on the last day of treatment all participants were given two handouts for their files. One was a list of sleep hygiene suggestions (Appendix E). Concern over the construct validity of the stimulus control paradigm (Haynes et al. 1982) and questions about the active component in the procedure (Zwart & Lisman, 1979) limited the addition of a stimulus conditioning component to the presentation of printed instructions embedded in this series of sleep hygiene tips. The other handout was chapter 12 from The Complete Book of Sleep (Hale, 1981). These handouts together provided information on good sleep habits and their relation to sleep.

Both the cognitive focusing techniques and the sleep hygiene information were presented to each treatment group in an attempt to avoid outcome differences due to lack of practice in concentrating and to limited knowledge of good sleep hygiene. Even though the homework in cognitive focusing was restricted to 8 minutes a day over the two-week treatment period, it was felt that the practice would provide a minimum baseline of skill common to each treated participant.

Cognitive focusing, as used in this study, should not be confused with meditation training, as used in the treatment of insomnia (e.g., Woolfolk et

al., 1976). In meditation emphasis is placed on the type of mental activities, such as focusing on breathing, or on repeating a mantra, or on visualizations of a calm scene. So important are these focusing activities that the major portion of each treatment session is spent rehearsing them. Further, up to sixty minutes of daily practice at home is also required for periods of three (e.g., Mitchell & White, 1977) or four weeks (e.g., Woolfolk et al., 1976).

In this study the goal of the cognitive focusing homework was to help the participants attain a minimal level of proficiency so that they could focus their attention on the relaxation sensations long enough to develop a personal standard of relaxation. The amount of time spent practising cognitive focusing exercises during the treatment phase of the study was significantly shorter than any such practice used in meditation training. Thus, the two focusing methods are sufficiently different to reduce the concern that the cognitive focusing homework may play a determining role in any outcome effects of treatment.

Treatment Conditions

REST only (R). Each person in this group received four 45-minute long floats. Before each float, the floater was reminded to become aware of feelings of relaxation, and to look for evidence that muscle tension had begun to be released.

REST and autogenic relaxation (RA). Before each of the four 45-minute floats, participants in this group were reminded to focus on the key phrases in the pre-recorded autogenic relaxation exercise. The autogenic relaxation tape began two minutes after entering the tank. It lasted for 30

minutes. Each floater was told to rehearse the key phrases from the exercise at least once during the last portion of the session.

Autogenic relaxation (A). People in this group received four 45-minute treatments while reclining comfortably on a bed in the dimly-lit REST chamber. The autogenic relaxation instructions were the same as in the RA condition. The exercise tape was played over the speaker at the head of the bed.

Delayed treatment control (C). People assigned to this group did two Baseline sleep monitoring phases. The first was identical to that of the three treatment groups. The second baseline occurred twelve weeks afterward. A seven-night sleep log was kept and for the last three of those nights SAD recordings were made. The Profile of Mood States and the Self-report Arousal Scale questionnaires were completed just prior to bedtime on each night of SAD recordings. On the last night the Beck Depression Inventory was also filled out.

After the second monitoring period, the people in this group received two autogenic relaxation and two REST only treatment plus three posttreatment assessments.

Posttreatment Assessments Phase

To assess the long-term effectiveness of the treatments, evaluations of subjective and objective sleep latency were made 12 weeks after the last treatment session. This assessment is referred to as the Followup. For the control group the Followup occurred 12 weeks after the Baseline evaluation.

Because the pattern of change for the three treatment groups was also of interest, two other assessments of sleep latency were also made. They occurred 1 week and 4 weeks after the last treatment session and are referred to as the 1-Week and 4-Weeks Posttest, respectively. They

represent an immediate and a short-term evaluation of improvements in sleep latency. The control condition did not receive these additional assessments because they were not relevant to its purpose. Because some people go through cycles of poor sleep, it was important to evaluate whether long-term improvements found after treatment could be explained by the simple passage of time.

Each assessment involved seven nights of sleep log monitoring, with SAD recordings occurring on the last three of those nights. In addition, Profile of Mood States and Self-report Arousal Scale forms were completed prior to bedtime on each night of SAD recording. The Beck Depression Inventory was completed on the last night of evaluation during the two Posttests and at Followup.

Final interview. This debriefing interview occurred when the sleep log, questionnaires and SAD machine were returned at the end of the twelve-week Followup. At this time the $50 deposit was returned, and a description of the other treatments in the study was given. Because it was not known which treatment would have the best results in terms of sleep latency improvement, all participants were offered the opportunity to take four more booster treatments of their choice. These were optional and were not considered part of the study.

Sixteen of the 36 participants chose to take one or more boosters in the form of REST only sessions. Of the sixteen repeaters, five had been in REST only, four in REST and relaxation, four in autogenic relaxation only, and three in the control group. The consensus was that floating was not only therapeutic, it was also fun.

Procedures for Data Reduction

Once all the data were collected the raw observations were

transformed into analyzable data using the following procedures.

Subjective sleep measure: The sleep log. An individual's mean sleep latency was calculated by summing the estimates recorded in answer to the question "How long did it take you to fall asleep last night?" and then averaging them for each applicable measurement period. Four means (i.e., Baseline, 1-Week and 4-Week Posttests, and Followup) were collected for each of the treatment groups and two means (Baseline and Followup) for the control group. Data from the first night that the SAD machine was being used in each monitoring period were omitted due to the novelty of having a machine in the bedroom that beeped every ten minutes throughout the night. Data from any "atypical" night (e.g., nights with occurrences such as illness; a serious car accident outside the house; an unsolicited and annoying phone call just after bedtime) were also omitted. Group means for all sleep variables were calculated from the appropriate individual means.

Nights that were omitted from sleep latency calculations were also deleted from mean total sleep time. The remaining estimates of total sleep were summed and averaged from the answers to the question "How much sleep did you get last night?"

Objective sleep measure: The SAD recordings: Individual means were calculated based on data from Nights 2, 3, and 4 of SAD recordings for the Baseline period and on data from Nights 2 and 3 in the Posttests and Followup recording periods.

Individuals' responses to the tone prompt were coded on a form under columns labelled "Awake" and "Asleep". Each column was divided into intervals. In transcribing the tape any meaningful response, such as

"yes", "hello", "I'm here" or even "Uh huh", heard after a tone was coded as "Awake" for that interval. Unintelligible grunts and sighs were not coded. If no meaningful sounds from the participant were heard, the interval was coded as "Asleep". Each interval normally represented 10 minutes (except for the three people, whose interval was 20 minutes). One minute of tape time was equal to 60 minutes of real time (120 minutes for three subjects)..

The coded transcriptions were used to calculate sleep latency. Recall that the tone and recording period came at the end of the 10-minute interval. Latency was determined by first counting the number of "Awake" intervals before the first "Asleep" interval, then multiplying by 10 minutes and adding 5 minutes. This method assumed that for each "Awake" interval, 10 minutes had gone by. The person must have fallen asleep in the time between the last "Awake" and the first "Asleep" interval. The best guess as to when sleep occurred was half way (5 minutes) between the tones marking the two intervals. The number of intervals was multiplied by 20 minutes and 10 minutes were added for those people using the 20-minute interval.

Here is an example of sleep latency calculations: The sleeper identifies the start time as 12 midnight and starts the timing device. At intervals 1, 2, and 3 a response of "I'm awake" is recorded; three check marks are placed in the "Awake" column. In the 10-second interval after the fourth tone no response is heard. A check mark is then placed in the "Asleep" column for interval 4. Sleep latency is determined as follows. For each of the three "Awake" intervals, 10 minutes of wake time is counted, for a total of 30 minutes. Halfway between tones 3 and 4 the volunteer is

assumed to have fallen asleep. Another 5 minutes are added to the total, making the sleep latency 35 minutes in all.

To calculate total sleep time, the number of "Asleep" intervals was counted and multiplied by 10 (or 20, if the inter-tone interval was 20 minutes). The first and last "Asleep" intervals are assumed to compensate for each other, so that this method of counting and multiplying gives the best estimate of total sleep time.

Subjective measures of arousal.

Profile of Mood States and Self-Report Arousal Scales. The 65 items on the Profile of Mood States questionnaire were rated on a scale of 0 to 4 (0 = not at all, 2 = moderately, and 4 = extremely). The 31 items on the Self-Report Arousal Scale were rated on a scale of 1 to 5 (1 = not at all, 3 = moderately, 5 = very much). Individual means for each subscale on these two tests were calculated by averaging the scores obtained during each of the administration periods. For example, during Baseline four scores were collected for each subscale. These scores were summed and divided by four to provide an individual's mean score on that subscale for that assessment. When data were missing, the mean was calculated from the scores which were present. Thus, each person had a mean score on every subscale for all testing times. Group means were calculated from individual means.

The Beck Depression Inventory. Scores were calculated by adding the value of the highest selected item under each question. The higher the score, the greater the depression. Total scores between 0 and 9 represent no depression, scores from 10 to 14, borderline depression, scores from 15 to 20, mild depression, scores between 21 and 30, moderate depression, scores from 31 to 40, severe depression, and finally, scores from 41 to 63, very severe depression.

The Spielberger State Trait Inventory. This inventory provided two anxiety scores: state and trait. Individual items were scored, according to a key, for the degree of anxiety expressed. The item scores were summed to provide a state anxiety score and a trait anxiety score. On both scales, the higher the score, the greater the anxiety.

The Morningness-Eveningness Questionnaire. A scoring key provided a value for each item. An individual's score was the sum of the values for all 18 items. High total scores were associated with people whose peak activity level occurred in the morning. Low total scores indicated evening-time peak activity levels.

Results

Analysis Procedures

The pretreatment assessment is referred to as Baseline, the 1-week and 4-week assessments as 1-Week Posttest and 4-Weeks Posttest, and the 12-week assessment as Followup. For the control group, Followup refers to the assessment which took place twelve weeks after Baseline. Assessments of affect and arousal variables collected immediately before and after each of the treatment sessions are referred to as Pretreatment and Posttreatment assessments. The unadjusted means and standard deviations for the sleep latency variables and the sleep duration variables at each assessment are presented in Appendices F and G.

In order to compare the subjective and objective measurements of sleep latency, individual pairs of subjective and objective latency means from each of the four assessment periods were correlated. Before calculating individual means, the first night of SAD-recording and the sleep log measure for the corresponding night in each assessment period were usually deleted. However, across the four assessment periods, the first night was all that was available in 8 instances: one in Baseline, 5 in the 1-Week Posttest, and 2 in the 4-Week Posttest. Therefore, the first-night pair was used for these 8 individual means. Nights, other than the first, when both measures of latency were available were used to calculate individual means for each period.

The maximum number of nights upon which the individual means for each assessment period were calculated, varied for two reasons. First, at Baseline, the means for the individuals in each of the treatment groups were based on up to three nights, whereas the individual means in the

control group were based on up to five nights. (The five nights included three from the first Baseline monitoring period and two from the second assessment, twelve weeks later. These were collapsed because the control group had received no treatment between the two assessments, and because there were no significant differences between the means at Baseline and the means at Followup.) At the Posttest assessments and at the Followup assessment the maximum number of nights incorporated in the individual means was two per assessment.

The second reason is that the number of nights on which any individual mean was based could vary if the individual experienced an atypical night while doing the monitoring. All atypical nights were omitted from the calculations.

The maximum number of pairs of individual means at each assessment period varied for two reasons. First, only the three treatment groups were compared at Posttests and Followup. Therefore, only twenty-seven means could be involved. Second, individuals occasionally did their SAD monitoring after their sleep log monitoring; therefore, no corresponding pairs of subjective and objective sleep latency values were available.

On the basis, then, of the individual subjective and objective sleep latency scores (means) calculated as described above, the two measures were correlated at each of the four time points. As the following values show, these correlations were significant and high: Baseline: r (32) = .60, p < .001; 1-Week Posttest: r (22) = .75, p < .001; 4-Weeks Posttest: r (23) = .55, p < .005; and Followup: r (22) = .65, p < .001.

The main analyses were divided into four sections: (a) primary and (b) secondary analyses of the sleep variables, (c) analyses of the affect and

arousal variables, and finally, (d) correlational analyses of the affect, arousal, and sleep latency variables.

First an overview of the procedures used in each of these sections is given; then the results are presented.

Primary analyses of sleep variables. The study employed a two-way factorial design with two levels of flotation REST (present, absent) and two levels of autogenic relaxation (present, absent). A multivariate analysis of covariance (MANCOVA) on the Followup means for subjective and objective sleep latency and subjective and objective sleep duration was conducted. The four corresponding Baseline means were covaried.

Because this analysis yielded a significant main effect and a significant interaction, two-way, univariate analyses of covariance (ANCOVA) were performed on each dependent variable with the corresponding Baseline mean as the covariate.

To determine whether flotation REST differed in effectiveness from autogenic relaxation, planned comparisons between the two levels of flotation REST treatments and the autogenic relaxation treatment alone were done on subjective and objective sleep latency means. A second set of planned comparisons between the three treatment groups and the control group was done to see whether receiving treatment was more effective in reducing sleep latency than the simple passage of time. A third set of planned comparisons between the two flotation REST conditions was also performed to see whether the two levels of REST differed in effectiveness. All comparisons were mutually orthogonal and all were conducted with the adjusted group means and adjusted mean squares according to the method described by Kirk (1982, pp. 734-736).

An identical set of three planned comparisons was carried out on adjusted subjective sleep duration means. No comparisons were justified for objective duration because there were no significant ANCOVA results for that variable.

Secondary analyses of sleep variables. A secondary analysis of the sleep latency variables at 1 week and 4 weeks posttreatment using a one-way, univariate ANCOVA on the individual dependent variables was conducted to examine the immediate and short-term effects of treatment. Univariate ANCOVAs, instead of a preliminary MANCOVA, were used because the variables of most interest in this study were only two in number: the subjective and objective sleep latency variables. Because significant effects were not found, no further analyses were carried out on these data.

Analyses of affect and arousal variables. The subscales of the Profile of Mood States (POMS) questionnaire and the subscales of the Self-Report Arousal Scale (SAS) were analysed separately. The analyses paralleled those used with the primary sleep variables. The analyses were concerned with answering several questions.

(a) The initial question addressed in this section was, "Was there a long term effect of treatment on affect and arousal?" The first analysis on the subscales of the POMS and of the SAS was a two-way MANCOVA on the Followup means with the appropriate six Baseline means as the covariates. Because the hypotheses related to these variables were of secondary interest in this study, and because so many analyses were being performed, a more stringent criterion, alpha = .01, was used to decrease the likelihood of a Type 1 error.

There were no significant effects in the MANCOVA for either the POMS or the SAS subscales. Thus, no further analyses were performed on the Followup means.

To answer the same question as above, a two-way, univariate ANCOVA was computed on the Followup group means for the Beck Depression Inventory, with the Baseline group means as the covariate. There was no significant effect. Further analyses, therefore, were not justified.

(b) To find what immediate impact treatment had on the affect and arousal variables, one-way MANCOVAs were computed on the Posttreatment group means for POMS and SAS variables with the appropriate Pretreatment group means as the covariates. The one-way design was used because the control group did not complete these measures. No significant MANCOVA effects were found for the POMS. However, a significant effect in the MANCOVA on the SAS subscales justified further analyses.

One-way, univariate ANCOVAs were computed on each of the six SAS subscales to determine which variable or variables were responsible for the significant effect.

Correlational analyses. Glass and Hopkins (1984, pp. 128-130) outlined a method of measuring change that avoided being complicated by the regression effect. By means of their method, change in the affect, arousal, and sleep variables was measured by predicting the Followup scores from the Baseline scores and then using the deviation between the actual Followup scores and predicted Followup scores as a measure of change beyond that predictable from the Baseline scores alone. The resulting deviations, sometimes called residual gain scores, but here called

residual change scores, were then correlated in order to find the degree of relation between changes in the affect and arousal variables and changes in the sleep variables. There were four main questions in this section. In an attempt to reduce the likelihood of a Type 1 error in the correlational analyses, alpha was set to .005. Based on support of prior empirical results and on theoretical grounds correlations between reductions in negative affect and arousal and reductions in sleep latency were expected to occur. Correlations between negative affect and arousal and Baseline sleep latency were also expected. For these analyses, one-tailed tests were used.

Question 1 in the correlational analyses asked, "How were changes in affect and arousal related to changes in sleep latency over the long term?" Residual change scores were calculated for Baseline to Followup on all affect and arousal variables (POMS, SAS, and BDI) and on subjective and objective sleep latency variables. These residuals were then correlated to determine the extent of the relation between changes in affect and arousal and changes in sleep latency.

Question 2 was concerned with the relation between changes in affect and arousal occurring at the time of the treatment sessions and changes in sleep latency over the long term. Do changes during treatment in affect and arousal correlate with changes in sleep latency over the long term? Residual change scores for POMS and SAS subscales which used Pretreatment scores as predictors of Posttreatment scores were correlated with residual change scores in sleep latency which had used Baseline latency means as predictors of Followup latency means.

Question 3 asked, "Are affect and arousal in persistent psychophysiological insomniacs related to sleep latency prior to treatment?" Two sets of Baseline affect and arousal variables (POMS, SAS, and BDI)

were collected. One was collected during the daytime at the Initial Interview, and the other was collected at night, during the Baseline sleep monitoring phase. Each of the daytime and nighttime affect and arousal variables was correlated with both subjective and objective sleep latency.

State and Trait Anxiety scores were gathered only during the daytime Initial Interview. They were correlated with both Baseline sleep latency variables to see if there was a relation between state or trait anxiety and length of sleep latency.

Question 4 asked, "What relation does circadian rhythm have to sleep latency?" Morningness-Eveningness Questionnaire (MEQ) scores, obtained during the daytime Initial Interview were correlated with Baseline subjective and objective sleep latency means to determine the extent of the relation between circadian peak activity and length of sleep latency.

Results of the Main Analyses

Primary analyses of sleep variables.

Hypothesis 1: Both REST only and REST and relaxation will significantly decrease sleep latency in persistent psychophysiological insomniacs as long as twelve weeks after treatment.

The central focus of this study was the long-term effectiveness of flotation REST, either in combination with autogenic relaxation or on its own. Alpha was set at .05 for the primary sleep analyses. A two-way factorial design, with flotation REST (present, absent) crossed with autogenic relaxation (present, absent), was used. Figure 3 shows the four cells of the 2 X 2 design and labels the conditions represented by each. To facilitate description, the four conditions, when referred to individually, will be called: REST only (represented by R), REST and autogenic relaxation (RA), autogenic relaxation only (A), and control (C). When used to describe

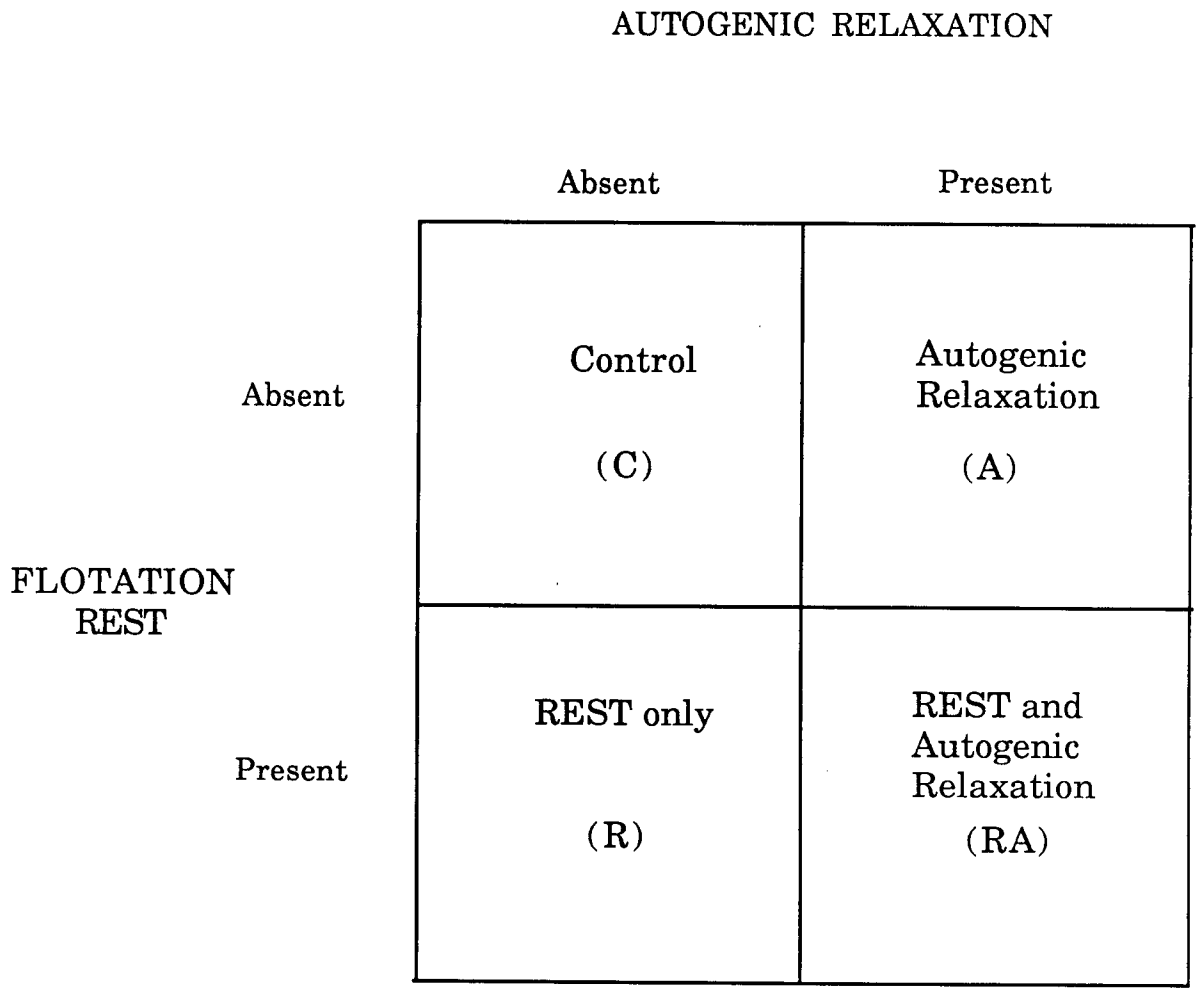

Figure 3. Two-way factorial design.

analytic effects, REST (R and RA) refers to both the REST only condition and the REST and relaxation condition, while AUTO (A and RA) refers to both the autogenic relaxation condition and the REST and autogenic relaxation condition. A two-way, multivariate analysis of covariance (MANCOVA) was computed on the Followup means for all four sleep variables: (a) subjective and (b) objective sleep latency, and (c) subjective and (d) objective sleep duration. All four Baseline sleep variable means were covaried. The test of significance used was Wilks' Lambda (Λ).

The MANCOVA yielded a significant AUTO main effect [F (4,25) = 5.20, $p < .01$, $\Lambda = .546$] and a significant REST by AUTO interaction [F (4,25) = 2.80, $p < .05$, $\Lambda = .691$]. The REST main effect was nonsignificant [F (4,25) = 1.34, $p > .25$, $\Lambda = .823$].

To probe the significant effects of the MANCOVA, two-way univariate ANCOVAs were performed in turn on the Followup means of each of the four sleep variables using the single corresponding Baseline measure as covariate. This plan was considered justified because the interaction effect and the autogenic relaxation main effect in the MANCOVA had established overall significance as well as Type 1 error protection.

The results from the univariate ANCOVAs are shown in Table 1. A significant REST main effect and a REST by AUTO interaction were found in the analyses of (a) subjective sleep latency, and (b) objective sleep latency. Figures 4 and 5 are graphs of the adjusted means, showing the interaction effects. No significant main effect for autogenic relaxation were found for either sleep latency variable.

There was a significant main effect for autogenic relaxation on subjective sleep duration as well as a significant REST by AUTO

Table 1
Results of Univariate Analyses of Covariance of Four Sleep Measures Taken 12 Weeks After Treatment

Dependent Variables		Sleep Latency		Sleep Duration	
		Subjective	Objective	Subjective	Objective
Group $M^{a,b}$					
R[c]		27.6	21.3	371.8	382.7
RA		45.9	32.1	394.8	383.3
A		44.4	31.2	414.5	390.5
C		67.4	57.0	335.4	351.9
MS					
(df = 31)		441.13	533.77	1063.41	2612.37
F -ratio for Effects					
REST[d]	*F*	5.63	5.01	.58	.48
	p	< .05	< .05	>.50	>.50
AUTO	*F*	.52	.89	22.26	1.59
	p	>.50	>.50	< .001	>.50
Interaction	*F*	4.65	5.71	6.46	1.42
	p	< .05	< .05	< .05	>.50

Note : For each dependent variable analyzed the Baseline score on the same variable was used as covariate.
[a] The group means are adjusted (for the effect of the covariate) means.
[b] $n = 9$ per group.
[c] R = REST only; RA = REST and autogenic relaxation; A = autogenic relaxation only; C = control.
[d] REST = R and RA; AUTO = A and RA; Interaction = REST by AUTO.

analytic effects, REST (R and RA) refers to both the REST only condition and the REST and relaxation condition, while AUTO (A and RA) refers to both the autogenic relaxation condition and the REST and autogenic relaxation condition. A two-way, multivariate analysis of covariance (MANCOVA) was computed on the Followup means for all four sleep variables: (a) subjective and (b) objective sleep latency, and (c) subjective and (d) objective sleep duration. All four Baseline sleep variable means were covaried. The test of significance used was Wilks' Lambda (Λ).

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There was a significant main effect for autogenic relaxation on subjective sleep duration as well as a significant REST by AUTO

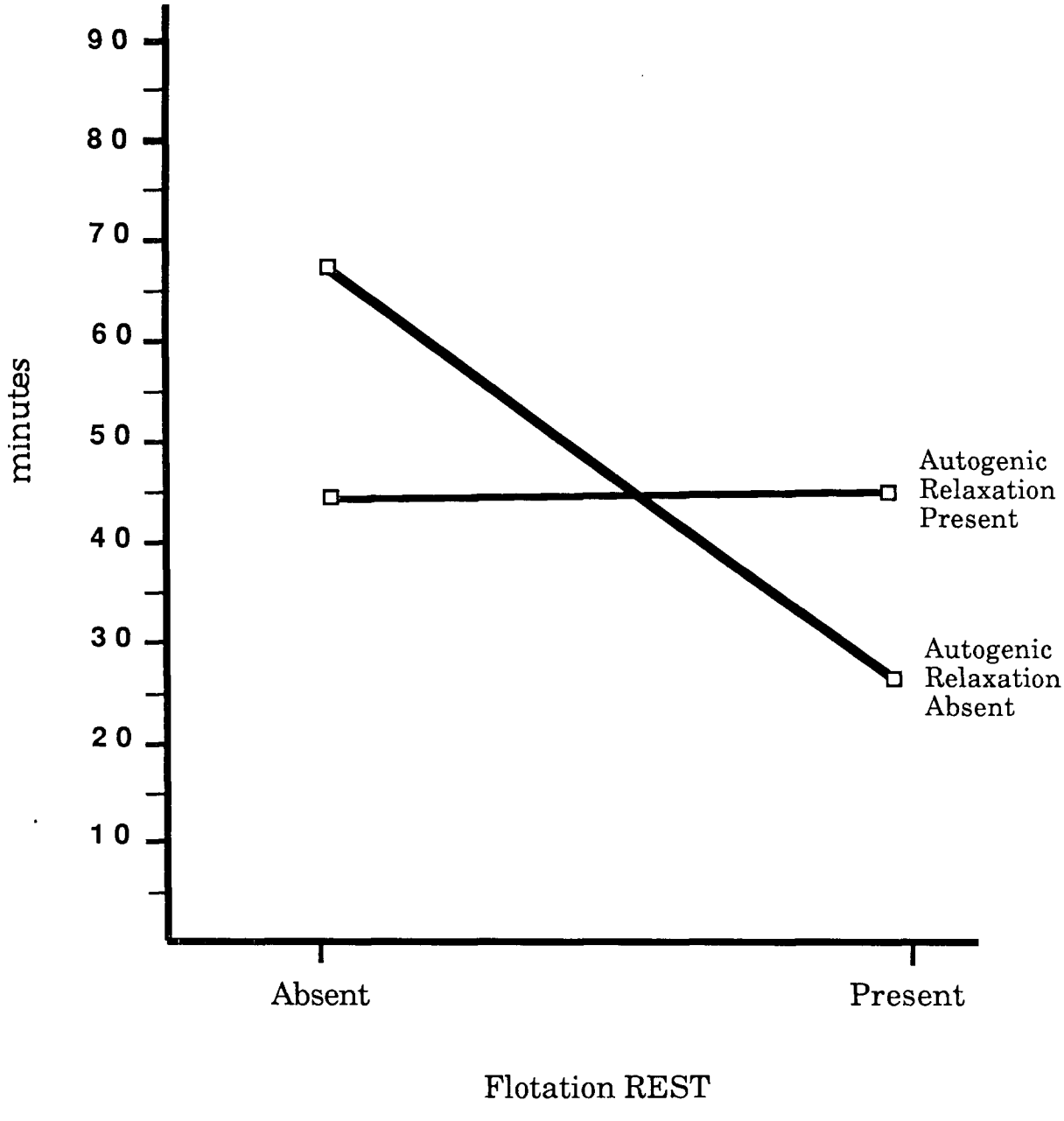

Figure 4. Subjective sleep latency interaction: REST present versus REST absent (based on adjusted means).

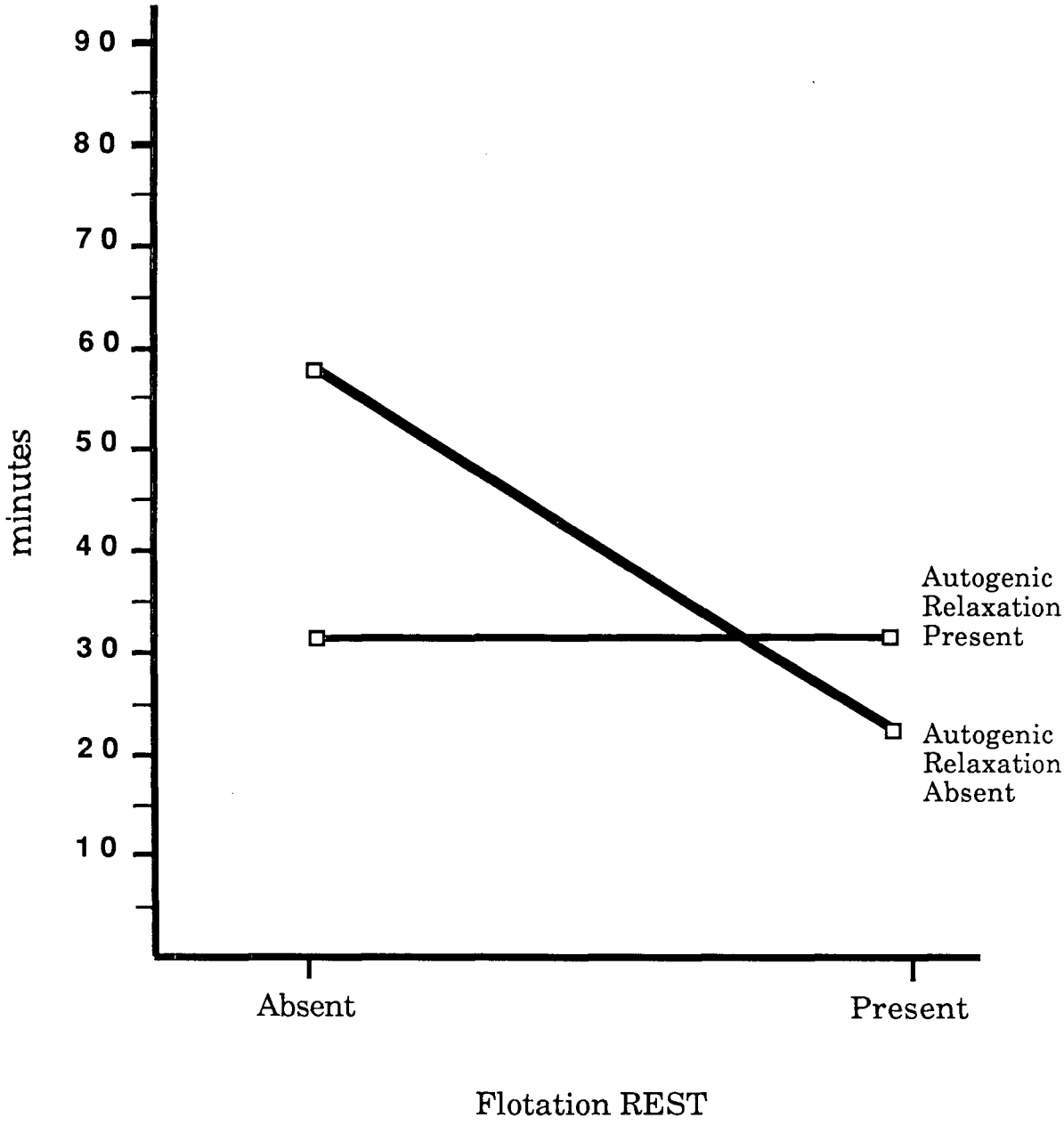

Figure 5. Objective sleep latency interaction: REST present versus REST absent (based on adjusted means).

interaction. Figure 6 is a graph of the adjusted means showing the interaction effect. The results of the analyses were all nonsignificant with respect to objective sleep duration.

Summary: The main effect of REST in both subjective and objective sleep latency is difficult to interpret in light of the significant REST by AUTO interactions. However, the planned comparisons, discussed below, help to clarify these results.

Likewise, the main effect of autogenic relaxation in subjective sleep duration is hard to interpret because of the significant interaction. The planned comparisons, discussed below, help to explain these findings.

Hypothesis 2: Flotation REST and flotation REST combined with autogenic relaxation training will not differ from autogenic relaxation training in decreasing sleep latency in persistent psychophysiological insomniacs as long as twelve weeks after treatment.

For the two sleep latency variables, planned comparisons were conducted on the Followup adjusted group means, with alpha set to .05. All comparisons were mutually orthogonal. The comparisons were: (a) flotation REST (R and RA) versus autogenic relaxation (A), (b) treatment (R, RA, and A) versus no treatment (C), and (c) REST only (R) versus REST and autogenic relaxation (RA). The t -values for the subjective sleep latency comparisons were: (a) R and RA versus A, t (31) = -.71, p > .10; (b) R, RA, and A versus C, t (31) = 2.92, p < .01; and (c) R versus RA, t (31) = -1.81, p > .05. The t -values for the objective sleep latency comparisons were: (a) R and RR versus AR, t (31) = -.47, p > .10; (b) R, RA, and A versus C, t (31) = 3.16, p < .01; and (c) R versus RA, t (31) = .96, p > .10. The results of the first and third planned comparisons were, therefore, nonsignificant for both latency variables. The results of the

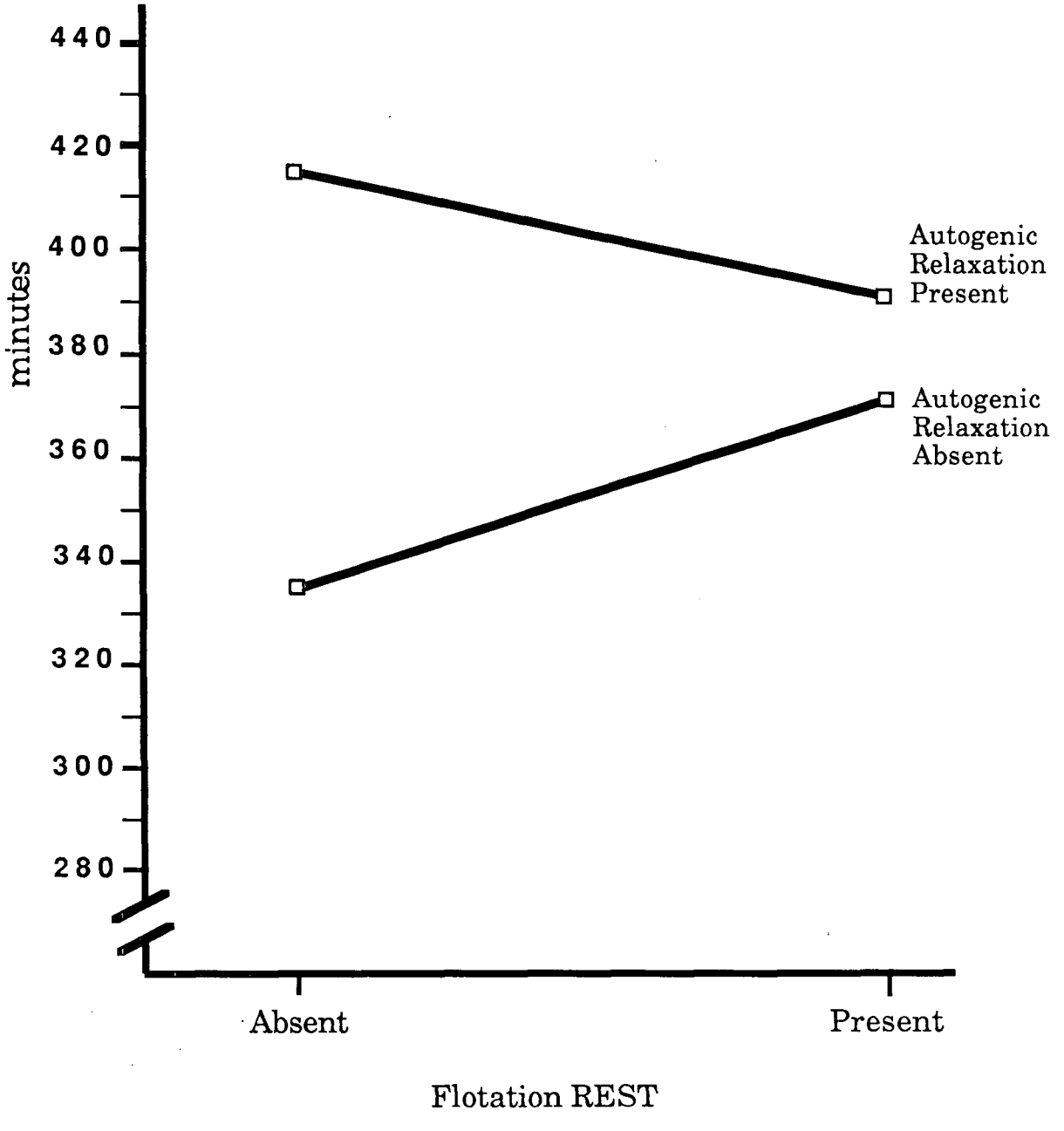

Figure 6. Subjective sleep duration interaction: REST present versus REST absent (based on adjusted means).

second set of comparisons were significant for both latency variables. These results indicate that there were no differences among the three treatment means at Followup, nor between the two REST group means at Followup, but that there was a significant difference between the treatment groups, on the one hand, and the control group on the other.

Summary: These analyses demonstrated that the flotation REST treatments do not differ from autogenic relaxation treatments, nor from each other, in their ability to reduce sleep latency over the long term. They also show that receiving any of the three treatments used in this study, is more effective in reducing sleep latency than receiving no treatment at all.

These results indicate that flotation REST and autogenic relaxation are equally effective in reducing sleep latency in persistent psychophysiological insomniacs and that they are more effective than just letting time pass. They further suggest that the main effect for REST found in the univariate ANCOVAs on the sleep latency variables may be interpreted as supporting the assertion that flotation REST significantly reduces sleep latency over the long term.

Three planned comparisons were carried out on the Followup adjusted group means for the subjective sleep duration variable only. Alpha was set at .05. All comparisons were orthogonal. The comparisons were the same as the three conducted on the two latency variables. The *t*-values for the subjective sleep duration comparisons were: (a) R and RA versus A, t (31) = -2.31, p < .05; (b) R, RA, and A versus C, t (31) = -4.58, p < .01; and (c) R versus RA, t (31) = -1.47, p > .10. The result of the first comparison shows that the flotation REST groups differed from the autogenic relaxation group in subjective sleep duration at Followup. The result of the second comparison indicates that there was a significant

difference between the control group and the three treatment groups in subjective sleep duration. The third comparison shows that the two REST groups do not differ in subjective sleep duration at Followup.

Summary: These comparisons demonstrated that receiving any of the treatments in this study led to significantly longer self-reported sleep duration than receiving no treatment. However, the autogenic relaxation appeared to be more effective in increasing self-reported sleep duration in persistent psychophysiological insomniacs than was flotation REST. There was no difference between the two flotation REST groups in ability to increase sleep duration. None of the main effects tested earlier with the objective sleep duration variable were significant.

Secondary analyses of sleep variables. A set of one-way, univariate analyses of covariance (ANCOVA) was conducted on the subjective and objective sleep latencies at the 1-Week Posttest (Appendix H) and the 4-Week Posttest (Appendix I) to look at the immediate and short-term effects of treatment, respectively. No explicit hypotheses were being tested. Only the three treatment groups were included in these analyses. The results were all nonsignificant.

Analyses of affect and arousal variables.

Question 1: Does treatment have a long-term effect on affect and arousal?

The purpose of this question was to focus attention on affect and arousal variables as potential mechanisms underlying prolonged sleep latency. Affect variables measured by the Profile of Mood States (POMS), and arousal variables measured by the Self-Report Arousal Scale (SAS), were analyzed separately at Followup using a two-way (flotation REST and autogenic relaxation) MANCOVA on the group means with the appropriate

subscale Baseline means as the covariates. Because of the number of analyses performed on these variables a more stringent criterion, alpha = .01, was used as Type 1 error protection.

The results of the MANCOVAs did not reach significance at alpha = .01. For the POMS analysis, the values were: REST main effect: F (6,21) = .39, p > .50, Λ = .899; AUTO main effect: F (6,21) = 2.62, p < .05, Λ = .572; and REST by AUTO interaction: F (6,21) = 1.24, p > .25, Λ = .737. For the SAS analysis, the values were: REST main effect: F (6,21) = .69, p > .50, Λ = .836; AUTO main effect: F (6,21) = 1.38, p > .25, Λ = .717; and REST by AUTO interaction: F (6,21) = 1.86, p > .10, Λ = .653. These results suggest that there were no differences among the group means at Followup on any of the affect and arousal variables when differences related to their Baseline means had been covaried

To test for long-term treatment effects on depression, another affect variable, a two-way, univariate ANCOVA was computed on the Followup group means for the Beck Depression Inventory, with the Baseline scores as the covariate (Appendix J). There was no significant main effect for either REST [F (1, 20) = .57, p > .50] or for autogenic relaxation [F (1,20) = 1.23, p > .25], nor was there a significant interaction [F (1,20) = .01, p > .50].

Summary: These analyses indicate that no significant differences in affect and arousal, as measured by the POMS, SAS, and BDI, were found among the groups twelve weeks after treatment (or after Baseline, for the control group).

Question 2: Do affect and arousal decrease during treatment?

To examine the immediate impact of treatment on affect and arousal, one-way MANCOVAs were computed on the Posttreatment group means

for the six Profile of Mood States subscales and for the six Self-Report Arousal Scale subscales with the corresponding Pretreatment group means as covariates.

The result of the MANCOVA on the POMS variables was nonsignificant [F (12,26) = 1.23, p > .10, Λ = .407]. For the SAS, however, the result was significant [F (12,26) = 3.58, p < .005, Λ = .142]. This overall significant result justified further probing to determine where the treatment groups differed.

One-way, univariate ANCOVAs were conducted on each of the six SAS subscales, with the corresponding individual Pretreatment variable as the covariate. The only significant finding was on the wakefulness scale [F (2,23) = 10.45, p < .001] (Appendix K). The REST groups reported a greater degree of wakefulness following treatment than did the autogenic relaxation group. The means were: 15.5, 14.7, and 12.7 for the REST only, REST and relaxation, and autogenic relaxation groups, respectively. Wakefulness does not mean the inability to sleep, but refers to the bodily state of feeling wide awake. Using the generalized studentized range statistic (see Kirk, 1982, pp. 734-736), post hoc Newman-Keuls comparisons of means (at the .01 level) showed that the REST only mean and the REST and relaxation mean were significantly higher than the autogenic relaxation group mean. Further, the two REST group means did not differ significantly.

Summary: The immediate impact of treatment on affect and arousal was minimal. Only the degree of wakefulness differed significantly among the treatment groups, with the two REST groups reporting greater wakefulness after floating than the autogenic relaxation group reported

after receiving the autogenic relaxation training session in a quiet, dimly-lit room.

Objective measurements of arousal, in the form of blood pressure and pulse recordings, were incorporated in the study to assess the impact of treatment on physiological measures. However, during the course of the study it became obvious that any observable differences in the posttreatment measurements of blood pressure and pulse would be confounded with the floaters' having a shower, dressing, and walking to the REST chamber. Therefore, the effect of the REST treatments on objective measures of arousal could not be accurately assessed. For general information, Table 2 presents the group systolic and diastolic mean blood pressures, along with the corresponding pulse rates, for Baseline, pretreatment and posttreatment assessments.

Correlational analyses. Four sets of correlational analyses were carried out. The first three dealt with relations among various combinations of affect, arousal, and sleep latency variables. The fourth correlational analysis dealt with the relation between circadian rhythm and sleep latency.

Question 1: How are changes in affect and arousal related to changes in sleep latency over the long term?

The purpose of the first set of correlational analyses was to determine the degree of the relation between changes in affect and arousal and changes in sleep latency. Residual change scores (beyond those predicted from the Baseline scores) for Baseline to Followup on the six Profile of Mood States affect variables and the six Self-Report Arousal Scale arousal variables were correlated with residual change scores for Baseline to Followup on the subjective and the objective sleep latency variables. As

Table 2

Blood Pressure and Pulse: Means and Standard Deviations

Group[a]		Baseline	Pretreatment	Posttreatment
			Systolic Blood Pressure	
REST Only	M	102.8	102.8	101.7
	SD	9.9	10.0	9.8
REST & Relaxation	M	112.0	109.7	110.7
	SD	16.0	15.4	14.9
Autogenic Relaxation	M	99.7	101.1	99.1
	SD	11.1	12.2	12.2
Control	M	105.3	-	-
	SD	10.6	-	-
			Diastolic Blood Pressure	
REST Only	M	71.0	71.2	69.9
	SD	6.9	8.3	7.0
REST & Relaxation	M	76.4	72.6	71.4
	SD	6.4	8.1	7.8
Autogenic Relaxation	M	70.0	69.6	70.8
	SD	12.4	12.3	11.9
Control	M	73.4	-	-
	SD	6.5	-	-
			Pulse	
REST Only	M	61.1	69.2	64.8
	SD	9.1	4.8	4.0
REST & Relaxation	M	62.2	67.0	62.8
	SD	3.5	5.7	6.3
Autogenic Relaxation	M	63.6	70.0	61.7
	SD	7.3	7.1	6.6
Control	M	63.8	-	-
	SD	10.2	-	-

[a] $n = 9$ per group

noted earlier, the alpha level was set to .005, one-tailed for these correlations.

The results of these correlations are shown in Table 3. Changes in tension, as measured by the POMS, correlate positively and significantly with changes in subjective sleep latency. Changes in anger correlated with changes in objective sleep latency. No changes in the SAS subscales correlated with changes in sleep latency.

Residual change scores for the Beck Depression Inventory, measured at Followup, did not correlate with changes in either subjective or objective sleep latency. This was true both when the residual change score used the nighttime depression mean (collected during the Baseline Sleep Monitoring Phase) as the predictor score and when the daytime mean (collected during the Initial Interview) was used.

It should be noted that throughout the study, the mean level of depression as measured by the Beck Depression Inventory was always below 10. Scores between 0 and 10 are classified as indicative of no depression.

Summary: The greater the reduction in tension, from Baseline to Followup, the greater the reduction in subjective sleep latency by Followup. In addition, the greater the reduction in anger, from Baseline to Followup, the greater the reduction in objective sleep latency over the same period.

Question 2: Do changes during treatment in affect and arousal correlate with changes in sleep latency over the long term?

The purpose of the second set of correlational analyses was to see whether changes from pretreatment to posttreatment in the affect and arousal variables were related to changes in the sleep latency variables from Baseline to Followup. Other treatment studies, where reductions in

Table 3

Correlations Between Affect and Arousal Residual Change Scores and Sleep Latency Residual Change Scores From Baseline to Followup

Subscales	Subjective Latency	Objective Latency
	Profile of Mood States[a]	
Tension	.44*	.33
Depression	.15	.11
Anger	.35	.47*
Vigour	.24	.23
Fatigue	.17	.04
Confusion	.01	.07
	Self-Report Arousal Scale[a]	
Wakefulness	-.04	.06
Energy	.12	-.03
Stress	.17	.32
Euphoria	.09	.09
Irritation	.16	.31
Concentration	-.03	.10
	Beck Depression Inventory	
Depression		
Day[b]	.34	.28
Night[c]	.15	.19

[a] $N = 36$; [b] $N = 34$; [c] $N = 25$.
* $p < .005$, one-tailed.

physiological arousal during treatment were expected to be positively related to reductions in sleep latency at the end of the study, asked a similar question (e.g., Borkovec et al., 1979; Coursey et al., 1980; Hauri, 1981). Their question was based on the idea that elevated physiological arousal was an underlying mechanism for prolonged sleep latency. Their results showed little relation between the immediate impact of treatment on physiological arousal and final treatment outcome with respect to sleep latency. It remained to be seen in this study whether immediate changes in affect and arousal would be related to changes in sleep latency at the end of the study.

Posttreatment residual change scores for the subscales on both the Profile of Mood States and the Self-Report Arousal Scale were correlated with the residuals from Baseline to Followup on the two sleep latency variables. The results showed that none of the POMS or SAS variables were significantly correlated with changes in sleep latency (Table 4).

Summary: There were no significant correlations between reductions in the affect or arousal variables over the two-week treatment period and reductions in sleep latency from Baseline to Followup.

Question 3: Are affect and arousal in persistent psychophysiological insomniacs related to sleep latency prior to treatment?

The purpose of the third set of correlational analyses was to see how closely the Baseline levels of affect and arousal correlated with the Baseline levels of sleep latency. There were two sets of Baseline affect and arousal variables. One was collected during the daytime at the Initial Interview, and the other was collected at night, during the Baseline Sleep Monitoring Phase. The daytime affect and arousal variables included the Profile of Mood States, the Self-Report Arousal Scale, the Beck Depression Inventory, and the State-Trait Anxiety Inventory. The nighttime affect and arousal

Table 4

Correlations Between Affect and Arousal Residual Change Scores (Pre- to Post treatment) and Sleep Latency Residual Change Scores (Baseline to Followup)

Subscale[a]	Subjective Latency	Objective Latency
	Profile of Mood States	
Tension	.04	.10
Depression	-.35	-.26
Anger	-.19	.07
Vigour	.24	.21
Fatigue	.33	.22
Confusion	-.23	-.05
	Self-Report Arousal Scale	
Wakefulness	-.04	-.01
Energy	.13	-.07
Stress	.01	.21
Euphoria	.23	.11
Irritation	-.01	.09
Concentration	.14	.19

[a] $N = 27$.

variables included only the first three of these measures. Individual Baseline mean ratings on all of the affect and arousal variables were correlated with individual Baseline means for the two sleep latency variables.

The results of the correlational analyses are presented in Table 5. No daytime or nighttime variables were significantly correlated with latency before treatment.

Summary: There was no relation between levels of daytime or nighttime affect or arousal before treatment and degree of sleep latency at Baseline.

As Table 5 shows, neither state nor trait anxiety scores were correlated significantly with subjective or objective Baseline sleep latency. The group means (and standard deviations) for anxiety were: state 36.64 (11.01) and trait 44.56 (10.65). The Manual for the State-Trait Anxiety Inventory provides norms for adult working men and adult working women separately. For state anxiety the means (and standard deviations) are: men 35.72 (10.40), and women 35.20 (10.61). For trait anxiety the means (and standard deviations) are: men 34.89 (9.19), and women 34.79 (9.22). When broken down by sex in this study, the state anxiety mean (and standard deviations) for men was 38.31 (11.12), and for women, 35.30 (11.02). The trait anxiety mean for men was 44.31 (11.10), and for women was 44.75 (10.56). In this study, there were no differences between the state and trait anxiety scores for men and women [state, t (34) = .81, p > .10; trait t (34) = -.12, p > .10]. Mean state anxiety scores were nonsignificantly higher than the corresponding norm [for men, t (15) = .93, p > .10; for women, t (19) = .04, p > .50]. Mean trait anxiety scores, however, were

Table 5
Correlations Between Affect and Arousal Measure Scores and Sleep Latency Scores at Baseline

Arousal Measure	Subjective Latency		Objective Latency	
	Interview (Daytime)	Baseline (Night)	Interview (Daytime)	Baseline (Night)
		Profile of Mood States[a]		
Tension	.09	.26	-.07	.05
Depression	.39	.18	-.06	-.08
Anger	.08	.20	-.25	.13
Vigour	-.05	-.14	-.20	-.30
Fatigue	.25	.15	-.01	-.19
Confusion	.29	.28	-.09	-.07
		Self-Report Arousal Scale[a]		
Wakefulness	.01	.09	-.05	.09
Energy	-.06	-.32	-.05	-.24
Stress	.18	.14	.08	-.10
Euphoria	.02	-.06	.00	-.26
Irritation	.01	.15	.01	.16
Concentration	-.13	.03	.09	-.09
		Beck Depression Inventory		
Depression	.27[a]	.16[b]	-.15	-.04
		State - Trait Anxiety[a]		
State	.04	-	-.09	-
Trait	.26	-	.06	-

[a]$N = 36$; [b]$N = 25$.

significantly higher than the norm for men [t (15) = 3.39, p < .01] and than the norm for women [t (19) = 4.22, p < .001].

Summary: Neither state nor trait anxiety was correlated significantly with sleep latency before treatment, although in this study the participants had a mean level of trait anxiety significantly higher than the norm.

Question 4: What relation does circadian rhythm have to sleep latency?

The final correlational analysis was between the Morningness-Eveningness Questionnaire (MEQ) score and Baseline sleep latency. This analysis tested the hypothesis that people who feel that their peak activity time occurs in the evening will have more difficulty in getting to sleep than people whose peak period is earlier in the day. Scores from the MEQ (high scores indicating morning peak activity) were correlated with Baseline subjective and objective mean sleep latency to see what relation peak activity time had to sleep onset. In the attempt to reduce Type 1 error, alpha was set at .005. One-tailed tests were conducted here because the relations were predicted earlier.

The results showed a nonsignificant negative correlation between MEQ scores and subjective latency at Baseline [r (34) = -.26, p > .05] and a significant negative correlation between MEQ scores and objective sleep latency at Baseline [r (34) = -.46, p < .005].

Summary: Objective sleep latency was longer for people who had more of an evening alertness peak than for people whose alertness peak was earlier in the day. No significant difference between evening-type and morning-type people was found in length of self-reported latency.

Discussion

Does Flotation REST Decrease Sleep Latency Over the Long Term in People with Persistent Psychophysiological Insomnia?

The results indicated that flotation REST significantly reduced sleep latency over the long term in people with persistent psychophysiological insomnia. This reduction was found in both the self-reported and in the objectively-recorded measurements.

The immediate and short-term effects of flotation REST treatments on sleep latency were nonsignificant. Thus, we have the situation where sleep latency appears to decrease over time, but does not reach a significant level of reduction until twelve weeks after the end of treatment.

Is Flotation REST as Effective as Autogenic Relaxation in Reducing Sleep Latency in Persistent Psychophysiological Insomniacs?

The results indicated that the flotation REST treatments did not differ significantly from the autogenic relaxation treatments in their ability to reduce sleep latency over the long term in people with persistent psychophysiological insomnia. Thus, in spite of the main effect for flotation REST in both subjective and objective sleep latency, the REST reductions were not significantly different from those obtained by the autogenic relaxation treatment. The results from the earlier analyses in light of these findings suggest that receiving treatment, at least any of the three treatments given in this study, led to greater reductions in sleep latency than receiving no systematic treatment.

The observation that flotation REST is as effective as autogenic relaxation in reducing sleep latency raises an interesting question as to why they did not differ. Might it be because autogenic relaxation is actually a minimal form of self-induced REST? As Suedfeld (1980) pointed out, there are some strong similarities between REST (i.e., the chamber form) and autogenic relaxation; for example, the emphasis of autogenic relaxation training on relaxation and concentration are similar to the goals of restricted environmental stimulation techniques. The reduction of afferent and efferent impulses is a major goal common to both REST and autogenic relaxation. The difference between the two techniques is in the method of achieving the reduction. REST is more stimulus-bound in its method of reducing afferent and efferent impulses, achieving this end through environmental control. On the other hand, autogenic training requires a quiet, non-stimulating environment in which to practice gaining cognitive control over the same impulses. In this study, for the autogenic relaxation condition, the training was limited to a pre-recorded set of procedures played over a speaker into a dimly-lit REST chamber. The participant heard these instructions while reclining comfortably on a bed. In other words, the environment used for autogenic relaxation training was very similar to the chamber REST environment itself. Perhaps the similarities between the components of the autogenic relaxation condition and the components of chamber REST might explain the finding that none of the three treatments used in this study differed among themselves in their ability to reduce sleep latency. Whether differences between flotation REST and chamber REST exist on variables relevant to this study (e.g., degree of

relaxation elicited, amount of concentration facilitated) is yet to be established.

We also see that there were no significant differences between the two REST treatments in their ability to reduce sleep latency. Perhaps "messages" (i.e., any verbal material presented to the participant during the REST session) do not necessarily enhance the therapeutic effect of flotation REST. Evidence to suggest this possibility comes from chamber REST research. In a recent study on smoking cessation, Suedfeld & Baker-Brown (1987) found that reductions in the number of cigarettes smoked after treatment did not differ between a group receiving "therapeutic messages" (such as "I owe my body respect and protection; I need my body to live; For my body, smoking is a poison") played over a speaker during their 24 hours in chamber REST and a group that received no messages at all while in the chamber. A review of other smoking studies where messages did not enhance the effectiveness of REST is provided by Suedfeld (1984).

There are, however, examples of messages enhancing the therapeutic outcome. Borrie and Suedfeld (1980) found that chamber REST plus messages led to a significantly greater weight loss in obese women six months after treatment than did REST without messages. Recently Cooper, Adams, and Scott (1988) reported that a brief period of chamber REST combined with factual messages and positive experimenter expectancy led to significant reductions in alcohol consumption in "early prodromal drinkers" two weeks after treatment. The reduction was maintained after three months. Unexpectedly, this group was not alone in achieving

significant long-term reduction. The chamber only REST control group, which did not receive messages, also achieved a statistically significant decrease in alcohol consumption three months after treatment. Interestingly, the reductions for chamber only REST two weeks after treatment were nonsignificant making the posttreatment pattern for the chamber only REST condition in the Cooper et al. (1988) study similar to the pattern for all three treatment conditions in the present study.

What is the Effect of Treatment on Sleep Duration?

When a treatment decreases the length of time it takes to fall asleep, one might logically expect to find a corresponding increase in the total amount of sleep obtained. However, an increase in sleep duration subsequent to treatment might not occur for a number of reasons. For example, the sleep latency decrease may have occurred because the individual learned to recognize when he or she was truly sleepy and so stayed out of bed until drowsy enough to fall asleep rapidly. In this case, the length of time it took to fall asleep would be decreased simply by starting the attempt later in the night. Thus the "awake" time would be spent outside of bed, and would, therefore, not contribute to the sleep latency variable. Another reason might be that the individual gets up earlier after treatment because the sleep obtained earlier in the night, due to the shorter latency, was sufficiently restful that prolonged bed time was not necessary.

In this study the results indicated that self-reported sleep duration was longer at Followup for the treatment groups than for the control group. They further showed that sleep duration was longer at Followup in the autogenic relaxation condition than in the flotation REST conditions. Thus,

not only did sleep latency decrease in all three treatment groups, but subjective sleep duration also increased in each. The self-reported duration, however, was greatest for the autogenic relaxation group. There were no significant effects for objective sleep duration, making it difficult to argue that treatment led to increased sleep duration.

It appears that the treatments in this study decreased both subjective and objective sleep latency but only increased subjective sleep duration. The possible role of extraneous factors, such as expectancy and demand characteristics, in achieving this pattern will be discussed below.

Methodological Considerations

Before discussing alternative explanations for the positive therapeutic outcome in this study, it is necessary to establish that the results were not simply due to a statistical artifact. One problem which affects studies using a pretest-posttest design is the risk of regression effects in the data. By using multivariate and univariate analyses of covariance the problem of a regression effect accounting for the observed outcome is removed. The observed results, therefore, may be attributable to an actual treatment effect.

Many of the studies examining the efficacy of behavioural treatments for sleep onset insomnia are conducted on small samples of insomniacs. This study is no different. Nine people per group is indeed a small sample. However, the results indicate that the sample size was not too small to find differences among the groups. With a larger sample, the results may have needed fewer qualifying statements.

Another category of concerns commonly found in treatment studies that rely on self-report data is the effect of demand characteristics on these data. Demand characteristics in an experimental situation, such as in this study, may lead participants to respond in a way that is unrelated to the experimental manipulation, but in a way that is in a direction they think will please the experimenter (Orne, 1969; Orne & Scheibe, 1964). It is difficult to control for the effect of demand characteristics. Steinmark and Borkovec's (1974) counterdemand paradigm alleviates the problem somewhat. However, this paradigm requires measuring the target behaviour during treatment in order to demonstrate the presence of therapeutic effects. If such effects are not necessarily found so soon, then it is difficult to evaluate effects of treatment unless followup data are collected.

Expectancy factors in this study may have influenced self-reported improvements (see Kazdin, 1980, for a review of this problem). Volunteers, who are solicited to participate in a treatment study examining an experimental therapy for sleep onset insomnia, are likely to generate an expectancy for improvement in sleep latency. Because the participants knew that this study was designed to examine the impact of treatment on sleep latency, they would likely develop the expectation that the treatment should help them to get to sleep more quickly. There would be less reason for them to develop a specific hypothesis about increases in total sleep time, although logically, such an hypothesis would follow. One might expect that treatment for any sleep problem would help improve sleep in general, including the duration, the quality, and the latency.

Demand characteristics and expectancy cannot be overlooked as factors potentially contributing to the apparent positive therapeutic outcome observed in this study. However, it is the nature of the pattern of results in this study that makes it particularly consistent with this interpretation. Indeed, any study in which all treatment groups have an effect and in which none of the treatment groups differ is highly susceptible to the interpretation that outcome is due, in some degree, to extraneous factors.

The Sleeper Effect

The observation that no significant improvements were noted in sleep latency prior to the twelve week Followup has been dubbed the "sleeper effect". The sleeper effect is an interesting phenomenon that could be accounted for by reference to the components of persistent psychophysiological insomnia (PPI) As we recall, PPI refers to a type of sleep problem in which a chronic, somatized tension-anxiety is coupled with a negative conditioning to internal or external factors leading to a prolonged sleep latency. If this definition describes the condition experienced by the participants in this study, then the relaxation treatment they received may have helped reduce the degree of somatized tension-anxiety that they experienced. Consequently a reduction in sleep latency would be expected after treatment. However, the degree of reduction may be related to several factors, such as practice and conditioning. First, practice. As the participants became more skilled at eliciting their own personal standard of relaxation, learned during treatment, they may have become more proficient at reducing the somatized tension-anxiety which was expected to be associated with their prolonged sleep latency.

The second factor related to the reduction of sleep latency may be conditioning. PPI has a negative conditioning component that suggests arousal is associated with the bed, bedtime thoughts, or bedtime routines. This arousal leads to a delay in sleep onset. If treatment reduces the tension-anxiety component of PPI, then, over time, the insomniac may develop a new association, one containing more relaxed, sleep-conducive features. This new association could replace the negative and arousing association that previously helped maintain the sleep problem. This two-step process of first reducing the somatized tension-anxiety and then changing the negative conditioning may take longer than four weeks but less than twelve. The results of this study showed that no significant reductions in sleep latency were found by one week or four weeks after treatment, but were present by twelve weeks. The two-step process might account for the observed delay in significant therapeutic outcome found in this study.

Alternative Theoretical Explanations for the Observed Reductions in Sleep Latency

All treatments led to some improvement in sleep latency by twelve weeks after treatment. Although the two-step process may explain the findings, there are alternative explanations.

One theoretically-based explanation of the positive psychotherapeutic changes is the effort justification paradigm (Cooper, 1980). It explains change in terms of the reduction of cognitive dissonance. If a client voluntarily takes part in a therapy procedure that requires considerable effort on the client's part, then the goal of the therapeutic intervention (in

the present case, latency reduction) becomes more attractive. The effortful therapy is assumed to arouse cognitive, emotional, or physical tension which can be reduced by achieving the desired goal. According to the effort justification hypothesis, any psychotherapy that involves significant participant effort and that is undertaken voluntarily would be likely to induce clinical improvement via the arousal and subsequent reduction of cognitive dissonance.

To the extent that participation in the present insomnia study was both effortful and voluntary, it is indeed possible that effort justification accounts for the therapeutic outcome. The affect and arousal data collected in this study, unfortunately, do not help clarify this explanation. Because we have no way of predicting when the arousal (which is predicted to lead to cognitive dissonance) is likely to arise, the likelihood of capturing changes which would demonstrate a reduction in cognitive dissonance is small. Further, it is not known whether the specific affect and arousal variables measured would reflect the type of cognitive, emotional, or physical tension hypothesized to be aroused and reduced in the effort justification paradigm. We, therefore, have no evidence for rejecting this paradigm as an alternative explanation for the outcome in this study.

In addition, the nature of the pattern of results in this study makes it difficult to rule out the effort justification explanation. All treatment groups showed a significant improvement; there were no differences among the groups; all treatments required the same amount of effort; and all were offered the same degree of choice. Any study resulting in a pattern

such as this would find it extremely difficult to reject the effort justification explanation of the outcome.

Another explanation for the outcome may be the effect of the instructions to try to remain awake during the treatment sessions. These instructions parallel those for the paradoxical intention paradigm. In that paradigm, insomniacs are asked to try to stay awake instead of trying to fall asleep. This switch in performance requirements is intended to relieve the anxiety created when the insomniac tries but fails to fall asleep. With a reduction in anxiety, the person should find it easier to fall asleep. Seven of the nine people in the autogenic relaxation condition reported falling asleep at least once during their four treatment sessions. This may have helped them reduce their anxiety about their ability to fall asleep in their own bed. However, only four of the 18 people who received some form of flotation REST treatment reported that they may have fallen asleep during the float. Therefore, the REST people were not learning to reduce their performance anxiety by falling asleep in a sleep-conducive environment. The fact that the treatment groups did not differ from each other in their ability to reduce sleep latency over the long term further suggests that the paradoxical intention paradigm is not an accurate explanation of the results.

Another possible explanation for the results is that latency decreased because the treatments led to reductions in negative affect and arousal prior to sleep. The reduction could have been achieved when attention was deliberately diverted from sleep-incompatible thoughts and their associated affect towards the pleasant, sleep-conducive images of personalized relaxation. These images were developed during treatment, as each

participant was establishing a mental and physical representation of his or her personal standard of relaxation, and were recalled at bedtime. Thus, sleep latency was decreased due to the redirecting of attentional focus from negative-toned wakeful mental activity to personalized, sleep-compatible sensations of relaxation. To determine the plausibility of this explanation, we must examine the results of the affect and arousal analyses.

The Role of Affect and Arousal

Affect and arousal variables were measured before, during, and after treatment, in an attempt to examine the effect of treatment on them and to look at their relation to sleep latency.

The results pertaining to the long-term effect of treatment on affect and arousal variables indicated that there were no significant differences among the groups twelve weeks after treatment (or twelve weeks after Baseline in the case of the control group) over and above those differences existing at Baseline. The long-term effect of treatment on these variables, therefore, was not significant.

Do these results imply that affect and arousal played no part in the maintenance or reduction of prolonged sleep latency? The results from the correlational analyses do not provide sufficient evidence to either confirm or disconfirm the role of affect and arousal. Of the 108 correlations calculated on the affect and arousal variables, only two were significant at the .005 level of significance. They were the correlation between (a) reduction in tension from Baseline to Followup and reduction in subjective sleep latency over the same period, and (b) reduction in anger from Baseline to Followup with reduction in objective sleep latency over the same period.

Because of the likelihood that at least one of the 108 correlations could be expected to be significant by chance (and only two were found to be significant -- as noted above), a great deal of weight should not be placed on these findings. With this caution in mind, the results from these correlational analyses are, at least, consistent with the idea expressed by a number of researchers (e.g., Borkovec, 1979, 1982; Coursey et al., 1975; Lichstein & Rosenthal, 1980) that anxiety-provoking, unpleasant, negative thoughts contributed to the origin and maintenance of sleep onset insomnia. Given that reductions in tension and anger are associated with reductions in sleep latency, it seems possible that the presence of negative affect and arousal at bedtime may be a mechanism that lengthens sleep latency. If these relations were dependable, then it would seem that even minimal reductions in negative affect can have a positive effect on sleep latency. The data in this study, however, do not provide sufficient evidence for a conclusion concerning the role of affect and arousal in prolonging sleep latency.

Affect and arousal during the treatment phase. The results in this study with respect to the immediate and direct effect of treatment on affect and arousal variables showed that only one arousal variable was significantly influenced by treatment. When measured right after the treatment session, the flotation REST groups were significantly more wakeful than was the autogenic relaxation group. This is consistent with the anecdotal reports of an alert but relaxed state in the REST individuals. These data however, do not indicate whether the state is due to the effects of

the flotation REST environment, to the showering necessary after floating, or to some combination of the two.

In earlier studies, a number of researchers looked at the relation of changes in physiological arousal (measured by muscle tension) that occurred during the treatment sessions to changes in sleep latency at the end of treatment (e.g., Borkovec et al., 1979; Coursey et al., 1980; Hauri, 1981). They failed to find any relation between reductions in physiological arousal during the daytime treatments and reductions in sleep latency by the end of treatment.

With respect to the relation between changes in affect and arousal during treatment and long-term sleep latency reduction, the results in this study were similar to those of earlier studies. No affect and arousal changes occurring right after the treatment sessions (which took place during the daytime) correlated with long-term reductions in sleep latency.

Perhaps the expectation that the immediate impact of treatment on either physical, cognitive, or emotional arousal should be directly related to the long-term outcome is unwarranted. Instead it may be more expedient to ask how changes in physical, cognitive, or emotional arousal over the long term are related to reductions in sleep latency over the long term. Evaluation of this relation between changes in various forms of arousal and sleep latency reductions may help identify the mechanisms underlying prolonged sleep latency.

Affect and arousal at baseline. Because psychophysiological insomnia is characterized by heightened somatized tension-anxiety, one might expect that variables measuring these characteristics would

correlate with sleep latency. In this study, there was no relation between scores on affect and arousal variables and Baseline sleep latency measures.

The correlations between the Baseline state and trait anxiety scores, measured during the daytime, and Baseline sleep latency were nonsignificant. Because all of the participants had an elevated anxiety level, the range of anxiety scores was limited. As a result of the range-restriction phenomenon the correlation between anxiety and sleep latency was constrained. However, the level of trait anxiety recorded in the participants during the day was significantly higher than the norm. This elevation is consistent with an expectation based on the definition that people with persistent, psychophysiological insomnia experience chronic, somatized tension-anxiety.

Taken together, these results do not offer much support to findings in some other studies (e.g., Beutler et al., 1978; Borkovec & Hennings, 1979, Van Egeren et al., 1983) which showed that presleep tension, anxiety, and dysphoric mood were positively related to the self-reported degree of sleep latency.

The Effect of Individual Differences in Circadian Rhythm on Sleep Latency

The results of this study showed that in addition to several of the affect and arousal variables, the degree of sleep latency experienced by persistent psychophysiological insomniacs is related to an individual difference in circadian rhythm (measured by the Morningness-Eveningness Questionnaire). The MEQ was designed to specify when a person felt his or her peak activity level occurred. People who experienced

their peak level of activity later in the day had objectively (but not subjectively) longer sleep latencies.

Thus, it may be that one of the factors underlying prolonged sleep latency is the individual's internal clock, which determines peak periods of wakefulness and sleep. If we try to go to sleep when our body is not ready, we may experience some difficulty because biologically we are still in an alert or wakeful phase. This is exactly the problem faced by people flying across time zones when travelling from west to east.

By practicing the relaxation technique learned during treatment, people with biological peaks later in the day may have been able to limit the degree of alertness and to decrease their sleep latency somewhat. Alternatively, people may have stopped trying to go to sleep according to the time on the clock on the wall. Instead they may have become more attuned to their internal clock and retired when they felt ready to go to sleep.

The Therapeutic Impact of REST

Restricted environmental stimulation techniques have had an impact on a number of behaviours. Chamber REST, for example, has been used to reduce smoking (Suedfeld & Baker-Brown, 1988) and drinking (Cooper et al., 1988). It has also been shown to help obese women lose weight (Borrie & Suedfeld, 1980). Further, pilot studies have shown chamber REST to be helpful in reducing blood pressure in essential hypertensives (e.g., Kristeller, Schwartz, & Black, 1982; Suedfeld, Roy, & Landon, 1982).

Flotation REST, as noted in the Introduction, has been successful in reducing blood pressure and the level of adrenal hormones in

hypertensives (Turner et al., 1987). This supports the results from an earlier pilot study on three essential hypertensives which found a reduction in blood pressure (Fine & Turner, 1982). Flotation REST has also been successful in inducing a state of deep objective and subjective relaxation (Jacobs et al., 1984).

Incidental Beneficial Effects

Although little has been reported on the unintended beneficial side effects of REST (in either form) there is some evidence that such effects do occur. Best and Suedfeld (1977), for example, reported five cases in which major lifestyle changes occurred that were attributed to treatment in a chamber REST smoking cessation programme. Among the beneficial effects were behaviour changes leading to improvements in perception of general health, and a personality change leading to appropriately assertive behaviour at home and at work.

In this study, eleven of the eighteen people who received flotation REST spontaneously reported some form of benefit after their treatment. These benefits included: acquiring the ability both to recognize the onset of tension during the day and to be able to reduce it by recalling the sensations experienced in the tank; taking up piano lessons after imagining playing the piano during a float (this person had wanted to learn to play throughout her adult life); perceiving a reduction of sinus pressure; deciding to seek professional advice and counselling concerning an intimate personal relationship; deciding to confront a family member over childhood abuse; changing a heavy-drinking lifestyle to one of moderation, even in the face of ridicule from drinking buddies; initiation of an active search for a new job;

significant attitude changes in one individual which led to a decrease in angry outbursts, a perceived increase in patience, and the development of an enterpreneurial idea.

Only two of the nine people in the autogenic relaxation condition spontaneously reported positive side effects of their treatment. For one person, the benefits were a continued motivation to make significant lifestyle changes (such as expanding cultural exposure to include attending good plays and movies), and thinking about starting a handyman business. For the other person, the benefit was the consideration of a major career change to accommodate a desire to work in a service position.

These ancedotal reports suggest that, in addition to the improvement in the target behaviour, flotation REST, like chamber REST, tends to provide unexpected benefits in other than the target area.

Gender Differences

The literature on the behavioural treatment of sleep onset insomnia has said little about gender differences. Survey data show that older women are more likely than men to report having serious insomnia (Mellinger et al., 1985), but this seems to be the extent of the references to gender differences in sleep patterns.

In this study there was only one gender difference in the sleep variables. Self-reported sleep latency at Followup was significantly longer for men (adjusted mean of 58.3 minutes) than for women (adjusted mean of 32.1 minutes). There were no gender differences on any other sleep variables. Nor were there any significant interactions between sex and either autogenic relaxation or flotation REST for sleep latency or for sleep

duration. Because there were no condition by gender interactions we may say that gender differences did not account for any of the observed main effects of treatment on sleep latency or sleep duration.

Men and women did not differ at Baseline in age, state or trait anxiety, daytime or nighttime depression, or peak activity time.

Future Research Directions

The results of this first attempt to use flotation REST to treat insomnia are encouraging. The expectation was that flotation REST would enhance the effects of autogenic relaxation training and if either of the REST treatments were to be more effective than the other, it would likely be REST and relaxation. The absence of a significant difference between the two flotation REST conditions suggests that adding therapeutic components does not always enhance the basic effect of the environment itself. Future research should replicate the current finding that flotation REST significantly decreases sleep latency. This research should also include a control group that is equated for the degree of effort and choice involved in participation. Thus, testing the effort justification explanation of therapeutic outcome in the presence of effort and choice.

A new line of thinking, concerning the possibility that flotation REST is an analogue of rapid eye movement (REM) sleep, was stimulated by the observation of a simple physical similarity between the two states. Both in REM sleep and while floating, the mind is very active, but the body is functionally immobile. Beside this physical similarity some of the cognitive effects of flotation REST (anecdotal reports from floaters) appear to parallel the type of imagery found in nighttime cycles of REM sleep (see Roffwarg,

Herman, Bowe-Anders, & Tauber, 1978). And further, an unpublished study at the University of British Columbia REST Laboratory found motor evidence suggesting that an hour of floating led to decreased differences in interhemispheric activity, which, in turn, implies increased interhemispheric synchrony. This is an exciting finding because increased interhemispheric EEG synchrony is a characteristic of REM sleep (e.g., Armitage, Hoffmann, Loewy, & Moffitt, 1988). This particular REST finding should be substantiated by electroencephalographic recordings. Future work in the UBC REST Laboratory is being planned to test the hypothesis that flotation REST is an analogue of REM sleep. Should this analogy hold, we may begin to ask some new questions about the nature of REM sleep and we may be able to develop a more complete theoretical explanation for the effects of flotation REST.

Summary

This study used flotation REST, autogenic relaxation, and a combination of the two, to treat people with persistent psychophysiological insomnia. All treatment conditions showed significant subjective and objective improvements in sleep latency twelve weeks after treatment. There were no significant improvements in sleep latency in the control condition. The three treatment groups increased their subjective sleep duration twelve weeks after the end of treatment, however, the autogenic relaxation group reported significantly longer sleep duration than either of the flotation REST groups. No significant increases were noted for objective sleep duration.

The only affect or arousal variable to change immediately after treatment was wakefulness. It increased significantly more for the REST groups than for the autogenic relaxation group. There were no significant differences in affect or arousal among the groups twelve weeks after treatment.

Reductions in subjective sleep latency were correlated with reductions in tension. Reductions in objective sleep latency were correlated with reductions in anger.

People whose peak activity level occurred in the late afternoon or evening took objectively longer to fall asleep at night than people who peaked earlier in the day.

Gender differences in sleep latency showed that men estimated that they took longer to fall asleep than women did. Objective measures of both sleep latency and sleep duration variables showed no differences between men and women.

Suggestions for future research include: testing the hypothesis that combining an intrusive technique with the basic flotation REST environment does not enhance and may, in fact, erode the REST effect; and testing the hypothesis that flotation REST is an analogue for REM sleep.

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Appendix A

Telephone Call-Back Script

PART 1

Hello, is _____ there please? Hello, this is _____ calling from the REST Lab at UBC. I am returning your call about the insomnia study. Do you have a few moments now to hear about it?

This experiment is part of my PhD research requirement. Being an experiment means that we are asking questions. The central one is: "What are the effects of various combinations of relaxation techniques on primary chronic insomnia?" It also means that we do not know the answer yet. We have good reason to believe that there will be some positive relation between what we're doing and sleep performance, but we won't know exactly what it is until we do the study.

Primary chronic insomnia refers to a particular sleep disorder. Primary means that the disorder is not the consequence or side effect of some physical or psychological condition. Chronic means that the problem has persisted over a long period of time, that is for months, and often for years. And insomnia means the inability to sleep. Our focus is on people who have trouble falling asleep, although these people may also have trouble staying asleep too.

We are specifically interested in the effects of various relaxation techniques on primary chronic insomnia. We are using two environments to test these techniques. One is a quiet, dimly lit room with a bed, intercom and speaker. The other is a flotation tank which is like a giant bathtub with a lid and a doorway in the lid. You will be assigned to receive treatment in one of these two environments. You will have no choice in your assignment because we are trying to assure that we have equivalent groups by randomly assigning people to treatments.

We know that many factors can bring about disturbances in sleeping patterns. So in order to evaluate the effect of these techniques on primary chronic insomnia, we need to minimize the impact of these factors as best we can. If I may then, I would now like to ask you some questions about these factors to determine your eligibility for this study.

1. How old are you?
2. a. Do you take any medication/therapy for a physical disease or syndrome?
   b. What is that medication/therapy?
3. a. Do you take any medication/therapy for a psychological/psychiatric disorder or syndrome?
   b. What is that medication/therapy?
4. Do you take any hypnotics, sedatives or sleep pills?
   a. What?
   b. For how long?
5. Do you work shift work?
   a. Which shift?

b. For how long have you done this?

6. a. How long does it usually take you to get to sleep?
   b. How often do you awaken during the night?
   c. How often in a 7-night period would you have a problem with sleep?

PART 2a - For Eligible Volunteers

You have met all of the initial criteria for inclusion in this study. The next step is for me to give you a broader sketch of the study, then if you are still interested, to send you a package of forms to be completed at home. The forms include a 14-page Sleep Assessment booklet that asks all about your sleep patterns. There is an answering guide to go along with it. Then there is a symptoms checklist, which is the front and back of one page. When they are completed you are to phone Dr. Fleming's receptionist, and make an appoint ment to see Dr. Fleming, who will go over your responses to these forms. He will also want to give you his own form and have a brief interview with you. There will be a cover letter, outlining all of this, included with the forms.

Dr. Fleming is one of my PhD research supervisors. He is also the director of the Sleep Disorders Clinic at Shaughnessy Hospital. He too is interested in the effects of non-pharmaceutical treatments on primary chronic insomnia.

If you meet the second stage of eligibility testing, then Dr. Fleming will tell me and I will call you to make an appointment for your first interview here in the REST Lab at UBC.

PART 2b - For Ineligible Volunteers

I am sorry to say that you do not meet the requirements for participation in this study. However, I can give you the phone number of the Sleep Disorders Clinic at Shaughnessy Hospital. Dr. Fleming, who is the director of this clinic, is also one of my research supervisors. You can have your family doctor refer you to the clinic. The telephone number is 875-2067.

PART 3

What will your participation involve? Your participation in this study will span about four months. Here is what is involved. There are seven steps.

Step 1: The Initial Interview

There is an initial interview with me at the REST Lab, which will take about 1 to 1 and 1/4 hours altogether. Baseline information will be collected. Dates for your treatment sessions will be worked out. You will be trained in the use of the SAD machine, which you will have to come and pick up and return a number of times throughout the study.

The SAD machine is a compact little machine with a tone generator and timing device connected to a cassette tape recorder. During the night a one-second tone will go off every 10 minutes, simultaneously the tape deck will record for 10 seconds. If you hear the tone your are to say something polite, like "I'm awake".

Because we have only 6 of these machines we are asking for a $50 refundable deposit . . . to encourage their prompt return for recycling.

Step Two (Baseline)

This step is the Baseline Sleep Monitoring step. For 14 nights you will be keeping a sleep log. This is a diary of your sleep behaviour. For the last 4 nights you will be asked to use a SAD machine at home to objectively monitor your sleep behaviour. There are also a few forms which you will be filling out just prior to going to bed.

Step Three (Treatment)

This is the treatment phase. You will have two treatment sessions per week for the next two weeks following your baseline period. Each session will take place in the morning and will last about 2 hours. There will be forms to be completed. Blood pressure and pulse readings will be taken as well.

In this experiment we are combining various techniques in unique ways to test if any combination has a superior effect. At the moment we don't know what the results will be. You will be randomly assigned to one of the groups.

Steps Four, Five and Six (Followup Assessments)

These three steps are identical in content. They are followups and they occur 1 week, 4 weeks and 12 weeks after your last treatment session. For each one you will be asked to keep a sleep log for 7 nights. Also on the last 3 of these nights you will be asked to use the SAD machine to take the objective measures. There will be forms to fill in on each of the last 3 nights.

Step Seven (Final Interview)

This is your final interview. After your last followup you will be asked to come in for a brief interview to discuss your progress, your views of the study, to answer any questions that are still remainng, to tell you about the rest of the groups and to return your $50 deposit. Because we don't know yet which treatment combination is going to have the best results we cannot offer you a chance at that treatment when you finish. Instead we will offer you an opportunity to try out one of the other treatments or to repeat your own. This is not part of the study and you need not do it.

This was a brief outline of the study. Are you still interested in taking part?

Appendix B

Reasons for Exclusion

After two levels of screening, 57 people qualified as having Persistent Psychophysiological Insomnia. Of those, 21 either dropped out of or were excluded from the study. Withdrawals occurred at five different levels of involvement:

1. Before the initial interview (3);
2. During Baseline and before assignment to treatment (11);
3. Upon assignment to treatment (1 - assigned to flotation REST);
4. During treatment (4 - all from REST conditions); and
5. During followup phase (2 - both from the autogenic relaxation only condition).

The reasons given for withdrawals in Levels 1 and 2 were unrelated to the unusual nature of the environments used in this study. They ranged from "We are moving" to "I broke my leg playing hockey", and from "Since I first called about this study I have been getting the best sleep I have ever had so I don't need to participate now" to "I feel good enough now just knowing that other people have the same problem I do that I don't think I want to take any treatment."

In Level 3, the participant decided that perhaps the tank seemed too small an enclosure for relaxation to occur. The people who withdrew during the Treatment Level were all in a REST condition, either with or without autogenic relaxation. Two people assigned to the REST and autogenic relaxation (RA) condition withdrew (one before the first treatment was scheduled and the other after postponing the first treatment) for personal reasons unrelated to the study that required their immediate

attention, and the third person in the RA condition withdrew before his final treatment when he found himself requiring medication for a psychological problem he had been ignoring. One person in the REST only (R) condition withdrew three-quarters of the way through his first treatment because he did not like the darkness, dampness and quietness of the tank nor the institutional fixtures and tiles of the washroom facilities.

Two people withdrew during the Followups. One withdrew because she felt that she was not "getting any use out of the followups". The other person withdrew because a change in his work schedule required him to travel on short notice for long periods of time, thus interfering with the regular pickup and return of the Sleep Assessment Device, questionnaires and Sleep Log.

Regardless of the level at which they withdrew, all people who had placed a $50 deposit on the Sleep Assessment Device received their refund immediately upon stating their decision.

Appendix C

SLEEP ASSESSMENT SCHEDULE

INTRODUCTION :

The questions enclosed in this booklet will help in obtaining an understanding of your sleeping problem. It is very important that you answer each question as completely and as accurately as you can.

If necessary you can ask someone who knows you well to help answer some of the questions.

A FEW GUIDELINES :

1. Do not spend too much time on any question. Your first impression is generally the best.

2. The time period of all the questions is THE PRESENT - which includes the last SIX MONTHS unless otherwise specified.

3. A weekday is any day on which you normally work outside the home.

4. If you are engaged in shift work or have any type of unusual sleep/ wake schedule then daytime and nighttime refer to your own major waking and sleeping periods.

REMEMBER :

1. To keep the RESPONSE SHEET examples alongside you to remind you how to answer the questions.

2. The questions are on BOTH sides of the paper.

PLEASE note the time. We are interested in knowing how long it took you to complete this questionnaire. Thank you.

Now you are ready to answer the questions, so please turn to the next section.

I. Please describe, briefly, your current sleep complaint/problem :

_____

_____

_____

_____

_____

a. Roughly, when did your current sleep problem begin (month, year)? _____19____

b. Since it began has it been a CONTINUOUS problem? ____ yes ____ no

c. At this time, are you taking ANYTHING to help you sleep? ____ yes ____ no

d. Has the problem that you have NOW been WORSE at any other time? ____ yes ____ no

e. Do you feel that you have insomnia? ____ yes ____ no

f. In the last MONTH, what was the AVERAGE number of hours of sleep obtained on regular WEEKDAYS or WORKDAYS?
_____ hours

g. In the last MONTH, what was the AVERAGE number of hours of sleep obtained on WEEKEND or NON-WORKDAYS?
_____ hours

II. During the last week, please indicate the AVERAGE :

a. Time of getting into bed _____ am pm (please circle am or pm)

b. Time of attempting to go to sleep _____ am pm (please circle am or pm)

c. Time between deciding to go to sleep and sleep onset _____ minutes

d. Number of awakenings before your final awakening _____ Average duration of awakenings ____ minutes

e. Duration of LONGEST awakening _____ minutes

f. How many times do you usually get out of bed in a night? _____ times

go on to page 2 . . .

g. If you awaken during the night which third of the night is it usually in?
[ ] first third
[ ] second third
[ ] last third

h. Time of final awakening _____ am pm (please circle am or pm)

i. Time of finally leaving bed _____ am pm (please circle am or pm)

j. On finally awakening, how long does it take you to feel FULLY alert?

_____ minutes

k. How often do you feel extremely alert and energetic during the whole day? 1 2 3 4 5

l. How well do you function in the :

- morning 1 2 3 4 5
- midday 1 2 3 4 5
- afternoon 1 2 3 4 5
- evening 1 2 3 4 5

III. HOW GREAT A PROBLEM DO YOU HAVE :

a. -with getting to sleep at night? 1 2 3 4 5

b. -with waking up during the night? 1 2 3 4 5

c. -with waking up and getting going in the morning? 1 2 3 4 5

d. -with non-restorative sleep (ie, no matter how much sleep you get, you don't wake up feeling rested/refreshed)? 1 2 3 4 5

e. -with SLEEPINESS (feeling sleepy or struggling to stay awake) in the day? 1 2 3 4 5

f. -with FATIGUE (tiredness, exhaustion WITHOUT feeling sleepy)? 1 2 3 4 5

g. What time do you usually GO TO bed on weekends?
_____ am pm (circle am/pm)

h. What time do you usually GET UP on weekends?
_____ am pm (circle am/pm)

go on to page 3 . . .

IV. Before FALLING OFF to sleep or after an awakening HOW OFTEN do you?

a,	- have thoughts racing through your mind?	1	2	3	4	5
b.	- feel sad and depressed?	1	2	3	4	5
c.	- feel anxious?	1	2	3	4	5
d.	- worry about things?	1	2	3	4	5
e.	- feel tension in your muscles?	1	2	3	4	5
f.	- feel afraid of not being able to get to sleep?	1	2	3	4	5
g.	- fear the dark?	1	2	3	4	5
h.	- fear going to sleep?	1	2	3	4	5
i.	- feel unable to move (paralyzed)?	1	2	3	4	5
j.	- notice that parts of your body startle or jerk?	1	2	3	4	5
k.	- experience restless legs (crawling or aching feelings and inability to keep your legs still)?	1	2	3	4	5
l.	- experience vivid, dream-like scenes (hallucinations) even though you know you are awake?	1	2	3	4	5
m.	- experience any kind of pain or physical discomfort?	1	2	3	4	5
n.	- feel wide awake?	1	2	3	4	5
o.	- feel panicky or very anxious?	1	2	3	4	5
p.	- feel distracted/disturbed by some environmental factor (noise, heat etc)?	1	2	3	4	5
q.	- feel worried about the stresses of the next day?	1	2	3	4	5
r.	- have worries about death or dying?	1	2	3	4	5
s.	- feel that time is speeded up and passing you by?	1	2	3	4	5
t.	- feel that you can't turn your mind off?	1	2	3	4	5

go on to page 4 . . .

V. A VARIETY OF BODILY COMPLAINTS CAN DISTURB YOUR SLEEP SO IT IS IMPORTANT TO KNOW HOW OFTEN YOUR SLEEP IS DISTURBED BY :

a.	- asthma?	1 2 3 4 5
b.	- a persistent cough?	1 2 3 4 5
c.	- being unable to breath in a flat position because of shortness of breath?	1 2 3 4 5
d.	- "gas" in your stomach, indigestion or heartburn	1 2 3 4 5
e.	- regurgitation, burning in throat or gagging on stomach contents?	1 2 3 4 5
f.	- being hungry?	1 2 3 4 5
g.	- being thirsty?	1 2 3 4 5
h.	- having an urgent desire to urinate?	1 2 3 4 5
i.	- intense heart pain?	1 2 3 4 5
j.	- other chest pain?	1 2 3 4 5
k.	- stomach or abdominal pains?	1 2 3 4 5
l.	- restless legs?	1 2 3 4 5
m.	- leg cramps (charley horses)?	1 2 3 4 5
n.	- "pins and needles" in your arms or legs	1 2 3 4 5
o.	- itching?	1 2 3 4 5
p.	- pain or discomfort?	1 2 3 4 5
q.	- heart beating very fast?	1 2 3 4 5
r.	- muscular tension?	1 2 3 4 5
s.	- frightening dreams (nightmares)?	1 2 3 4 5
t.	- racing thoughts during your sleep?	1 2 3 4 5
u.	- a recurring dream/nightmare?	1 2 3 4 5
v.	- other problems not mentioned above?	1 2 3 4 5

go on to page 5 . . .

VI. SOME COMPLAINTS APPEAR OR GET WORSE DURING SLEEP, TO YOUR KNOWLEDGE, HOW OFTEN DO YOU :

a.	- walk in your sleep?	1	2	3	4	5
b.	- talk in your sleep?	1	2	3	4	5
c.	- grind your teeth during your sleep?	1	2	3	4	5
d.	- bang your head (on pillow, bed or wall) during your sleep?	1	2	3	4	5
e.	- make rocking or rolling movements during your sleep?	1	2	3	4	5
f.	- fall out of bed whilst asleep?	1	2	3	4	5
g.	- awaken from sleep screaming, confused or violent (night terrors)?	1	2	3	4	5
h.	- wet your bed during your sleep?	1	2	3	4	5
i.	- your legs twitch during your sleep?	1	2	3	4	5
j.	- have convulsions (fit, seizure or epilepsy) during your sleep?	1	2	3	4	5
k.	- have unusual and pronounced body movements during your sleep?	1	2	3	4	5
l.	- have headaches?	1	2	3	4	5
m.	- have pain in your neck, back, spine, muscles, joints, arms or legs?	1	2	3	4	5

VII. YOUR SLEEP ENVIRONMENT

When you go to bed DO YOU ever? : [CHECK THOSE THAT APPLY]

a.	- wear ear plugs?	[ ]
b.	- wear eye shades?	[ ]
c.	- go to sleep with music or some other sound?	[ ]
d.	- go to sleep with the light on?	[ ]
e.	- use some special routine or ritual?	[ ]
f.	- sleep with someone else in the room?	[ ]
g.	- sleep with someone else in your bed?	[ ]
h.	- sleep on a special surface (bed board, orthopedic mattress, floor, etc.)?	[ ]
i.	- disturb the sleep of your bed partner?	[ ]
j.	- provide assistance or attention to someone (child, invalid, bed-partner) during the night?	[ ]
k.	- provide attention to something else (a pet, hobby or project)	[ ]

go on to page 6 . . .

VIII. SOME SLEEP PATTERNS OR HABITS BECOME ESTABLISHED IN CHILDHOOD. AS A CHILD DID YOU HAVE A PROBLEM WITH :

[CHECK THOSE THAT APPLY]

a.	- getting to sleep at night?	[ ]
b.	- waking up during the night?	[ ]
c.	- waking up and getting going in the morning?	[ ]
d.	- feeling unrestored by your sleep, no matter how much you got?	[ ]
e.	- sleepiness during the day?	[ ]
f.	- fatigue during the day?	[ ]
g.	- thumb sucking?	[ ]
h.	- rocking yourself to sleep?	[ ]
i.	- head-banging?	[ ]
j.	- bed wetting?	[ ]
k.	- sleep talking?	[ ]
l.	- sleep walking?	[ ]
m.	- nightmares?	[ ]
n.	- awakening from sleep screaming?	[ ]
o.	- convulsions during your sleep?	[ ]
p.	- fear of the dark?	[ ]
q.	- fear of sleep?	[ ]
r.	- grinding your teeth when asleep?	[ ]
s.	- hyperactivity?	[ ]

IX. During the past SIX MONTHS have you had either spontaneous episodes of falling asleep WITHOUT INTENDING TO or SEVERE sleepiness WITHOUT actually falling asleep in any of the following situations :

[CHECK ONLY THOSE BOXES THAT APPLY]

		SPONTANEOUSLY FALLING ASLEEP	FIGHTING SLEEP
a.	- at meals?	[ ]	[ ]
b.	- on the telephone?	[ ]	[ ]
c.	- in conversation with another person at work?	[ ]	[ ]
d.	- in conversation with any person?	[ ]	[ ]
e.	- talking in a group?	[ ]	[ ]
f.	- travelling (car, bus, train etc.)	[ ]	[ ]
g.	- attending a performance (theatre, movie etc)?	[ ]	[ ]
h.	- watching television or listening to the radio or stereo?	[ ]	[ ]
i.	- sitting and reading?	[ ]	[ ]
j.	- during love making?	[ ]	[ ]

- how many naps (actually falling asleep for 5 minutes or more) do you take ON PURPOSE during a usual weekday?

____ naps

go on to page 7 . . .

- how many rest periods (where you lie down BUT DO NOT SLEEP) do you take in a usual weekday?

____ rests

- how many times, in a usual weekday, do you try to take a nap but can't fall asleep?

____ times

X. Please list ALL current medications (including birth control pills, aspirin, vitamins and over-the-counter-medications) :

NAME	DOSAGE PER DAY	DATE STARTED
_____	_____	_____ mth ____ year
_____	_____	_____ mth ____ year
_____	_____	_____ mth ____ year
_____	_____	_____ mth ____ year
_____	_____	_____ mth ____ year
_____	_____	_____ mth ____ year

XI. If you have had a long-standing difficulty with your sleep, it is likely that you have taken medications to help you sleep. If you HAVE taken any medications to help you sleep please list them here. If you HAVE NOT taken any sleeping pills please go on to the next section :

DRUG NAME?	TAKEN FOR HOW LONG?	HOW EFFECTIVE WAS IT?
_____	_____ weeks	1 2 3 4 5
_____	_____ weeks	1 2 3 4 5
_____	_____ weeks	1 2 3 4 5
_____	_____ weeks	1 2 3 4 5

go on to page 8 . . .

XII. How much of these fluids do you drink :

A.	IN 24 HOURS?	WITHIN 3 HOURS BEFORE BEDTIME?	DURING THE NIGHT?
- coffee	_____ cups	_____ cups	_____ cups
- tea	_____ cups	_____ cups	_____ cups
- other fluid	_____ cups	_____ cups	_____ cups
- cola drinks	_____ bottles	_____ bottles	_____ bottles

How many alcoholic drinks do you have during a usual 24 hour period?

	WEEKDAY	WEEKEND DAY
- beer	_____ bottles/cans	_____ bottles/cans
- wine	_____ glasses	_____ glasses
- liquor	_____ shots	_____ shots

B. HAVE YOU EVER been a smoker? ___ yes ___ no

- for how many years were you a smoker? _____ years
- how many cigarettes do you smoke NOW? _____ packs
- how many cigarettes did you smoke 5 years ago? _____ packs

HOW OFTEN :

- have you used tobacco within two hours of going to sleep? 1 2 3 4 5

C. HOW OFTEN HAVE YOU USED THE FOLLOWING NON-DRUG TECHNIQUES TO HELP YOU SLEEP?

- hypnosis 1 2 3 4 5
- relaxation exercises 1 2 3 4 5
- biofeedback 1 2 3 4 5
- other techniques 1 2 3 4 5

go on to page 9 . . .

What is your personal interpretation as to why you have your particular sleep problem?

_____

_____

_____

_____

_____

If your sleep/wake behaviour is not adequately covered by the above questions, briefly describe the nature of your sleep/wake behaviour and list anything else that has not been covered which you think would help in understanding you or your sleep complaint:

_____

_____

_____

Please state as specifically as possible, what you hope to achieve in your participation in the Primary Insomnia Research Project:

_____

_____

_____

_____

Time taken to complete questionnaire: _____ minutes.

Thank you very much for taking time and care to complete this. Please check through the questionnaire to see that you have answered all the questions ON BOTH SIDES of the paper.

KEEP THIS SHEET ALONGSIDE YOU
AS YOU ANSWER THE QUESTIONS

PROCEDURE FOR ANSWERING DIFFERENT TYPES OF QUESTIONS

1. Degree Type

1	2	3	4	5
none (not at all)	slight	moderate	considerable	very great (a lot)

Example: How great a problem do you have with paying your bills? 1 2 3 4 5

2. Frequency Type

1	2	3	4	5
never	just a few times	sometimes	quite often	usually (always or almost always)

Example: How often have you been to the North Pole? 1 2 3 4 5

3. Yes/No Type

Example: Have you ever filled out this questionnaire before? __ yes __ no

4. Box Type [ ] Check only those boxes that apply
- leave all other boxes blank

Example: Have you ever eaten any of the following foods?

	for breakfast	for lunch	for dinner
cereal	[ ]	[ ]	[ ]
pizza	[ ]	[ ]	[ ]
octopus	[ ]	[ ]	[ ]
fruit	[ ]	[ ]	[ ]

5. X Response

Place an X beside any question that you do not understand, or which does not apply to you, or which cannot be given a valid answer, or which you choose not to answer.

Example: Have you ever had Campbell's disease of the uterus? __ yes __no

Person may respond with an X for several reasons:
(a) The person is a male.
(b) The person does not know what Campbell's disease is.
(c) The person does not wish to answer this question.

6. Literal response

Example: What time do you usually go to work? _____ am/pm

Appendix D

DEPARTMENT OF PSYCHOLOGY

THE UNIVERSITY OF BRITISH COLUMBIA

Basic Rights and Privileges of Research Participants

The person who volunteers to particpate in experiments conducted by full or part-time members of the faculty of the Department of Psychology at the University of British Columbia, by their employees, or by the graduate and undergraduate students working under the direction of faculty members of the above named Department, is entitled to the following rights and privileges.

1. The participant may terminate and withdraw from the experiment at any time without being accountable for the reasons for such an action.

2. The participant shall be informed, prior to the beginning of an experiment, of the maximum length of time the experiment might take and of the general nature of the experiment.

3. The participant shall be informed, prior to the beginning of an experiment, of the nature and function of any mechanical and electrical equipment which is to be used in the experiment. In cases where subjects are in direct contact with such equipment, they shall be informed of the safety measures designed to protect them from physical injury, regardless of how slight the possibility of such injury is.

4. Participants shall be informed, prior to the beginning of an experiment, of the aspects of their behaviour that are to be observed and recorded and how this is to be done.

5. Any behavioural record that is obtained during the course of the experiment is confidential. Any behavioural records that are made public through either journal papers or books, public addresses, research colloquia, or classroom presentations for teaching purposes, shall be anonymous.

6. The participant shall be offered, at the end of an experiment, a complete explanation of the purpose of the experiment, either orally by the experimenter or, at the option of the experimenter, in writing. The participant shall also have the opportunity to ask questions pertaining to the experiment and shall be entitled to have these questions answered.

7. The participant has the right to inform the Chairman of the Departmental Committee on Research with Human Subjects of any perceived violations of, or questions about, the aforementioned rights and privileges.

(Flotation) CODE:_____

Consent Form

As a volunteer for this study, you should know what this part of the project is going to be like. You will spend up to one hour floating in warm, salt water in a dark, quiet flotation tank. You are instructed to remain reasonably quiet while in the water. That means you are to avoid whistling, singing, talking to yourself, and the like. You are also asked to refrain from excessive movement while you are floating.

Before and after the flotation session you will be asked to shower thoroughly and to wash your hair. Earplugs will be supplied for use during your float. At all times when you are in the tank, there will be a monitor next door who will listen through the intercom to make sure that you are all right and that you are not moving around too much or talking to yourself and so on. From time to time you may be asked questions about how you are feeling.

Flotation tanks are used commercially for relaxation. Research indicates that most floaters have found the experience very pleasant. However, if you should find the situation unpleasant, you may end the experiment simply by notifying the monitor over the intercom and then stepping out of the tank. Doing this does not reflect upon you in any way. Some people just find such a situation unappealing and there is no particular reason why they should force themselves to continue in it.

Dr. Peter Suedfeld
Project Director

I have received and read a copy of the above and agree to participate in the project described.

Date: _____ Signature: _____

Printed Name: _____ Birth Date:_____

Address: _____ Phone Number: _____

_____ Code: _____

(F & R) CODE:_____

Consent Form

As a volunteer for this study, you should know what this part of the project is going to be like. You will spend up to one hour floating in warm, salt water in a dark, quiet flotation tank. You are instructed to remain reasonably quiet while in the water. That means you are to avoid whistling, singing, talking to yourself, and the like. You are also asked to refrain from excessive movement while you are floating.

Before and after the flotation session you will be asked to shower thoroughly and to wash your hair. Earplugs will be supplied for use during your float. At all times when you are in the tank, there will be a monitor next door who will listen through the intercom to make sure that you are all right and that you are not moving around to much or talking to yourself and so on. From time to time you may be asked questions about how you are feeling.

Shortly after your float begins a guided-relaxation exercise will be played over the speakers. You are to follow the instructions as well as you can.

Flotation tanks are used commercially for relaxation. Research indicates that most floaters have found the experience very pleasant. However, if you should find the situation unpleasant, you may end the experiment simply by notifying the monitor over the intercom and then stepping out of the tank. Doing this does not reflect upon you in any way. Some people just find such a situation unappealing and there is no particular reason why they should force themselves to continue in it.

Dr. Peter Suedfeld
Project Director

I have received and read a copy of the above and agree to participate in the project described.

Date: _____ Signature: _____

Printed Name: _____ Birth Date:_____

Address: _____ Phone Number: _____

_____ Code: _____

(RX) CODE:_____

Consent Form

As a volunteer for this study, you should know what this part of the project is going to be like. You will spend up to one hour reclining on a bed in a closed, quiet, softly lit room. Shortly after you enter the room and lie down, a guided-relaxation exercise will be played over the speakers in the room. You are to follow the instructions as well as you can.

When you are in the quiet room, there will be a monitor next door who can hear you over the intercom. You are asked to make yourself as comfortable as possible on the bed while avoiding unnecessary movement or noise.

Should you at any time decide not to continue your participation, you can end the experiment by informing the monitor of your intention and then walking out of the room. Doing this does not reflect on you in any way. Some people just fund such a situation unappealing and there is no particular reason why they should force themselves to continue in it.

Dr. Peter Suedfeld
Project Director

I have received and read a copy of the above and agree to participate in the project as described.

Date: _____ Signature: _____

Printed Name: _____ Birth Date: _____

Address: _____ Phone Number:_____

_____ Code: _____

Appendix E

SLEEP HYGIENE

Many people with sleep disturbances have poor sleep habits. Correction of these habits goes a long way towards removing the sleep problem.

Here are some recommendations which could help improve your sleep.

1. Avoid taking naps. Avoid daytime sleep. Eliminate any sleep beyond normal nocturnal sleep.

2. Do not stay in bed more than ten minutes before falling asleep. The association between worrying, tossing and turning, not sleeping etc. and lying in bed tends to discourage the association of bed with rest. By regularly getting out of bed if sleep does not occur within ten minutes, the association adverse to good sleep patterns can be broken.

3. Only go to bed when tired. As in the second recommendation, you should try to discourage the association between cognitive activities while lying in bed and the bed itself. Once the body's natural circadian rhythm is re-established, the discrepancy between the time you go to bed and subsequently fall asleep will decrease.

4. Select a boring or dutiful activity and do it until tired enough to return to bed. Interesting, stimulating activities tend to maintain arousal and alertness. Repetitious and unexciting ones reduce arousal and promote rest and relaxation.

5. While in bed, count backwards from 500. Continue this sequence until asleep. Again, such repetitious mental activity tends to elicit a state of relaxation that aids sleep. In addition, counting backwards interferes with stimulating mental activities and troublesome thoughts that cannot be "willed" away. Rather, they can be replaced with repetitious, monotonous cognitive activity.

6. Get up at the same time of day each day regardless of the amount of sleep obtained. By entraining the sleep-wake cycle to a fixed pattern, the body's circadian rhythm can be reset. While doing this may initially lead to feelings of increased tiredness, such feelings may result in future improved sleep.

7. Do as much exercise as possible. Exercise is a very effective tranquilizer and can therefore reduce distress and emotional disturbances. While dealing with the problems and stressors directly is an obvious way of reducing stress-related insomnia, sleep disturbance can interfere with a healthy response to stressors and emotional problems. Excerise promotes three beneficial components of this cycle: good sleep, stress reduction, and emotional health. Moreover, physical exercise allows the body to more easily distinguish between awake and sleep settings. Such a distinction becomes more difficult during extended periods of inactivity. But remember, avoid heavy exercise just before bedtime as such exertion is very arousing.

. . . 2

8. Monitor and reduce caffeine intake. Caffeine found in coffee, cocoa, cola beverages and tea can have a profound effect on arousal levels. Wide individual differences exist in tolerance of caffeine and many people may be extremely sensitive to even small amounts. If coffee must be consumed, attempts should be made to not drink it after four p.m. and to drink decaffeinated coffee whenever possible. Moreover, nicotine found in tobacco is a stimulant and especially when coupled with coffee may be a major impediment to the relaxation necessary for sleep to occur.

9. Do not drink alcohol after supper. It will interfer with the quality of your sleep.

10 Allow yourself time to wind down from the day's physical and mental activities. Going to bed directly after a stimulating day or evening tends to strengthen the association of the bed with physical or mental arousal. Try engaging in quiet, relaxing activities immediately before bedtime --- soak in a hot bath, read, listen to soft, calming music.

11. Use the bedroom for sleeping (and recreation). Use some other part of the house for homework, billpaying, family discussions, TV watching. Try to develop the association of rest with the bedroom as well as with the bed.

12. Control your sleep environment. Make the bedroom as quiet as possible. Shut the door, turn down the phone bell, replace a noisy ticking clock with a quiet one. People with sleep disturbances may be extra sensitive to environmental sounds.

    Keep the room dark since light tends to signal to our body that it is time to become alert. Use heavy drapes or good blinds. Also keep the room on the cool side since we tend to sleep better in such an environment than in a warm, possibly stuffy room. Use an extra blanket or a quilt to keep yourself warm.

As with most attempts to change habits, conscientious implementation of appropriate recommendations is essential if you expect to see any improvements.

Appendix F

Sleep Latency: Unadjusted Means and Standard Deviations

Condition[a]		Baseline	Posttest 1-Week	Posttest 4-Weeks	Followup
		Subjective Sleep Latency (minutes)			
REST Only	M	69.0	46.8	42.5	31.5
	SD	26.5	48.9	43.9	22.7
REST and Relaxation	M	79.3	54.7	64.6	48.9
	SD	45.4	36.6	48.6	35.9
Autogenic Relaxation	M	62.9	44.5	47.1	39.2
	SD	29.4	21.6	35.2	24.3
Control	M	57.1	-	-	55.3
	SD	25.4	-	-	37.6
		Objective Sleep Latency (minutes)			
REST Only	M	65.3	53.3	34.4	26.5
	SD	30.3	68.8	42.4	15.1
REST and Relaxation	M	40.6	28.4	23.1	28.9
	SD	18.5	14.8	15.3	15.6
Autogenic Relaxation	M	42.6	41.4	41.5	28.7
	SD	16.4	39.9	32.7	13.7
Control	M	51.6	-	-	57.5
	SD	37.1	-	-	42.9

[a] n = 9 per group

Appendix G

Sleep Duration: Unadjusted Means and Standard Deviations

Condition[a]		Baseline	Posttest		Followup
			1-Week	4-Weeks	
		Subjective Duration (minutes)			
REST Only	M	372.5	381.5	407.9	391.3
	SD	76.4	71.4	65.8	57.6
REST and Relaxation	M	323.0	363.9	371.3	377.6
	SD	54.8	46.4	59.3	66.5
Autogenic Relaxation	M	354.8	361.7	394.5	420.8
	SD	61.9	61.4	51.8	39.9
Control	M	334.6	-	-	326.8
	SD	84.1	-	-	72.3
		Objective Duration (minutes)			
REST Only	M	301.9	347.0	351.7	369.4
	SD	103.1	132.3	121.6	89.6
REST and Relaxation	M	351.1	352.4	388.9	396.1
	SD	72.9	58.7	76.6	57.1
Autogenic Relaxation	M	372.2	356.3	388.3	414.4
	SD	56.9	55.1	60.4	43.9
Control	M	282.3	-	-	328.3
	SD	86.9	-	-	49.2

[a] n = 9 per group

Appendix H

Summary Tables for Analysis of Covariance on Sleep Latency Means for 1-Week Posttest with Baseline as the Covariate

Source	Sum of Squares	*df*	Mean Squares	*F*	*p*
		Subjective Sleep Latency			
Covariate	1726.003	1	1726.003	23.774	0.000
Main Effect Condition	33.023	2	16.511	0.023	0.978
Residual	16713.766	23	726.685		
		Objective Sleep Latency			
Covariate	9111.454	1	9111.454	4.611	0.043
Main Effect Condition	601.441	2	300.720	0.152	0.860
Residual	45444.652	23	1975.854		

Appendix I

Summary Tables for Analysis of Covariance on Sleep Latency Means for 4-Weeks Posttest with Baseline as the Covariate

Source	Sum of Squares	*df*	Mean Squares	*F*	*p*
			Subjective Sleep Latency		
Covariate	31959.089	1	31959.089	52.681	0.000
Main Effect Condition	747.906	2	373.953	0.616	0.549
Residual	13953.092	23	606.656		
			Objective Sleep Latency		
Covariate	3197.082	1	3197.082	3.460	0.076
Main Effect Condition	1913.635	2	956.817	1.036	0.371
Residual	21250.693	23	923.943		

Appendix J

Summary Table for Analysis of Covariance on Beck Depression Inventory Means at Followup with Baseline as Covariate

Source	Sum of Squares	*df*	Mean Squares	*F*	*p*
Covariate	201.387	1	201.387	10.01	0.005
Main Effects					
REST[a]	11.499	1	11.499	0.572	0.458
AUTO[b]	24.733	1	24.733	1.229	0.281
Interaction					
REST by AUTO	0.118	1	0.118	0.006	0.940
Residual	402.362	20	20.118		

[a] REST includes the REST only and REST and autogenic relaxation conditions

[b] AUTO includes autogenic relaxation and REST and autogenic relaxtion conditions

Appendix K
Summary Tables for Analysis of Covariance on Self-Report Arousal Scales with Baseline as the Covariate

Source	Sum of Squares	*df*	Mean Squares	*F*	*p*
			Wakefulness		
Covariate	39.330	1	39.330	22.299	0.000
Main Effect Condition	36.859	2	18.429	10.449	0.001
Residual	40.566	23	1.764		
			Energy		
Covariate	151.892	1	151.892	18.625	0.000
Main Effect Condition	6.345	2	3.172	.389	0.682
Residual	187.566	23	8.155		
			Stress		
Covariate	36.385	1	36.385	9.164	0.006
Main Effect Condition	0.822	2	0.411	0.104	0.902
Residual	91.317	23	3.970		
			Euphoria		
Covariate	128.048	1	128.048	41.825	0.000
Main Effect Condition	9.941	2	4.971	1.624	0.219
Residual	70.415	23	3.062		

Appendix K - continued

Summary Tables for Analysis of Covariance on Self-Report Arousal Scales with Baseline as the Covariate

			Irritation		
Covariate	3.962	1	3.962	9.456	0.005
Main Effect Condition	0.649	2	0.774	0.774	0.473
Residual	9.638	23	3.668		
			Concentration		
Covariate	15.423	1	15.423	7.210	0.013
Main Effect Condition	1.058	2	0.529	0.247	0.783
Residual	49.197	23	2.139		