

EFFECTS OF THERAPIST CONTACT  
AND A SELF-HELP MANUAL IN THE TREATMENT  
OF SLEEP-ONSET INSOMNIA

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# ABSTRACT

## Effects of Therapist Contact and a Self-Help Manual in the Treatment of Sleep-Onset Insomnia

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The study evaluated a commercially available self-help treatment manual for insomnia. Thirty adult subjects complaining of problems getting to sleep (sleep-onset insomnia) were given a copy of a manual containing general knowledge about sleep and instructions for a variety of behavioural self-control techniques. These subjects were randomly assigned to one of three treatment conditions which differed in the type of contact they had with the therapist. In one condition (group contact) the subjects met as a group with the therapist for a weekly meeting. In the other conditions the subjects either received a brief weekly phone call (minimal contact) or totally self-administered the manual (self-directed). The results show that the two month bibliotherapy program was effective for the subjects who had some contact with the therapist. The group which totally self-administered the manual did not exhibit significant reductions in the time it took them to fall asleep. The two therapist contact conditions achieved significant decreases in the time it took the subjects to fall asleep and the difficulty they experienced in falling asleep. In addition, they significantly increased the number of hours they slept each night. Elevated levels of self-esteem occurred for the group which

personally met with the therapist: Two variables emerged which were favorable prognostic signs with this treatment: female gender and scores on the social introversion scale of the MMPI. Although subjects in all conditions voiced a satisfaction with the program, the results suggest that therapist consultation may be necessary for significant improvements in sleep latency to occur with the usage of a behavioural self-help manual. Furthermore, the high correspondence between the subjective and objective indices of the sleep disturbance observed in the present study suggests that these conclusions may apply to both of these aspects of the problem.

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## Introduction

The term insomnia is used to refer to a wide range of disturbances of sleep function, arising from a myriad of sources. While etiology is one scheme which may be used to classify insomnia, it has become customary practice to diagnose it in accordance with any or all of the three periods of the night when it occurs: at the onset, when one has difficulties falling asleep; after sleep onset, when there are frequent and/or prolonged awakenings; and in the morning, when arousal occurs very early (Karacan, Williams, Salis & Hirsch, 1971). Such complaints have brought patients to their physicians since the time of Hippocrates (circa 469-377). Recent epidemiological studies highlight the magnitude of this problem in the general population.

An inability to obtain adequate sleep was the primary presenting concern of 20% of the patients of physicians canvassed throughout the United States (Bixler, Kales, Scharf, Kales & Leo, 1976). Thirty-two percent of the sample of adults surveyed in Los Angeles (Kales, Bixler, Leo, Healy & Slye, 1974) and thirty-five percent of an urban Florida county sample (Karacan, Thornby, Anch, Holzer, Worheit, Schwab & Williams, 1976) said they were afflicted with either mild or severe insomnia. A similar incidence of insomnia within the general population has been reported in Scotland (McGhie & Russell, 1962) and Australia (Krupinski & Stoller, 1971). These surveys also found trends similar to those in America for the distribution within age and sex categories. Complaints of sleep dysfunction show a gradual but steady increase with age, with women, especially over 50, more likely to complain of

this difficulty than men (Prinz & Raskind, 1978).

Other findings in these surveys have clarified the incidence of different kinds of insomnia. Only one study (Kales et al., 1974) found frequent awakenings to be the most prevalent type of sleep problem. Otherwise, sleep onset difficulties were considerably more frequently reported as either the initial disturbance marking the beginning of sleep problems (Bixler, Kales, Kales, Bianchi & Fahs, 1978) or the primary complaint (eg. Karacan et al., 1976). In addition, recent preliminary findings (Bixler et al., 1978) suggest age may be related to the type of sleep problem. On the average, difficulties falling asleep began at an earlier age (29 years) than difficulties staying asleep (31 years) or early terminal awakening (37 years).

Bearing in mind the tentative nature of some of these results, these measures indicate that a substantial portion of the general population complain of trouble sleeping, especially trouble falling asleep. Another measure which lends considerable credence to this observation is the documented purchase and usage of sleep inducing hypnotic drugs, the most prevalent form of treatment for insomnia. In 1970 it was estimated that Americans were spending about \$170 million for retail prescriptions of hypnotics. This tally did not include the money spent on nonprescription preparations available such as Somnex. Considering both types of drugs, Karacan et al. (1973) found that 26% of a sample of over 1600 adults attested to regular use of medication to enhance their ability to fall asleep. These findings are particularly disturbing in light of the decade



of research which points to the many adverse effects of anything but very brief usage of most sleep inducing and maintaining medication. When these drugs have been used to treat insomnia, effects such as rapid tolerance, disruption of normal electrical brain wave sleep patterns (EEG), and insomnia-like withdrawal reactions have been repeatedly reported (eg. Kales, 1970; Kales, Scharf & Kales, 1978).

These drawbacks of the pharmacological approaches have contributed to the recent interest in the development and evaluation of behavioural treatments of insomnia. Along with the development of these programs has come the need for more refined and comprehensive assessment methods. Sleep may be assessed according to subjective and/or objective criteria. A variety of objective measures have been monitored over a night's sleep including bodily movements, eye movements, respiration rates, blood pressure, cerebral blood flow, body temperature and electrical brain wave activity (EEG). Unfortunately the high costs of equipment and the inconvenience of evaluating subjects in a laboratory frequently make the measurement of these variables difficult, if not impossible. General questionnaires of sleep functioning and daily sleep charts have therefore been the main sources of information about insomnia for behavioural researchers. The relative importance of subjective and objective measures in the diagnosis of sleep dysfunction is a matter of debate, with some clinicians tending to ascribe greater weight to the patient's self-report (Kleitman, 1963; Bootzin & Nicassio, 1978; Bootzin & Engel-Friedman, 1981).

## I Assessment Methodologies in Insomnia.

### Subjective parameters.

It is a commonly held belief that everyone needs at least eight hours of sleep each night in order to function well during the day. While this may be the average amount, when one considers a large general healthy adult population, what is less appreciated is the extremely wide variations in sleep needs. Sleep requirements necessary to maintain a favorable disposition and energy level differ among individuals and change over the life span. For example, there are case histories of people who sleep from one to four hours (Dement, 1970; Jones & Oswald, 1968) and show no deleterious consequences to their health or well-being. Studies have also examined the personality and daytime functioning of naturally short sleepers, that is people who average five and a half hours of sleep a night. Overall, this group does not report having fatigue or irritability during the day. Their anxiety levels are within the normal range and they seem to be adaptive and well adjusted (Beutler, Thornby & Karacan, 1978).

Besides inappropriately expecting eight hours of sleep a night, some people complain of insomnia because they are unfamiliar with the relationship of changes in sleep pattern with age. An infant sleeps about 20 hours a day. A high percentage of this time is spent in Stage 4, the deepest levels of sleep (Dement, 1972). Very gradual decreases in the average eight hour sleep requirements of young adults is evident after thirty. It is after 60-65 that men and women seem to require less sleep. They tend to exhibit fewer and shorter

episodes of the deeper levels of sleep; they also wake up more frequently throughout the night (Williams, Karacan & Hirsch, 1974). The resulting diminished total duration of sleep may partly account for the frequent complaint of the elderly of waking early in the morning.

General sleep surveys. There are many dimensions, besides total duration of sleep, which are used to subjectively assess a good night's sleep. When a clinician is attempting to establish the extent of sleep disturbance, a general sleep questionnaire may be utilized. Questions about the time it takes to fall asleep, the frequency of awakenings, ongoing life stresses, and aspects of the sleep setting must be considered if a thorough understanding of the disorder is to be gained. Unfortunately, many questionnaires are not comprehensive. Some questionnaires have been submitted to factor analysis (Evan, 1977; Johns, 1975). However, item variability across investigations make comparisons of salient factors found across measurement instruments difficult.

General questionnaires also have a number of other shortcomings, not the least of which is their retrospective nature. Sleep functioning in chronic insomniacs can vary widely from night to night (Karacan, 1971). Therefore, the insomniac's assessment may be based on the worst nights, especially if the report is intended to present the problem in a severe light in order to obtain treatment. In fact, studies which have correlated responses to general sleep surveys and responses to

either subjective (daily sleep charts) or objective (EEG) criteria of sleep variables have not found significant results (Freedman & Papsdorf, 1976; Carskadon, Dement, Mitter, Guilleminault, Zarconi & Spiegel, 1976). Global self-assessment of sleep must therefore be interpreted cautiously when used as a means of determining treatment effects. They do, however, have a place in a screening interview as a means of establishing that a patient considers poor sleep a problem. They may also be used as a convergent check of improvement obtained from other sleep report measures such as daily sleep charts.

Daily sleep charts. Detailed daily sleep charts continue to be the most popular method for evaluating sleep problems. Patients are required to log specific information about sleep and its associated rituals, shortly after rising in the morning. These schedules have the advantage of being an inexpensive, relatively nonintrusive way of determining the extent of the difficulties. They are more reliable than general questionnaires, and information is obtained in the home setting where the complaint presumably developed and is maintained. Besides aiding in a functional analysis of the problem, this sleep-related material may be correlated with other measures of functioning such as general health and thinking patterns (Whitton, 1978).

While self-report sleep charts provide important information about the patient's view of the problem, the limitations of this

means of assessment must be acknowledged. In particular, the sleep charts may intentionally or unintentionally be completed in response to various demand characteristics of the situation. The insomniac may wish to appear very debilitated in order to obtain treatment. After treatment, excessively favorable estimates of improvement may be the patient's way of indicating appreciation for the treatment or for the therapist's efforts. Meanwhile, fear of termination of contact with the therapist may lead some patients to underestimate gains. Therefore, responses to the daily sleep questionnaires may be influenced by certain client response sets. However, recent preliminary findings indicate that responses to the most typical sleep diary item, that is, latency to sleep onset, do not appear to be a reflection of the insomniac's intelligence, tendency to be influenced by suggestion or the wish to give socially desirable responses (Turner & Ascher, Note 3).

Ratings of sleep variables every morning is more sensitive to treatment effects than general retrospective questionnaires (Nicassio & Bootzin, 1974). However, behavioural researchers have reported that some patients have reported difficulties in completing the charts on a daily basis. Because these investigations depend heavily on this source of information in evaluating treatment effectiveness, they have begun to have patients phone or mail in records daily (Lick & Heffler, 1977).

Another source of bias revolves around the factors believed to influence a crucial aspect of sleep charts, time estimation.

Every sleep chart has an item about the amount of time taken to fall asleep. Insomniacs tend to overestimate this interval (Borkovec & Weerts, 1976; Carskadon, Devent, Mitler, Guilleminault, Zarconi & Speigel, 1976). Ornstein (1969) has demonstrated that subjects estimate time intervals in accordance with the quality of activity filling it. Borkovec (1979) has argued that the longer estimates of sleep onset by insomniacs may be related to the many cognitive intrusions they have while trying to go asleep.

#### Subjective vs. objective measures of sleep.

Daily sleep charts are a practical and efficient means of gathering information about a sleep disturbance. On the other hand they are also vulnerable to a substantial set of biases. Therefore, considerable research has addressed the question of the relationship between subjective and objective estimates of sleep constructs.

One issue which must be considered in the usage of self-report or objective assessments of sleep is the replicability of test results. In normal subjects both general questionnaires of the patient's sleep habits (Webb, 1965) and daily sleep ratings (Coates, Killen, Marchini, Silverman & Thoresen, Note 1) have yielded satisfactory test-retest reliabilities. What is perhaps more interesting, however, are the data which indicate that test-retest reliabilities on both subjective and objective measures of sleep onset are higher for poor sleepers when compared to normals. Normally good sleepers showed a test-retest correlation of .58 on the sleep charts and .58 on EEG measures.

Reliabilities for the insomniacs on the same measures were .93 and .70 respectively. Therefore, preliminary results indicate that daily sleep measures may be at least as reliable as EEG and perhaps even more reliable for insomniacs.

Studies correlating sleep records with objective measures of sleep tend to support the validity of subjective assessments. In particular, some investigators have trained observers to use specific criteria to assess the sleep of people reporting sleep difficulties. Correlations between observer estimates of sleep onset and the self-reports ranged from .84 (Turner & Ascher, 1979) to .99 (Tokarz & Lawrence, 1974). According to these results self-observation may not only be a reliable but a valid indicator of sleep behaviour. The fact that other laboratories (Nicassio, Boylan & McCabe, 1976; Nicassio & Bootzin, 1974) have found equally high congruence between an insomniac's and a spouse's or roommate's rating lends considerable support to these conclusions.

There are, however, some practical problems which have been encountered when using home observers. Spouses and roommates generally can only be used to gauge sleep onset difficulties as they are justifiably unwilling to monitor the insomniac's sleep all night. Reports of observers falling asleep before the insomniac are not unusual. This presents problems in the continuous collection of data. In addition, the reports of spouses and roommates may be influenced by the demand and expectancy characteristics of the situation. While observers may act as a valuable convergent validation of the insomniac's report,

they probably should not be used as the sole measure.

#### Electrophysiological recordings of sleep.

An electroencephalogram is probably the single most widely used and respected measure of sleep and insomnia (Webb, 1975). Even more widespread adoption of this means of measurement has occurred recently because of the availability of standard scoring procedures (Rechtschaffen & Kales, 1965). Visual discrimination of both wakefulness and the components of sleep processes is possible.

According to electrophysiological recordings, the quality of brain activity cyclically varies over a night's sleep. Specific but identifiable patterns mark the progression from arousal through to the deepest stages of sleep. Normal healthy adults exhibit evidence of four stages of sleep, representing increasing depths as judged by arousability thresholds (Coleman, Gray & Watanabe, 1959; Williams, 1967).

The night of sleep begins as the sleeper descends through the four stages. This takes about 40 minutes. About 15 minutes of this time is spent in the deepest, soundest stage of sleep, stage four. The sleeper is said to be in this stage when high voltage, slow waves (1-4 cycles per second) appear and occur more than fifty percent of the time.

The sleeper then returns through the stages. The slow waves of stage four, called delta, diminish and occupy the EEG less than half the time. This is stage three. Stage two becomes discriminable



with the appearance of two defining characteristics: (1) sleep spindles - short bursts of 12 to 14 cycles per second, and (2) K complexes - a large negative wave preceded by a large positive wave. When the sleeper returns to stage one (4-6 cycles per second), approximately 90 minutes will have elapsed. After this first typical progression-regression cycle of the night, there is great variability in the frequency and duration of sleep stages (Dement, 1972; Feinberg, 1974).

In contrast to the variability of sleep stages is the occurrence, at about 90 minute intervals, of a sleep state known as REM (rapid eye movement). This kind of sleep is identified with reference to typical patterns of EEG, EOG (eye movements) and EMG (bodily muscle activity). The EEG indicates the sleeper is in stage 1 sleep. However, unlike the first occurrence of this stage at sleep onset, the pattern is interrupted by sharp sawtooth waves. In concert with these changes come a marked increase in eye movements and a reduction in submental muscle activity (Jacobson, Kales, Leibman & Hoedemarcher, 1964). This period of sleep has come to be known as both active and paradoxical sleep. It has been called active sleep because aside from muscle tension, physiological functions increase, sometimes to levels comparable to a waking state. Body and brain temperature increases and blood pressure rises. The pulse rate becomes rapid and erratic when compared to non-REM sleep stages. Respiration is halting and irregular. Blood flow to the brain is elevated (Reivich, Isaacs, Evarts & Kety, 1968). Penile erections occur at least once in 90%

of REM periods (Karacan, Salis & Williams, 1978) regardless of dream content (Fischer, Gross & Zuch, 1965).

REM is considered paradoxical sleep because, despite these physiological signs of arousal, the sleeper is extremely difficult to awaken. The irony becomes even more difficult to reconcile when EEG correlates of REM are considered. Frequently the EEG resembles the pattern of someone fully awake and involved in a concentrated life experience. Otherwise, the EEG resembles very light sleep (Williams, Hammack, Daly, Dement & Lubin, 1964).

In general young healthy adults will spend a greater proportion of time in the first half of the night in stages 3 and 4. Periods of REM are shorter but change from 10-15 minute segments in the early night to up to an hour towards morning. Overall, 25% of the time is spent in REM, while 5% is spent in stage 1, 50% in stage 2, 10% in stage 3, and 10% in stage 4 (Mendelson, Gillin & Wyatt, 1977).

Studies which have analyzed the EEG characteristics of sleep in insomniac subjects have yielded inconsistent results. Coursey, Buchsbaum and Frankel (1975) found poor sleepers obtained less delta sleep. These results were contrary to an earlier report by Monroe (1967) who found no differences in this component between good and poor sleepers. It is also a matter of controversy as to whether insomniacs get more or less REM sleep than normal sleepers (Gaillard, 1976). Some investigations have found no REM differences between patients and controls (Kales, 1964).

Parameters of sleep: relationship between EEG and self-report.

There are equivocal findings as to the EEG qualities of poor sleep. EEG estimates of sleep onset, however, have been found in several studies to correlate well with self-report measures taken in the morning. In particular, in an evaluation of 122 insomniacs, Carskadon et al. (1976) found that there was a significant .64 correlation between the subject's morning-after estimates of sleep latency and Stage 1 EEG estimates. This significant relationship between self-report measures of sleep onset and EEG estimates has been reported in other investigations. Using Stage 1 EEG criteria to sleep onset, Freedman and Papsdorf (1976) reported that there was a significant .68 correlation between morning-after subjective estimates and EEG latencies in an insomniac sample. A significant .70 correlation between Stage 1 EEG and subjective estimates of sleep onset by insomniacs was found in a study by Coates et al. (Note 1). These authors thus conclude that "if the EEG is to be regarded as the criterion of sleep..., then self-report of minutes to sleep onset provides a reliable and valid relative index for insomniacs (p. 14)". This conclusion would appear to be fortified by the very similar results which have been found between self-report and Stage 1 EEG estimates of sleep onset in other investigations of insomniac samples (Frankel, Coursey, Buchbinder & Snyder, 1976; Baekeland & Hoy, 1971).

These significant correlations are even more remarkable when one considers the potential sources of error involved in recording sleep in a laboratory. Since some sleep problems may originate from

associations of arousal in the home sleep setting, the insomniac may show less disturbances in the laboratory (de la Pêna, 1978). However, it is more likely that given the variability of sleep patterns in the insomniac (Coates, Rosekind, Strossen, Thoresen & Kirmil - Gray, 1979; Hartmann, 1973) that the night's sleep in the laboratory will not be representative of the problem. Therefore, it is becoming more routine practice among behavioural researchers to record two or more consecutive night's sleep (Freedman & Papsdorf, 1976; Borkovec, Grayson, O'Brien & Weerts, 1979). Once the subject has physically and emotionally adapted to the laboratory setting, more reliable estimates of treatment effects may be obtained. However, variability between investigations will continue so long as only one night of polysomnography is obtained and different measures of sleep are used.

These drawbacks of using sleep laboratories do not detract from their essential value in the measurement of sleep problems. Gathering information on a wide range of physiological parameters during sleep allows the clinician to determine the similarities and differences in the presenting patterns of insomniac subjects. For some patients measures of anxiety such as GSR and heart rate (Haynes, Follingstad & McGowan, 1974) may provide additional clues to the etiology of the disorder or to other bodily systems which are affected by sleep disorder. Circadian rhythms are sometimes found to be disruptive during periods of sleep difficulties. Fluctuations in temperature have been used as a measure of these alterations. In addition, two sleep disturbing syndromes, trouble breathing (sleep apnea) (Guilleminault, Eldridge

& Dement, 1973) and twitching muscles (nocturnal myoclonus) (Frankel, Patten & Gullin, 1974) were first definitively discovered in a laboratory. Though a thorough subjective evaluation may be sufficient to identify these disorders, a laboratory assessment may be necessary to confirm the diagnosis.

It is not disputed that having the subject sleep in a laboratory is an important part of a comprehensive, accurate and detailed evaluation of a sleep disturbance. What is controversial, however, is the interpretation given to the objective criteria of sleep when subjective information is also available.

There has been a tendency to downplay the significance of the subjective assessment of sleep. As Bootzin and Nicassio (1978) point out, when there is a discrepancy, the sleeper's report is considered inaccurate; the EEG is considered the "real" reflection of the problem. The somewhat derogatory description "pseudoinsonniac" was attached to the approximately 50% of self-proclaimed insomniacs who came to the Stanford Sleep Centre, but who had grossly normal EEGs (Dement, 1973). This label was recently upgraded to "subjective insomnia" on the basis of several arguments.

Sleep may be conceptualized in several mutually complementary ways. It involves physiological components as indicated by the nightly fluctuations in GSR, respiration, heart rate, penile erections and electrical activity in the brain. However, there are several dimensions of the sleep experience which may coincide with, but are separate from the physiological. In particular, some people use bedtime to sort out problems or review the day's events. These

cognitive events, which may not be detected by the EEG, may alter the subjective evaluation of sleep onset through their effect on time estimation (Borkovec, 1979). It is also possible that if the insomniac is less able to distinguish sleep from waking mental events (Borkovec, 1979; Rechtschaffen & Monroe, 1969), the subjective feeling of sleep onset occurs at a later stage of EEG than in normals. A study by Slama (see Borkovec, 1979) which has recently been replicated (Borkovec, Lane & VanOot, 1981) found that 80% of the insomniacs reported feeling awake when signaled in Stage 2. All of their good sleepers reported being asleep. This finding may account for the observation in several studies that insomniacs tend to overestimate sleep onset, underestimate the frequencies of arousals and the total amount of sleep compared to EEG measures (Lewis, 1969; Monroe, 1967).

These discrepancies in subjective judgement by insomniacs are important especially if the correlations between EEG and sleep logs are not substantial. But as previously indicated, the available evidence supports a reasonably strong association between these two measures. It is likely, therefore, that subjective and objective measures are tapping a large common underlying dimension (Youkilis & Bootzin, 1981).

However, the discrepancy between subjective and objective measures deserves careful consideration. It is possible that both measures make a unique contribution to the understanding of the sleep disturbance. That is, the EEG measures may not be sensitive to the kinds of cognitive and perceptual distortions leading "pseudo"

insomniacs to complain of difficulties (Youkilis & Bootzin, 1981). Furthermore, a sleep problem may be reflected in one or all of three basic dimensions: cognitive, behavioural and physiological. It is conceivable that one characteristic of a normal sleeper is the synchronization among these systems. Some insomniacs may show less correspondence among these response measures. As an effective treatment program proceeds greater synchronization of response systems may occur, though response systems may change at different rates (Lang, 1969). It is also widely recognized that there is desynchronization of EEG and sleep after cardiac surgery because of the use of certain drugs (Bradly & Elkes, 1967), during sleep deprivation (Blake, Gerard & Kleitman, 1939), and in elderly patients with senile dementia (Prinz & Raskind, 1978): In these instances, subjects are awake and reacting, yet waves characteristic of deep sleep occur in their EEG.

On the other hand, it is possible that the already high correlation between EEG and subjective measures may increase as behavioural researchers compile more discriminative criteria of sleep and sleep onset (Bootzin & Engle - Friedman, 1981). Nevertheless, much more behavioural research utilizing both EEG and subjective measures of assessment will have to be done before the meaning of the present discrepancy between different measures of sleep disturbances can be ascertained. In the meantime, it would seem that subjective measures of sleep have much greater validity and reliability when compared to EEG than was once believed. Perhaps the place of an experiential evaluation in the comprehensive assessment of insomnia should be

reconsidered and given some of the attention and crediability generally accorded EEG alone.

## II. Insomnia: Multiple Factors in Etiology.

Disturbed sleep may originate from a variety of overlapping sources. Two prevalent notions among behavioural researchers are that poor sleeping habits and poor conditioning experiences in the bedroom could be responsible for the complaint of insomnia. One sleep habit which could disrupt the normal sleep rhythm is going to bed at irregular times, either from day to day or on weekends. In addition, naps may be used to overcome transient bouts of daytime sleepiness. These practices could adversely affect circadian rhythms. These are the cyclical biological changes which are believed to be co-ordinated so as to produce regular sleep (Hauri, 1975).

Good sleep patterns may also be unfavorably modified during short-term, unusual physical or psychological demands or stresses. Excessive noise (Hartley, 1977), an intern's nocturnal work schedule (Paulton, Hunt, Carpenter & Edwards, 1978), sensory deprivation and hospitalization for surgery (Broughton, 1978) have all been implicated as stressful factors producing insomnia. Unfortunately, the poor sleep practices can persist long after the aggravating situation has been resolved. For example, a new mother may find that she is unable to sleep well years after the birth of her first child.



Another poor sleep habit is to go to bed when one is not sleepy. It is believed that some insomniacs go to bed early in order to compensate for a previous night's poor sleep. Spending hours in bed with a mind busy churning through the day's events and concerns is believed to promote a counter-productive association between the bed and arousal. Other insomniacs may foster this association by eating, reading, listening to the radio or watching T.V. while in bed. The bed and bedroom become signals for alertness rather than soporific stimuli leading to sleep. This type of unfortunate conditioning may also arise when, after waking in the night, the sleeper becomes frustrated when attempts to return to sleep prove futile. Dismal visions of having to function the next day without "adequate" rest may also contribute to the exacerbation of the sleeplessness cycle (Bootzin, 1972).

Some promising treatment procedures have been developed from these hypothesized causes of sleep disturbances. However, some investigators have failed to find a relationship between poor sleep and poor sleep habits (Arand, Kramer, Czaya & Roth, 1972; Zwart & Lisan, 1979). . . It is only recently that the sleep habits and conditioning models of insomnia have received some limited empirical support (Kazarian, Howe & Csapo, 1979).

On the other hand, there are other causes of sleep disturbances which have received confirmation in a scientific setting. In particular, remarkable strides have been made in recent years in the identification of physical disorders which sometimes markedly

disrupt sleep. One syndrome has been discovered in which the patient stops breathing for a brief time, up to a minute (Guilleminault, Eldridge & Dement, 1973). These difficulties are called sleep apneas and may occur several hundred times a night (Dement, 1973). This inhibition of breathing momentarily wakes the patient, preventing the descent to the deeper levels of sleep. Two distinct, though probably interacting systems have been identified as likely sites underlying this disorder. In some patients the predominant problem appears to be an upper airway obstruction. That is, inspiratory movements are initiated after exhaling, but continued collapse of the throat prevents air from filling the lungs. In the other type of apnea, commonly known as central apnea, the patient exhibits no signs of respiration movements, despite the prolonged cessation of airflow (Guilleminault & Dement, 1978).

Another unusual syndrome which may repeatedly interrupt sleep is nocturnal myoclonus. This disorder is more common among males and is characterized by short but intense muscle spasms generally in the legs (Lugaresi, Coccagna, Berti-Ceroni, & Ambrosetto, 1968). As in the sleep apneas, each occurrence of the symptom, is only for a brief period, possibly too short for the patients to readily recall.

Often one of the primary presenting complaints may be excessive daytime sleepiness. However, laboratory investigations of this syndrome indicate a positive correlation between the disruptive muscle activity and EEG awakenings (Lugaresi et al., 1968).

Frequent, though less abrupt muscle movements characterize

another syndrome associated with sleep problems. This syndrome known as Restless Legs (Frankel, Patten & Gillin, 1974), is believed to arise out of unpleasant sensations which lead the patient to be continually shifting postures of the lower limbs. It has been suggested that patients with this disorder are particularly sensitive to sensations which occur during muscle inactivity (Ekblom, 1960). Very little is known about the etiology of this problem. Much more basic research will have to be conducted before there is a solid foundation for organizing theories concerning these unusual muscle movements. In particular, there is a dearth of information on the prevalence of myoclonus and Restless Legs Syndrome in the general population. Very preliminary findings indicate that at least for nocturnal myoclonus, gross patterns of muscle twitches between good and poor sleepers are similar (Bixler, Soldatos, Scarone, Martin, Kales & Charney, 1978). Therefore, the role of this and possibly other types of muscle activity in causing insomnia will have to await a more fine-grained analysis of objective and subjective sleep variables in normal and insomniac subjects. Perhaps insomniacs are more sensitive to the sleep disrupting properties of myoclonus. Another possibility is that once the insomnia has become a chronic problem, the insomniac has generally fewer resources to tolerate the additional sleep deprivation caused by myoclonus or Restless Legs.

Sleep apneas and nocturnal myoclonus are quite subtle sleep disturbing disorders. On the other hand, there are other physical

causes of insomnia which are more readily apparent. Any kind of chronic medical illness which has discomfort, distress and/or pain as a common feature is likely to give rise to varying degrees of sleep difficulties. Sound sleep is affected in patients with the frequently persistent, sometimes severe pain of the joints in rheumatoid arthritis, of the stomach or duodenum in ulcer disease (Armstrong, Burnap, Jacobson, Kales, Ward & Golden, 1965), and of the chest in coronary heart disease (Nowlin, Troyer, Collins, Silverman, Nichols, McIntosh, Estes, & Bodgonoff, 1965). With other diseases such as bronchial asthma, kidney failure and hypothyroidism it appears there is a direct modification of normal EEG sleep patterns (Freeman, 1978). Sleep disorders have also been found to attend the general deterioration of the central nervous system as observed in senile dementia (Prinz & Raskind, 1978). Similar theories have been advanced to account for the shorter sleep times of mentally retarded subjects (Feinberg, Braun & Shulman, 1969) and other brain damaged subjects with aphasia (Greenberg & Dewan, 1969) and encephalitis (Torda, 1969).

Insomnia therefore may be an unfortunate byproduct of various disease processes. Alternatively, the sleep problems may be related to the drugs used to treat the pathology. An obvious category of drugs which is likely to disrupt normal sleep is central nervous system stimulants. Amphetamines and similar stimulants may be prescribed to suppress appetite in the treatment of obesity. Migraine headaches may respond favorably when the patient is put on a caffeine-based drug regime. The sleep disrupting effects of

caffeine, in regular coffee, tea and cola beverages is well established (Karacan, Thornby, Anch, Booth, Williams & Salis, 1976). The average noninsomniac adult need ingest only four cups of regular coffee to exhibit markedly increased sleep latency and sleep interruptions similar to the waking patterns of very old subjects. In general, caffeine has been shown to substantially decrease awakening thresholds (Bonnet, Webb & Barnard, 1979).

Besides caffeine, there are well documented studies of the sleep disruptive properties of steroid medication used in the treatment of various inflammatory diseases and of the broncho-dilating, ephedrine based drugs used to ameliorate asthma.

Therefore, drugs which may promote arousal should be carefully reviewed when a person presents with a complaint of insomnia. In addition, contrary to expectation, medications which have a depressant effect on the central nervous system have also been found to be involved in sleep disorders. Both the sedative-hypnotic drugs commonly prescribed for insomnia and alcohol have been indentified as significant factors in disorganizing normal sleep (Pokorny, 1978; Kales, Scharf & Kales, 1978).

Hypnotic medications have a favorable effect on sleep onset and maintenance problems. However, laboratory investigations have highlighted the very short-term nature of these effects. After a couple nights of drug usage, changes in the normal EEG sleep pattern occur. Some preparations severely reduce or eliminate the REM phase, while others promote increases in the total amount of

Stage 4. Invariably there are alterations of one or more sleep constructs measuring the frequency, duration and/or sequencing of sleep stages. Habituation to the drug occurs generally within two weeks. The patient must then increase the dosage to obtain a continued alleviation of the sleep problem. Along with the physiological dependency which is developing over this initial period of usage, may come a psychological dependency. The belief that it is necessary to take medication in order to obtain a good sleep is further reinforced when the patient stops taking it. Kales, Scharf and Kales (1978) have recently demonstrated that even after a few days of usage of these drugs, dramatic withdrawal responses occur. Because these reactions resemble severe insomnia the terms "drug withdrawal insomnia" and "rebound insomnia" have been used to describe this syndrome (Kales & Kales, 1973). After discontinuing the drug it is not uncommon for the patient to have increased difficulties getting to sleep and remaining asleep. Also the patient frequently exhibits levels of REM in excess of normal sleep. These REM segments, which are associated with dream activity in normal sleepers, are often associated with nightmares and hallucinations in the patient just off drugs (Kales, Scharf & Kales, 1978). These anxiety-like, disturbing experiences should be attributed to drug withdrawal. However apparently many people construe them as evidence of the continuance of their sleep problem, and on a more general level, as proof that they are not dealing well with the underlying cause of the sleep disturbance, that is personal daily life problems.

Thus, the drug withdrawal reaction may potentially reinforce a low self-regard and growing sense of incompetence (Youkilis & Bootzin, 1981). Unfortunately returning to drug usage only leads to a vicious spiral where increased drug intake only makes future attempts at withdrawal more disturbing. These withdrawal syndromes are probably occurring more frequently than is realized, especially in light of the recent observation that over one third of prescriptions for sleeping pills in the United States are for periods exceeding three months (Bixler, Kales & Kales, 1976).

Rather than seeking medical advice for insomnia, some people treat themselves by using over-the-counter medications. These drugs are aggressively publicized as significant aids to improving sleep onset. However, in clinical trials these drugs have not been found to affect any type of sleep problem. Unfortunately these pills are not harmless. For example, one chemical in Somnex, scopolamine, has been shown to produce glaucoma in the elderly at normal doses and dizziness and hallucinations at double the recommended dose (Bernstein & Leff, 1967).

Besides prescriptions and over-the-counter sleeping pills, some insomniacs resort to alcohol to promote sleep. Research efforts aimed at understanding the relationship between alcohol and sleep have mainly used normal or alcoholic subjects. Unfortunately the effects of alcohol on the sleeping pattern of insomniacs has yet to be evaluated. However, it is clear from available studies that alcohol may initially decrease sleep latency but it has deleterious effects on other aspects of normal sleep (Pokorny, 1978). In particular, the frequency and

duration of sleep staging is altered. Periods of REM are shorter and there are more changes from one stage to another throughout the night. During the first half of the night when the subject is in an acute state of intoxication, Stage four sleep is predominant. The withdrawal phase is characterized by frequent awakenings and distressing dreams (Smith, Johnson & Burbich, 1971). In addition, when alcoholics abstain they exhibit a marked increase in the percentage of time spent in REM (Allen, Wagman, Faillace, & McIntosh, 1971). This is in contrast to normal subjects who tend to gradually drift back to average levels of nightly REM. Normal subjects generally return to typical sleep patterns within several days after abstinence (Williams & Salamy, 1972). Meanwhile, chronic alcoholics show EEG and subjective evidence of sleep disturbances up to a year later. During this time the quality of the alcoholic's sleep is mixed. On some nights both stage 4 and total sleep time are sharply decreased. On other nights sleep is exceptionally deep and prolonged. As with the use of hypnotics, regular use of alcohol may lead to a tolerance and withdrawal reaction which make it difficult to discontinue (Pokorny, 1978).

### III. Personality Patterns and Insomnia.

In the absence of physical disorders or sleep disrupting drug regimes, the patient's insomnia is frequently attributed to psychological problems. Many attempts have been made to characterize the insomniac's personality pattern and emotional status. The subjects used in these investigations have been selected on the basis of



several different criteria, making generalizations across studies difficult. One series of studies used college students who described having a poor sleep pattern (eg. Monroe, 1967; Johns, Egan & Gay, 1971); these subjects had no intention of seeking treatment for these difficulties. In other research only subjects who are presenting to a clinic with a complaint of insomnia are included (eg. Karacan, Williams, Littell & Salis, 1972; Coursey, Buchsbaum & Frankel, 1975). These clinical subjects are sometimes further subdivided. Individuals whose sleeping problem exists in the absence of evidence of significant emotional disturbances are called primary insomniacs. Where insomnia co-exists with a severe psychiatric disorder, patients have been labelled secondary insomniacs. Unfortunately little research has examined the personality characteristics associated with different types of insomnia within each of these subject categories. However, several researchers have emphasized the importance of this distinction for treatment planning (Greenberg, 1977; Kales & Kales, 1973).

Poor versus Good Sleepers. The study of personality measures in poor versus good sleepers has yielded inconsistent results. Johns, Egan and Gray (1971) studied the MMPI personality measures of a group of male medical students. Overall the poor sleepers had higher levels of anxiety. However they were neither more neurotic nor more introverted than the good sleepers. There were some trends indicating the poor sleepers were more emotionally reactive and hysterical than good sleepers, but no reliable patterns were apparent.

Monroe (1967) compared the MMPI profiles of 16 volunteer college students who attested on a general questionnaire to having poor sleep, with 16 age, education and occupation matched good sleepers selected from the same community. Several scales were more elevated for the poor sleepers than the controls. He concluded that poor sleepers had significantly more psychopathology. In particular they were preoccupied with bodily symptoms, socially withdrawn, depressed, anxious and neurotic.

Like Monroe, Elenewski (see Beutler, Thornby & Karacan, 1978) also used a group of college students who reported generally poor or good sleeping patterns. Subjects participated in the study as part of course requirements in an introductory psychology course. Elenewski's results tended to support the previous finding of an overall higher level of pathology in poor sleepers. However, the scales which were elevated for the poor sleepers differed between Monroe's and Elenewski's studies. For example, paranoia was a predominant feature of the poor sleepers in Monroe's investigation, while psychopathy typified the pattern obtained by Elenewski. Sex differences were not assessed in Monroe's study but Elenewski found no differences on this dimension. Finally while good sleepers in Monroe's study always had lower scale scores than poor sleepers, Elenewski found good sleepers were more defensive and prone to denial than poor sleepers.

There are differences across studies in the salient personality attributes of poor sleepers. However, excessive levels of depression

and anxiety did appear to be common features of their patterns. Nonetheless, one must be cautious in extending these findings from poor sleeping volunteer college students to populations of subjects who unlike the students, are seeking professional advice about their sleep problem. Furthermore, of the subjects who are bothered by difficulties with sleep, a portion have significant signs of psychopathology. Some empirical studies exist on these patient populations, which permit a comparison of their personality profiles to poor sleepers drawn from a college group.

Personality patterns in primary insomniacs. Karacan, Williams, Littell, and Salis (1972) examined the MMPI personality patterns of a group of patients who were seeking treatment for their insomnia. The findings from these patients were compared to an age and sex matched control group. Like Monroe and Elenewski, insomniacs in this study had elevated scale scores on depression, schizophrenia and items measuring preoccupation with bodily symptoms. In addition there was some evidence that such patients tended to be anxious, felt socially incompetent and were withdrawn.

Coursey, Buchsbaum, and Frankel (1975) compared a group of primary insomniacs to eighteen age and sex matched controls. Of the patient group, four were unable to stay asleep, six experienced both sleep onset and maintenance insomnia and eight complained of difficulties getting to sleep. Their subjective complaints were verified by an EEG evaluation. In line with previous MMPI studies of patients

and college students, insomniacs scored higher than controls on measures of anxiety and depression. Otherwise insomniacs did not have a distinctive personality pattern on the MMPI. However on personality measures besides the MMPI, patients were found to be sensitizers rather than repressors (Byrne's Repression - Sensitization Scale) and somewhat neurotic (Eysenck's Neuroticism Scale).

Overall it is difficult from these inconsistent findings, to draw any firm conclusions about the personality profile of the insomniac. For one thing the research suffers from a number of methodological shortcomings. Few efforts have been made to subdivide the insomniacs into relevant categories. Sleep onset, sleep maintenance and mixed insomnia patients are usually analyzed together, potentially masking unique personality styles. Further confounding the search for the insomniac's personality is the practice of considering subjects whose EEG confirm their subjective complaint along with subjects having grossly normal EEG sleep patterns.

Beutler, Thornby and Karacan, (1978) studied 22 age and sex-matched pairs of insomniacs and controls. To clarify some of the issues outstanding in the determination of the psychological characteristics of insomniacs they analyzed their subjects according to the type of sleep disturbance (i.e. sleep onset, sleep maintenance or mixed) and whether the complaint was confirmed by EEG tracings. They found that subjects who subjectively felt they took a long time to fall asleep but manifested no objective evidence of sleep problems were relatively devoid of psychopathology. Insomniacs who exhibited

long sleep latencies on their EEG in accordance with the complaint had a distinctly neurotic personality pattern usually typified by high scores on the "Neurotic Triad", that is, hypochondriasis, depression and hysteria.

In contrast to the basically neurotic style of adjustment of sleep onset insomniacs, is the personality disturbances observed in patients exhibiting nocturnal arousals. Patients with sleep maintenance problems have been depicted in the clinical literature as having more severe psychiatric difficulties especially endogenous depression (Costello & Selby, 1963). Beutler et al. (1978) endorsed this view by finding indications of severe confusion, agitation, social withdrawal, anti-social tendencies and emotional seclusiveness in patients with frequent awakening.

Finally, the mixed group of insomniacs who exhibited both difficulties getting to sleep and staying asleep, was similar in personality to the sleep onset only group. Their personality would be described as neurotic but apparently they have developed better ego defense systems than the sleep onset group.

Clearly much more research will have to be executed before the relationship between these three types of sleep patterns and personality variables is clarified. Researchers would do well in this area to pay more attention to the distinction between subjective and objective qualities of the patient's complaint using measures to tap both aspects of the sleep disturbances. However, where circumstances limit research to the use of self-report, it is

reassuring that like many previous investigations of primary insomniacs, Beutler et al. (1978) found a high correspondence between the subjective impressions of sleep latency and objective indices. A similar correspondence between objective and subjective measures has not been found however, in patients complaining of frequent nocturnal awakenings (Beutler et al., 1978).

#### IV. Insomnia and Depression.

Of all of the personality characteristics which have appeared on the profiles of primary insomniacs and poor sleeping volunteers, depression seems most prominent and consistent. From the elevated scores on this dimension it is impossible to determine whether the depression is a relatively permanent fixture of the personality or whether it is a result of the effects of having a chronic shortage of sleep. Symptoms of depression and anxiety have been found to persist in the face of reduction or elimination of the sleep problem (Nicassio & Bootzin, 1974). Depressive tendencies may therefore predispose individuals towards developing poor sleep, but other nonemotional factors may eventually come to maintain the difficulty (Bootzin & Nicassio, 1978).

Alternatively, high depression scores may be a function of the general kinds of self-descriptions used by insomniacs such as listlessness, lack of spontaneity, feeling drained and "washed out", depleted and without reserves (Hauri, 1979). This kind of depression would probably be distinguishable from the dysphoria that

characterizes patients with clinical depressive disorders. While the significance of elevated depression scores in primary insomnia must await further exploration, the sleep pattern of depressed psychiatric patients has received considerable attention.

Insomnia and depressive disorders. Several parameters of the sleep process have been measured in depressed patients and compared to the sleep of normals. However, contradictory findings abound in this area. Only one component of sleep - Stage 4 - has been consistently found to be reduced in depressives (Mendelson, Gillin & Wyatt, 1973). Otherwise sleep in depressives has been demonstrated to be longer (Samuel, 1964), shorter or the same (McGhie, 1966) as normals.

Besides total duration, both total REM time and REM latency have been studied in depressed patients. Contradictory results also exist in this area. Investigators have reported that REM time and latency is reduced during peak periods of the disorder. However, other studies report finding either no evidence of alteration of these variables or sometimes increased REM latencies (Williams & Karacan, 1973).

Attempts to clarify these inconsistent findings have revolved around subclassifying patients according to their type of depression and/or type of sleep disorder. Kupfer and Foster (1975) have argued for a distinction between agitated versus withdrawn depressions. They believe the agitated state is associated with less sleep than normals, while being withdrawn lends itself to hypersomnia. Limited

support for this view has come from the study of unipolar versus bipolar depressed patients. The bipolar patients who had agitated phases in their illness, were found to oversleep. In contrast, unipolar depressions were characterized by insomnia (Detre, Himmelhock, Schwartzburg, Anderson, Byck & Kupfer, 1972).

Another frequently used subclassification of depression is reactive versus endogenous depressions. According to clinical tradition, patients with reactive depressions are believed to be suffering from severe anxiety which leads to problems getting to sleep. On the other hand, endogenous depression is considered to be associated with early morning awakenings. Willis (1965) studied the sleep pattern of a large group of depressed inpatients. His results indicated that contrary to expectation, reactive patients were more likely than endogenous depressives to exhibit frequent nocturnal arousals and early morning arousals. Costello and Selby (1963) and Hinton (1963) were unable to find distinctive patterns in the sleep of reactive and endogenously depressed patients.

Some researchers have offered possible interpretations of these mixed results. Zarcone, Azumi, Dement, Gulvich, Kraemer, and Pivik (1975) have suggested that perhaps the type of psychopathology may be unrelated to particular kinds of sleep disorders. However, it is also possible that rather than type of depression, it is the enduring features of the patient's personality which are systematically related to the type of sleep disturbance. This prospect was raised by Costello and Smith (1963) who found that introversion-extroversion



personality differences were more highly related to sleep pattern than the diagnosis of the patients as endogenous or reactive depression.

From present results therefore it does not seem there is any clear relationship between having a diagnosis of depression and suffering from a specific sleep disorder (Beutler, Thornby & Karacan, 1978). Furthermore, high levels of depression within a mixed group of psychiatric patients were not found to be related to either subjective or objective assessments of sleep disturbances (MacDonald & King, 1975). What did distinguish the patients with high levels of sleep difficulties from those relatively untroubled was overall elevated levels of psychopathology. In particular, MMPI measures indicated that they were preoccupied with bodily symptoms, had schizoid tendencies and were anxious. It may be at least for a psychiatric population, variables other than, but possibly related to depression mediate sleep disturbances.

Though the nature of the relationship between sleep patterns and depressive diagnoses remains equivocal, generally favorable results have been obtained in the treatment of depression by various manipulations of sleep function. In particular Vogel, Thompson, Thurmond, Gieler and Barrowdough (1972) studied the changes in depressive symptomatology after selectively depriving patients of REM stages of sleep. Endogenous depressives were more favorably responsive to this treatment than reactive patients, but both groups were improved. Total sleep deprivation from one to several days has also been found to reduce depression (Bhanji & Roy, 1975). There

has been much speculation about underlying mechanisms involved in the efficacy of sleep deprivation for depression. Some investigators favor biochemical hypotheses implicating the accumulation or depletion of certain neurotransmitters during sleep deprivation. These substances are believed to be responsible for some depressive symptomatology. Then there are others who feel that forcing the patient to stay awake is tantamount to having him confront his fear of being unable to sleep. Prolonged exposure to staying awake may lead to anxiety reduction and subsequent improved sleep and mood. It is likely, however, that clinical improvement is a function of a combination of cognitive, behavioural and physiological alterations. At this time, research offers few leads which seem promising for untangling the complex relationship between sleep, sleep deprivation treatments and depression.

In summary, a variety of personality and psychopathological factors have been examined with respect to sleep disturbances. Unfortunately few consistent findings have emerged from this pursuit. This is probably a reflection of the multidimensional nature of both sleep disorders and depressive syndromes. In both of these realms there are a variety of diagnostic criteria which may be utilized in order to classify the problem, as well as variations in the severity of the disturbance. Insight into the associations between emotional difficulties and sleep disturbances will be further limited as long as methodological flaws plague the research. Greater attention should be paid to obtaining comparable control groups and controlling

confounding variables such as the patient's medication and diet.

Considerations of these basic principles of experimental design may also expedite our understanding of the factors involved in the improvement of depression observed after sleep deprivation.

The interface of sleep problems and psychopathology is complex and multifaceted. As technological advances occur in the measurement of physiological events during sleep, there is an increased awareness of the many as yet unsystematically recorded qualitative aspects. It could be that as Karacan and his colleagues have suggested, substantially greater knowledge of sleep disorders will come from the study of group differences in abnormalities other than percentages of sleep stages or gross EEG patterns (Williams & Karacan, 1973). Examination of phenomena such as subtle wave form disturbances during sleep may in turn promote a better understanding of the place of sleep disturbances in the schema of emotional disorders. Whether insomnia is merely a symptom of other underlying psychiatric distresses or whether the distress is one of the consequences of repeated bouts of sleeplessness is a moot question at this time. Until more information relevant to this issue becomes available, it would probably be in the patient's best interest to provide separate though coordinated treatment for the sleep disorder and the emotional difficulties (Youkilis, 1979).

Interestingly enough, several behavioural therapies which have been effectively applied to the treatment of emotional problems are showing some promise as treatment modalities for ameliorating insomnia.

Pharmacological approaches to depression and anxiety have shown no

similar, consistent trends in the alleviation of sleep problems. On the contrary, the clinician must be aware that such approaches may in fact exacerbate the sleep component of the disorder.

#### V. Sleep Deprivation Research and Insomnia.

Before considering treatment modalities, it would perhaps be worthwhile first to gain a greater appreciation for the psychological and physical effects of insomnia. One body of literature which may be relevant to understanding the consequences of insomnia is the research on the effects of sleep deprivation. In fact, however, many aspects of the design of these studies markedly limit the applicability of their findings to the study of insomnia. Firstly, for both practical and ethical reasons the subjects typically used in these experiments are healthy, young normal-sleeping males. Total sleep deprivation does not seem to produce any significant biochemical or psychophysiological effects in this population, even after substantial periods of sleep loss (Horne, 1978). However, the extent to which this group of subjects is representative of other groups in the general population remains to be determined.

Secondly, experimentally induced sleep deprivation and naturally occurring deprivation may have different effects. In sleep deprivation experiments the subject is either prevented from having any sleep at all or he is selectively deprived of various sleep stages. Keeping the subject awake has usually involved engaging him in various forms of mental stimulation while restricting his physical activities.

Generally the subject is not required to carry on normal day-to-day functioning while involved in the experiment. Many subjects in these studies apparently look upon the sleep deprivation as a challenge (Hauri, 1979).

In contrast to the conditions experienced by the subject in a sleep deprivation experiment is the plight of the chronic insomniac: This individual unwillingly must confront night after night of unpredictable levels of total sleep. Many insomniacs have a combination of the three most prevalent sleep patterns. Therefore, the insomniac may have trouble getting to sleep, staying asleep and waking before he wishes in the morning. The insomniac is then expected to function in normal daytime activities. It is no wonder then that many insomniacs describe a growing dread at the prospect of facing another night of poor sleep (Hauri, 1979).

Thirdly, sleep deprivation effects in experiments are usually calculated on the basis of a group average. This type of analysis does not sufficiently take into consideration the well established, wide individual differences in automatic responsiveness to stressful stimuli. Moreover, the subject's physiological responsiveness to sleep deprivation may be related to his natural sleeping patterns. Naturally longer sleepers who are deprived of sleep may exhibit signs of physical deterioration long before a person who requires very little sleep. Single-case studies using normal and insomniac subjects may greatly enhance our understanding of the effects of sleep deprivation on various aspects of human functioning (Youkilis & Bootzin, 1981).

There are therefore some differences between experimentally-induced sleep deprivation and its natural occurrence in insomniac populations. However, it is somewhat surprising that, even after very substantial periods of time, subjects exhibit few psychological or physical detrimental effects of sleep loss. It is only after about sixty hours that some minimal changes occur. For instance, a neurological evaluation may detect some evidence of rhythmic body movements and hand tremors and nystagmus (Sassin, 1970). There may be some temporal or cognitive disorientation. Speech may become incoherent for brief spells. Hallucinations are rare (Webb & Cartwright, 1978) and there is some intellectual functioning impairment observed (Webb & Cartwright, 1978). It is difficult to unambiguously attribute this to the sleep loss because the subject is kept awake with mentally stimulating tasks. Performance decrements do occur, but they tend to be task specific. All of these effects, however, usually disappear after a night's sleep (Naiteh, 1976).

The function of sleep. Many sleep deprivation experiments were designed to assess the merits of various theories of the role of the sleep state. These theories frequently emphasize the restorative and regenerative functions of sleep (Hartmann, 1973). Sleep is believed to occur in order to allow energy deficits to be renewed, bodily toxins to be cleansed and critical biochemical substances to be synthesized. On a psychological level, sleep is reputed to

allow repressed material from the unconscious to be discharged and memories to be either consolidated or erased (Dement, 1972). Unfortunately, the available evidence does not seem to support any particular view of the purpose of sleep. Therefore our present understanding of the basis of the severe subjective complaints of the insomniac is unclear. What is undeniable, however, is the recognition that insomnia is a major health problem which has prompted the extensive and chronic usage of sleep medications (eg. Kales et al., 1974; Karacan et al., 1972).

#### VI. Treatment Modalities:

##### Pharmacological.

Medication continues to be the most widely utilized remedy for sleeping problems (Greenblatt, 1978). This unfortunate reality is alarming because there are many well documented reports of the deleterious side effects of both over-the-counter and prescription drugs (Kales, Bixler & Tan, 1974). This conclusion applies particularly to barbiturates but it is also relevant to the use of chloral hydrate and benzodiazepin derivatives (Greenblatt & Shader, 1974). In addition, as was noted earlier, alcohol also has a profound and negative effect on a variety of sleep parameters (Pokorny, 1978). Only one chemical, L-tryptophan has been demonstrated to be free of unfavorable effects which modify the subjective wellbeing or EEG sleep patterns of the patient (Harmann, Cravens & List, 1974). However, its effectiveness in correcting sleep dysfunction appears

to be very limited (Wyatt, 1970).

It was in the early 1900's that the soporific effects of barbiturates were recognized (Goodman & Gilman, 1975). The unbridled usage of this drug and its derivatives has been greatly tempered recently, partly because of the high abuse potential and many drug overdose deaths associated with it (Koch-Weser & Greenblatt, 1974). In the case of treatment of insomnia there seems to be a general consensus that this category of medication has untoward effects on normal sleeping patterns (Greenblatt, 1978). In particular, REM sleep appears to be adversely affected both during the period of administration of the drug and after it is discontinued. In general, when the drug is given over a prolonged period, there is an initial interval in which the amount of REM decreases. It may eventually drift back toward pre-drug levels. However, once the drug is withdrawn, levels of REM are abnormally high (REM rebound). This sequence has been documented in the usage of secobarbital and pentobarbital (Kales & Kales, 1973) and amylobarbitane (Oswald & Priest, 1965). Besides the objective alterations in EEG pattern which often persist for several weeks after termination of drug usage, patients describe having had a poorer quality of sleep. Frequently they report having had frightening dreams and frequent arousals. While chlorpromazine may reduce the REM rebound effect, some patients may opt to return to former levels of drug ingestion in order to improve their sleep. This may be fatal (Kales, Bixler & Tan, 1974).

Similar and equally unacceptable objective and subjective



changes in sleep have been observed with the usage of other central nervous system depressants. Freeman, Agnew & Williams (1965) found that subjects administered a dose of meprobamate exhibited substantial reductions in REM time. They were prone to remain in one sleep stage, especially stage 2, longer than usual rather than having the number of sleep stage changes characteristic of normal sleep.

Hoffman and Domino (1967) found reserpine produced REM suppression and marked rebound on withdrawal in humans, though this conclusion did not apply to cats. These consistent findings across studies seem particularly remarkable when one considers the variety of subject samples and experimental designs utilized. When evaluating these studies one must consider the many possible sources of bias. Both the drug dosage and duration of its administration should be varied in order to provide information about the range and extent of its effects. Also, as much as possible, attempts should be made to monitor and control subject variables which could confound the results. In drug studies, it is important there be an evaluation of the patient's blood level of the chemical since individuals may vary in their metabolism of it. Careful assessment of the patient's diet and drug regimes reduces the likelihood that sleep changes are attributable to factors other than the manipulation of the specific pharmacological treatment of insomnia. It is encouraging that in the study of other nonbarbiturate hypnotic drugs and psychotropic medication investigators are frequently aware of the importance of controlling these variables. In addition, they are sometimes able to use a double-blind procedure

and, frequently, more than one outcome measure (Kales, Kales, Bixler & Scharf, 1975). Having subjective and objective assessments of change fortifies the validity of the results and improves the probability that there can be comparisons between studies (Youkilis & Bootzin, 1981).

Of the nonbarbituate group, only chloral hydrate and flurazepam (Dalmane) have been shown to be relatively free of unacceptable changes on normal sleep (Greenblatt, 1978). After about two weeks chloral hydrate loses its ability to decrease sleep onset and increase the total amount of sleep. Flurazepam has been found across several studies to retain its effectiveness for up to a month. It is also remarkable that not only do these two drugs not alter REM sleep, but also that extended usage does not lead to a withdrawal reaction. Other nonbarbituates such as glutethimide, methyprylon and nitrazepam have the same unfortunate effects as barbiturates. The reactions of severe rebound insomnia and marked REM depression occur even after very brief treatment periods (Kales, Scharf & Kales, 1978; Kales & Kales, 1974).

Therefore, except for perhaps flurazepam, hypnotic drugs of both the barbituate and nonbarbituate variety would seem to be contraindicated in the effective treatment of insomnia. Recently, attention has been directed towards evaluating the effects of enhancing the action of certain neurotransmitter substances which are believed to promote sleep. One precursor of the neurotransmitter, serotonin is L-tryptophan. Serotonin is widely regarded as a CNS monoamine

which may be related to specific sleep patterns and waking behavior (Jouvet, 1969). Though quantifiable amounts of L-tryptophan occur in many common foods which are high in protein, investigators have not manipulated the diet of their patients to observe the subsequent changes in sleep patterns. Using tablet forms of the drug, some studies have reported a significant reduction in sleep onset times (Hartmann, Cravens & List, 1974; Oswald, Berger, Evans & Zacone, 1964). Many more reports cite increases of total sleep time and slow wave sleep as a result of moderate doses of L-tryptophan. However there is recent evidence that with repeated administrations the effects become negligible (Wyatt, 1970). This finding points to the potential addictive and abuse properties of this compound once a tolerance to it has been attained. Future research will have to clarify the possibly dose-related effects of L-tryptophan on REM sleep, results which are presently mixed. Finally, if it is to be eventually used as a viable alternative to other medications, the occurrence of this amino acid in the diet must be carefully controlled. In addition, the carbohydrate content of the diet has been demonstrated to modify levels of serotonin in the blood. However, this dietary factor is frequently ignored and there has been no systematic manipulation of it in an insomniac population.

Some tricyclic antidepressant medication, sometimes used in the treatment of depression, also alters levels of serotonin in the blood by blocking its reuptake. Because depression often seems to co-exist with sleep problems, clinicians have prescribed these

drugs for insomnia. Though this medication may favorably modify the patient's mood, generally it has unfavorable effects on normal sleeping patterns. Only one study was located where doxepin, a commonly prescribed tricyclic, improved both mood and sleep qualities in a neurotically depressed patient sample (Beutler, Thornby & Karacan, 1978). Otherwise, antidepressant medication has been shown to be associated with increased wakefulness and bodily movements throughout the night (Hartmann, 1969). Compounding this situation are the suppressive effects these drugs have on REM. Furthermore, reduced amounts of REM do not abate with chronic administration. Finally, once the drug is discontinued severe rebound insomnia generally occurs (Hartmann, 1973). Much additional research will be required before the utility of this medication in the treatment of insomnia can be established. Clarification of this issue will probably be limited until there is a greater understanding of the relationship between depression and insomnia.

#### The Behavioural Treatment of Insomnia.

Drugs, judiciously prescribed, may be useful in reversing a short standing sleep problem. Because of the many potential hazards of this treatment modality, clinicians have begun to explore nonpharmacological options in the management of insomnia. The patient may be given general directives such as to increase his/her level of physical activity during the day or to decrease mentally arousing tasks in the evening (Hauri, 1968). Naps and caffeine-based

beverages may be discouraged (Bonnet, Webb & Barnard, 1979). However, it is only recently that some of these suggestions have been incorporated into programs which are undergoing scientific evaluation (Rosen, 1976). Though the mechanisms of action of many of these behavioural treatment packages are still poorly understood (Bootzin & Nicassio, 1978), research on the behavioural treatment of insomnia does lend empirical support to the belief that the behavioural treatment of insomnia may be a viable alternative to drugs.

It is quite commonly believed that some form of physical and/or mental tension is involved in difficulties with falling asleep (Coates & Thoresen, 1981). Therefore, treatment regimes frequently include strategies which are designed to reduce one or both of these sources of tension.

Methods of physical relaxation. Perhaps the most widely prescribed and certainly the most extensively evaluated behavioural treatment for insomnia is progressive relaxation training (Bootzin & Nicassio, 1978; Borkovec, 1979). Advocates of this method point to the lines of evidence indicating insomniacs may be more physiologically aroused than normals. Unfortunately, few studies have evaluated the validity of this assumption about insomniacs. Furthermore, available evidence on this issue is contradictory. For instance, Monroe (1967) did record higher levels of body movement, vasoconstriction and skin resistance in poor sleepers when compared to good sleepers. Haynes, Follingstad and McGowan (1974) measuring frontalis muscle

tension, were unable to find significant differences between good and poor sleepers. This observation is supported by other investigators who also failed to find an association between basal levels of muscle tension and the delay in sleep onset (Freedman & Papsdorf, 1976). The extent to which specific muscle groups, such as the frontalis, are representative of overall bodily tension and arousal is a matter of much debate. More definitive statements about the role of physical arousal in insomnia must await the results of studies which use a variety of bodily sites for the measurement of tension. However, even if there seems to be a relationship between physical tension and insomnia, the exact nature of the association will be difficult to establish. Tension could be causing the sleep difficulties. Alternatively, it may be merely co-existing with other causal agents. It is also possible that the prospect of another night of restless, inadequate sleep generates its own set of tension.

While causal links between muscle activity and insomnia have yet to be specified, progressive relaxation training has been demonstrated to be a moderately effective treatment modality for problems with sleep. This technique, which involves sequentially tensing and relaxing muscle groups from feet to head is reputed by Jacobson (1938), its originator, to have many beneficial effects on tension responses. Responses such as reduced muscle activity and decreased levels of automatic arousal have been reported in noninsomniac, neurotic and normal subjects (Paul, 1969).

Summarizing the results of available controlled studies Bootzin

and Nicassio (1978) conclude that progressive relaxation more effectively reduces sleep onset insomnia than placebo and waiting list control groups. This conclusion applied to both college students and more severely afflicted adults drawn from the community. In addition, these results were validated across objective and subjective measures of the sleep disturbance. Borkovec, Grayson, O'Brien and Weerts (1979) extended these findings to subjects who complained of delayed sleep onset but whose gross EEG sleep patterns were normal. They found that neither these "subjective" insomniacs nor subjects with objective evidence of their sleep disturbance improved under placebo and no treatment conditions.

Several studies have attempted to pinpoint the active component in progressive relaxation training. It does not appear that improved sleep is attributable to reduced physiological arousal since changes in indices of physiological arousal achieved during training are uncorrelated with the reported sleep at home (Borkovec & Fowles, 1973; Lick & Heffler, 1977). Furthermore, the effects of relaxation exercises seem to go beyond its value in focusing the subject's attention on his muscles (Borkovec, Kaloupek & Slama, 1975). However, tension-relaxation cycles seem to be an important ingredient before subjective improvements are reported. Borkovec (1979) has executed some component analyses of progressive relaxation in insomniac population. It is his contention that the procedure seems to be acting on certain central-cognitive processes (eg. time estimation) rather than on peripheral-physiological activity. An

additional cognitive factor which may contribute to the effectiveness of relaxation exercises is the subject's feelings of control over the sleeping problem (Bandura, 1977; Davison, Tsujimoto & Glasros, 1973).

Methods of mental relaxation. There are a number of techniques which aim to bring about a state of relaxation by having the subject engage in a specific cognitive activity. For example, subjects would in the case of meditation, be instructed to repeat to themselves a word while passively focusing on their breathing or a pleasant image (Woolfolk, Carr-Koffashan, McNulty & Lehrer, 1976). Meanwhile autogenic training may involve either repeating positive self-statements about one's drowsiness (Graham, Wright, Toman & Mark, 1975) or imagining that one's extremities are becoming warm and heavy (Schultz & Luthe, 1959).

Clinicians have also evaluated the efficacy of desensitization procedures in ameliorating insomnia (eg. Steinmark & Borkovec, 1974; Lick & Heffler, 1977). In this approach, subjects are instructed to physically relax and then to imagine various scenes associated with bedtime and/or being unable to fall asleep. The rationale behind this procedure is to reduce insomnia by having the subject experience relaxation in the presence of cues previously associated with tension and arousal.

In general, cognitive relaxation methods are as effective as progressive relaxation (Nicassio & Bootzin, 1974) in relieving sleep onset problems. Moreover, desensitization does not appear to improve upon gains achieved by relaxation alone (Gershman & Clouser, 1974).



Thus relaxation training, whether directed at the subject's physical or mental state, diminishes the time taken to fall asleep. On the average these methods lead to a 50% improvement in sleep onset (Nicassio & Bootzin, 1978). This change may represent a significant improvement for mild insomniacs but moderate and severe subjects may still be taking up to an hour to fall asleep (Hauri, 1979; Nicassio & Bootzin, 1974). Obviously it may be necessary to supplement the relaxation training program with other approaches. One such promising treatment strategy is stimulus control instructions introduced by Bootzin (1972).

The stimulus control of sleep. According to an operant model of sleep, falling asleep occurs in order to bring about reinforcement (i.e. sleep). The stimuli which signal the occurrence of the reinforcement become discriminative stimuli. In this case, the discriminative stimuli may be any stimuli associated with sleep. These may include cues in the sleep environment, activities done in the bedroom and/or the posture or mental attitude which is adopted upon retiring for the night.

Within this operant model of sleep, sleep problems may originate from the absence of strong discriminative stimuli for sleep. Besides this, discriminative stimuli for behaviors incompatible with sleep may exist in the bed and bedroom. In particular, the poor sleeper may engage in a variety of behaviors in the bedroom which are not conducive to sleeping. For example, the insomniac may talk on the

telephone, listen to the radio or watch T.V. and possibly eat and read in bed. In addition, bedtime may signal a time when the insomniac reviews the day's difficulties and anticipates future problems. Compounding these worries can be the anxiety and frustration associated with trying to fall asleep.

Bootzin (1976) has developed a set of six rules which are intended to promote the strength of cues in the bedroom for eliciting sleep. This goal is primarily achieved by separating the cues for sleep from the cues for other arousing-type activities. The rules are as follows:

1. Lie down intending to go to sleep only when you are sleepy.
2. Do not use your bed for anything except sleep; that is, do not read, watch television, eat, or worry in bed. Sexual activity is the only exception to this rule. On such occasions, the instructions are to be followed afterward when you intend to go to sleep.
3. If you find yourself unable to fall asleep, get up and go into another room. Stay up as long as you wish and then return to the bedroom to sleep. Although we do not want you to watch the clock, we want you to get out of bed if you do not fall asleep immediately. Remember the goal is to associate your bed with falling asleep quickly! If you are in bed more than about 10 minutes without falling asleep and have not gotten up, you are not following this instruction.
4. If you still cannot fall asleep, repeat Step 3. Do this as often as is necessary throughout the night.
5. Set your alarm and get up at the same time every morning irrespective of how much sleep you got during the night. This will

help your body acquire a consistent sleep rhythm.

6. Do not nap during the day.

Bootzin and Nicassio (1978) reviewed the evidence for the efficacy of this treatment regime. Not only did it lead to an impressive 70% improvement in sleep onset, but it was more effective than relaxation treatments. These results are particularly remarkable when it is realized they were generated in five different laboratories and gains were obtained for both moderately impaired and chronic/severe insomniac subjects. Unfortunately, no studies have validated these findings using EEG measures, although separate objective assessments using roommates have sometimes served as a reliability check.

The active ingredients in stimulus control treatment remain to be specified. There are at least three different hypotheses which have been advanced for the effectiveness of stimulus control programs. Because these programs require that the subjects regularize their sleep habits, especially the time at which they arise, it is possible that the stimulus control instructions synchronize the sleep-wake cycle with other cyclical biological variables. Desynchronization of biological rhythms is believed to be one potential cause of insomnia (Coates & Thoresen, 1981). Lending some empirical support to this theory is a study by Tokarz and Lawrence (1979). They instructed subjects to use only two of the stimulus control instructions, that is, to get up at a regular hour every day and to avoid napping during the day. Their findings indicated that improvement in sleep onset insomnia in this

group was comparable to a group that received the full set of instructions.

Besides regularizing biological functions, stimulus control instructions may be effective because they disrupt the normal chain of behaviors which lead to sleeplessness. By getting up when one is unable to sleep a person may be diminishing the likelihood that behaviors counterproductive to sleep such as worrying and tossing and turning will occur. This raises the possibility that any behavior performed in bed, besides lying awake could favorably modify the insomnia.

Zwart and Lisman (1979) had a group of subjects engage in various activities in bed if they woke in the night (counter control group). The suggested list of activities included reading, eating and watching T.V.. This group was compared to a group given the list of stimulus control instructions and a group which received the parts of the instructions dealing with the temporal components of the sleep problem (viz. (1). lie down to sleep only when sleepy (2). Do not take naps (3). Arise by alarm at the same time each morning.). These groups were compared to a waiting list control.

Only the stimulus control and counter control groups demonstrated significantly improved sleep onset. Moreover, these procedures led to equivalent improvement. While these results reinforce previous findings on the effectiveness of the stimulus control procedures, the improvement of the counter control group suggests that this improvement could not be considered a function of the bed becoming a discriminative

stimulus for sleep. However, in both groups, there was a disruption of the bed and bedtime as cues for the type of arousal particularly characteristic of insomniacs. That is, some investigators have noticed that insomniacs seem to become aroused because, during the difficult time prior to sleep onset, they begin to worry, to toss and turn, and to become frustrated with their inability to sleep. Stimulus control and counter control instructions contingently disrupt these sleep incompatible activities and cognitions. Another interpretation for the equivalent success of these two groups is that both groups were punished for their continued wakefulness by having to engage in an aversive activity (eg. getting out of bed). Subjects have not been systematically evaluated with respect to this variable. Obviously more work will have to be done before the potent elements in the stimulus control treatment procedure are teased out.

#### VII. A Self-help Approach to the Treatment of Insomnia.

The preceding review of behavioural treatments for insomnia indicates that there are some moderately effective programs for reducing sleep onset difficulties. In addition, some of these techniques, in particular relaxation methods and stimulus instructions, would seem to readily lend themselves to a self-administered format. That is, these procedures could be written up in a booklet form which the insomniac could read, practice and subsequently apply to the sleep disorder. In fact, self-help manuals concerning a variety of psychological problems including insomnia have been available for

many years (eg. Samuel Smiles' 1889 text, *Self-Help*). However, it is only in the past six years that psychologists have begun to focus some attention on self-management programs which do not involve a therapist's assistance.

In most current self-management/self-help books a very similar set of skills are taught. Initial chapters are devoted to a general description of the problem and an outline of scientific knowledge on the topic. The patient is then taught how to monitor the aspects of his/her behaviour which may be related to the problem. Chapters describing therapy give detailed descriptions of how one may learn to relax the body and the mind by using physical relaxation exercises and positive, coping self-statements. The patient may also be encouraged to reward himself with, for example, a special meal after he has successfully completed various phases of the program.

These concepts and strategies were the basis of a self-help manual for the treatment of insomnia written by Coates and Thoresen (1977). A text like this, if effective, would be very helpful in clinical settings. While reducing the cost of treatment to the patient, it may also allow the therapist to concentrate on the cases which are complicated by additional psychiatric symptoms or which do not respond to usage of the treatment manual alone. Additionally, the patient may be more likely to maintain therapeutic gains if he views that his progress is a function of his own efforts and resources (Kopel & Arkowitz, 1975).

However, though there has recently been a remarkable proliferation of behavioural self-help manuals, very little attention has been given

to the empirical evaluation of their merits. Rosen (1976) finds this situation alarming especially in the light of the potential as well as documented hazards involved in the usage of these materials. For instance, a patient with a medical disorder may treat the problem with a behavioural program thereby postponing appropriate treatment. Also, if the patient either fails to understand the instructions or to follow through on them "there is a risk of negative self-attributions, of anger toward self or others and of reduced belief in the efficacy of today's therapeutic techniques (p. 6)". These potentially serious consequences highlight the need to empirically evaluate these commercially available do-it-yourself therapy books.

Glasgow and Rosen (1978) surveyed the expanding body of literature devoted to testing the merits of self-help manuals. They indicated that these texts may have some utility in the treatment of obesity, study habits and phobias. Evaluation of self-help programs for the treatment of many other problems such as insomnia, assertiveness, children's behavior and sexual dysfunctions were either non-existent or have led to mixed results.

Since that review, one study was located which has examined the efficacy of a self-administered treatment program for insomnia.

Alperson and Biglan (1979) randomly assigned people suffering from sleep-onset insomnia to three groups. One group self-administered a brief, non-commercial booklet containing stimulus control instructions and meditation relaxation exercises. The second group was given a booklet containing back exercises and a list of activities

(eg. knitting) which they were to do in bed when they were unable to sleep. The final group just continued to monitor their sleep after baseline measures were taken.

After four weeks of treatment their results indicated that both groups receiving the booklets had decreased their sleep onset. The group which received the relaxation exercises and stimulus control instructions was about twice as improved as the group receiving back instructions (59% to 32%). It is possible then that any activities which interrupt the nonsleep behavior of the insomniac may alter the discriminative function of the bedtime situation and thereby improve sleep onset.

However, a number of factors make it difficult to confidently and unambiguously interpret the results of this study. Besides using a small sample size, the subjects were not medically examined in order to eliminate those insomniacs whose problem was secondary to a medical or psychiatric problem. In addition, the intake of sleep medication was not controlled. Also, the second group received a part of the stimulus control instructions (no napping) included in the treatment package of the first group. Moreover, expectancy of improvement ratings were not taken for the self-monitor-only group. Furthermore, the results of this study are based on self-report data; no objective measures of the subject's estimate of sleep latency were available. Finally, because the subjects had an opportunity to discuss the manuals with a therapist, the treatment cannot be considered totally self-administered. It is unclear therefore the



extent to which contact with a therapist was an important factor in the appropriate and successful usage of these materials. This variable has been evaluated in the usage of manuals for treatment of premature ejaculation (Zeiss, 1979) and toilet training (Matson & Ollendick, 1977). In both of these studies having some contact with the therapist was crucial in the successful application of the self-help manual.

#### VIII. Statement of Purpose.

Part of the purpose of the present study was to assess the contribution of therapist contact to the effectiveness of self-help manuals in the treatment of idiopathic sleep-onset insomnia. The present study was also concerned with evaluating the utility of a commercially available behavioural self-help manual, namely the text by Coates and Thoresen (1977). This book was selected for two reasons. Firstly, because it is a commercially available book which the general public is using to treat themselves, it would be worthwhile to gather information on its utility. In addition, this manual contained a wide variety of information on insomnia and many treatment approaches. This multimodal approach was considered to be particularly important in the treatment of a complex, multidimensional problem such as insomnia (Kales, 1981). That is because non-organic sleep onset insomnia is considered to originate from a variety of physical, situational and psychological factors it does not consistently respond to any one approach. Coates and Thoresen (1977) instruct the insomniac in ways to bolster many different skills which may be related to the sleeping

disorder. Specific skills related to insomnia such as modifying body and mental tensions are discussed. They also devote several chapters to the examination of daytime behaviors which may be contributing to poor sleep. The insomniac is given suggestions as to how he may improve his self-concept and reduce depression through self-congratulation and increased self-assertion.

The present study was also designed to locate subject variables which may be related to outcome. This information would aid in prognosis. Presently only three variables have been identified which were favorable prognostic indicators for improvement in sleep onset latency after relaxation training. They were female gender (Nicassio & Bootzin, 1974) and high validity (F) scores and low hysteria (Hy) scores on the MMPI (Borkovec, Steirmark, & Nau, 1973). Meanwhile Shealy, Lowe and Ritzer (1980), found acute insomniacs showed significantly greater decreases than chronic insomniacs in sleep onset latency after a relaxation plus stimulus control package.

To summarize, the present study asked the following questions:

1. Is a commercially available behavioural self-help manual an effective treatment modality for idiopathic sleep-onset insomnia?

The word "idiopathic" is used here to connote those types of insomnia which are of unknown etiology (i.e. neither secondary to psychiatric disturbance or medical problems nor primary syndromes such as sleep apneas and nocturnal myoclonus).

2. What, if any, is the relative contribution of therapist contact to the effectiveness of a self-help manual in the treatment of sleep-onset insomnia?

3. What, if any, is the relationship between measures of the subject's personality, anxiety, depression and self-esteem and the treatment outcome?

## Experiment 1

### METHOD

#### Subjects.

Subjects were recruited through advertisements placed in the Montreal area anglophone newspapers and interviews on local radio stations. Subjects suffering from insomnia were being offered a treatment program. Of the 127 individuals who replied to these advertisements and interviews, thirty subjects were selected who met the following criteria: (a) primary complaint was difficulty falling off to sleep; (b) take at least thirty minutes to fall asleep on at least five nights of the week; (c) had the problem for at least one year; (d) obtain evidence from their physician that they have no medical condition which could be directly altering sleep patterns (eg. thyroid dysfunction; asthma, kidney or liver disease) or be indirectly affecting the quality of sleep (eg. diabetes, pain syndromes, pregnancy, menopause, or dementia); (e) report no symptoms of sleep apnea, nocturnal myoclonus, restless legs or narcolepsy; (f) not be under psychiatric care; (g) not presently taking sleep medication; (h) non-cyclical problem; (i) F T-Scale score less than 70 on MMPI; (j) age 55 or under.

Appendix A presents the demographic characteristics of the present sample. The sample included 16 females (mean age 41.9) and 14 males (mean age 42.1). The average age was 42.0 (S.D. = 9.3) with

a range of 25-55. The average duration of the problem was 10.1 years (S.D. = 7.3).

Therapist:

The present study was conducted by the author, a Ph.D. student in clinical psychology. Besides two years of practicum work during her master's degree, the author spent three years as a full time clinical psychologist at the Behavior Therapy Clinic at the Montreal General Hospital. During this time she treated a wide variety of behavior problems including various types of insomnia. She was familiar with the principles of cognitive-behavioural therapy and the literature on self-help programs.

Procedure:

Subjects who telephoned for information about the program were given a general idea of the treatment approach. A continued display of interest led to a brief interview attempting to determine the nature and extent of the person's sleep problem. Subjects who attested to drug or alcohol abuse were not recruited for the program.

If, after this evaluation the subject appeared to be a suitable candidate, the subjects were told they were going to be mailed a two week supply of sleep charts. The subjects were then given a verbal description of the sleep variables which they were going to be required to log on the sleep charts. The subjects were instructed to complete the sleep diary each morning upon awakening for two consecutive weeks. It was requested that the subjects try to be as accurate as possible in completing the diary. A bedside clock was recommended as an aid

to this assessment if it would not cause undue concern. Finally the subjects were told they would also be required to complete a sleep history questionnaire. This questionnaire and the two weeks supply of sleep charts were, upon completion, to be promptly returned to the therapist by mail. Any questions the subject may have had about these self-report materials were then answered. All major points verbally communicated to the subjects were repeated in a brief written note accompanying the self-report materials.

If the baseline measurements indicated that they met the requirements of the study, they were then contacted by phone and asked to deposit a refundable sum of \$50. When this was received by mail they were then contacted by phone and told they were going to receive the pretreatment assessment package through the mail. As with the other self-report materials, the subjects were given a brief verbal description of the general nature of the questionnaires and told to complete them promptly and return them by mail to the therapist. A brief written note repeating these instructions was included with the copy of the MMPI, the Beck Depression Inventory, the measure of anxiety and the Tennessee Self-Concept Scale.

Appropriate subjects were randomly assigned to one of the three treatment conditions.

One week after the two months of bibliotherapy were completed, all subjects were mailed the posttherapy assessment package. This included a two week supply of the daily sleep charts and the measures of depression, anxiety and self-esteem. An enclosed letter indicated

that the two consecutive weeks of sleep charts and questionnaires were to be completed as soon as possible and returned by mail to the therapist. The \$50 deposit would be refunded once this material was received by the therapist.

After the first week of treatment all subjects answered the following questions on a 1-7 scale: (1) Does the treatment which has just been described to you seem like it would be likely to help you with your insomnia? (1 = not likely help; 7 = extremely likely to be helpful). (2) Do you think your therapist is warm and accepting of your problem? (1 = little warmth and acceptance; 7 = extremely warm and accepting). Subjects in the self-administered manual (no contact) and self-administered manual with phone contact with the therapist (see Treatment Groups Section) received and returned this questionnaire by mail. Subjects who met with the therapist in person completed the questionnaire at the end of the first session.

These questions were posed in order to evaluate the credibility of the treatment rationale, the expectancy of improvement, and the characteristics of the therapist. They were selected from the Pretherapy Evaluation Questionnaire developed by Borkovec and Nau (1972).

All subjects received the same expectancy instructions, that is, not to expect improvement for two weeks. Also all subjects were told that the program they would be receiving had been found to be effective.

### Outcome Measure.

Daily sleep charts. The primary dependent measure in the present investigation was the Daily Sleep Chart on which the subjects recorded their daily sleep patterns (Appendix B). These sleep charts required subjects to assess the adequacy of each night's sleep on five behaviourally-specific sleep dimensions. Subjects upon waking each morning, were requested to indicate how long they thought it took them to get to sleep (sleep-onset latency), how many times they thought they woke up during the night, how many hours they thought they had slept, how much difficulty they experienced falling asleep (1 = not very difficult; 5 = extremely difficult) and how rested they felt in the morning (1 = very rested; 5 = poorly rested).

These sleep variables were culled from Monroe's Daily Sleep Questionnaire (1967). This questionnaire, originally designed to study differences between good and poor sleepers, has been used as the basis of outcome measures in the literature on the behavioural treatment of insomnia (Borkovec & Fowles, 1973; Borkovec, Kaloupek & Sloma, 1975; Bootzin & Nicassio, 1977). In addition, many major sleep research centers operationally define insomnia in terms of the patient's sleep onset latency and number of hours of sleep per night. Such patients must be taking at least 30 minutes to fall asleep and sleeping less than 6.5 hours each night in order to be classified as insomniacs (Dement & Guilleminault, 1973).

Ratings of sleep onset and number of hours of sleep per night are, therefore, frequently included in the daily subjective and/or



objective assessment of insomnia. Many researchers also include measures which have been found to discriminate between good and poor sleepers using daily sleep charts. One such measure is the individual's perceived difficulty getting to sleep. This variable has emerged as a salient factor in a number of factor analytic studies of sleep questionnaires (Evans, 1977; Parrott & Hindmarch, 1978; Johns, 1975). It would therefore appear to be worthwhile to include this variable in the assessment of the sleep problem.

Another item which is frequently included in daily self-report measures of insomnia is the degree of morning restedness (Bootzin & Engle-Friedman, 1981). Ratings of how refreshed the individual feels in the morning are believed to provide information about the soundness of the sleep experience and degree of daytime fatigue (Bootzin & Nicassio, 1977; Coates & Thoresen, 1981). Including variables which regularly measure the effects of the insomnia on the individual's daytime functioning is considered by some sleep researchers as an integral part of the assessment of the sleep problem (Bootzin & Youkilis, 1981; Coates & Thoresen, 1977). Degree of morning restedness was, therefore, included as the fourth primary dependent measure in the present investigation.

#### Treatment Manual.

#### The Coates and Thoresen (1977) Manual.

The paperback version utilized in this study is a 14-chapter, (323-page) manual that consists of seven parts. Part One gives a

general summary of present scientific knowledge about normal and disturbed sleep.

In the second section, the reader is asked to become a "personal scientist" by beginning to complete a daily sleep diary. This brief schedule collects information about prebedtime and inbed activities, as well as aspects of the sleep problem (i.e. time taken to fall asleep and number of awakenings). It also documents the level of physical arousal before bed and any thoughts occurring during the interval before sleep. The authors emphasize that the diary will serve two important purposes in the program. Firstly, it will aid in the diagnosis of the sleep problem. Secondly, it will be one means of charting changes in the sleep problem once treatment strategies are being used. To enhance the likelihood that the diary is completed regularly it is suggested that the reader make out a written contract with himself or another person whereby a favorable event will occur (eg. going to a movie) if the diary is completed or an unfavorable event will occur (eg. clean out the garage) if it is not.

The second chapter in this section (chapter 4) puts forth the idea that there may be certain thoughts or attitudes about sleep which interfere with it. For example, there may be excessive worry about getting enough sleep to function (eg. "I need to get to sleep. I'll be no good tomorrow if I don't!"). It is also possible that inappropriate and frustrating expectations about not sleeping are developed (eg. "I've been lying here for an hour already. I'll bet it's going to be a miserable night!"). Once an awareness of these

thoughts is created by keeping a record of them, it is suggested that the reader generate helpful thoughts which offset the content of these self-defeating thoughts or attitudes. For instance to the unhelpful thought noted above "I've been lying here for an hour etc.", the more potentially soothing thought "That's Okay. The house is peaceful and I am resting. I'll be asleep soon" is suggested. For excessive worries about falling asleep it is suggested one think "I'll get to sleep. A half-hours sleep is not going to make that much difference".

Part three devotes a chapter to learning how to relax the body and a chapter to learning how to "cool down" an overactive mind. The technique which is described for bodily relaxation is a combination of Jacobson's progressive deep muscle relaxation procedure (Jacobson, 1938) and meditation exercises. That is, first the reader is instructed to become aware of bodily tensions by tensing and then relaxing various muscle groups throughout the body (eg. hands and arms; head and face; throat, neck and shoulders). When they are in the relaxation phase of this procedure, it is recommended that they give themselves suggestions that they are becoming warm, calm and restful. Besides these self-suggestions, which are frequently used in meditation approaches to relaxation, they are encouraged to tell themselves that with relaxation comes a sense of wellbeing and freedom from cares and worries.

In the second chapter of this section (chapter 6) which discusses ways of dealing with mental tensions, other techniques frequently used in meditation exercises are described. In particular the reader is

requested to record in great detail a pleasant scene (eg. taking a stroll in the woods; basking on a beach). At bedtime when he is troubled by intrusive thoughts, he is to evoke this scene, thereby slowing down his mind and replacing negative thoughts with a soporific image. Alternatively, he may wish to focus his mind on a single object (eg. lighted candle) or a single soothing word or phrase (eg. "calm"). He could also picture himself on an escalator which is descending to deeper and deeper levels of relaxation. Chapter 7 which begins Part Four, describes the usage of these positive imagery and self-statement skills when one wakes during the night.

The remainder of Part Four examines the many environmental factors which may be contributing to the sleep problem. The reader is instructed to keep a log of activities which are done in the evening while in bed (eg. reading, eating, watching T.V.). It is suggested that any activities performed towards bedtime should be conducive to promoting a restful physical and mental state. In addition, it is recommended that the bed and bedroom be restricted to sleep. Therefore the reader should only go to bed when he is sleepy. If he is unable to sleep he should go out of the bedroom and wait until he feels sleepy before returning to bed. These chapters also discuss the deleterious effects which irregular bedtimes have on the sleep rhythm, using the classic case of jet lag as an example. One factor which appears to disrupt regular bedtimes is naps. They are discouraged.

The fifth section examines various facets of daytime behavior which may be related to poor sleep. Chapter 10 teaches the reader to

identify three salient components of daily stress responses, namely, physiological, physical and thinking styles. On each occasion during the day, when there is marked stress or tension, the reader is required to document the time, the event and the effects of this experience. Several strategies are suggested as a means of dealing with these stressful events. It may be possible to modify the environment in order to reduce the stress. For example, it may be stressful commuting to work in rush hour traffic. Therefore, leaving home and work at different times may minimize this type of tension. Another way to reduce tension is to discuss the stressful situation with someone in a position to understand and possibly aid in problem solving about it. For instance, it may be stressful to have the boss expect you to complete an assignment within a certain time. Letting the boss know that you do not feel you can do an optimal job with such short notice may reduce this source of stress. Finally, some daily tensions may be reduced with the conscious application of bodily relaxation exercises. Details of how to apply the relaxation in daily activities are given.

Being physically more relaxed during the day may enhance one's chances of getting a good night's sleep. In addition, feeling good about oneself may elevate one's mood and, thereby, reduce another source of sleep-disrupting stress. This is the reasoning behind many of the suggestions made in chapters 11 and 12 which are intended to improve the reader's self-concept. It is recommended that the reader review three important activities during the day and indicate the standards he is using to judge his behavior. A low self-esteem may

be partially based on unrealistically high standards for one's behavior. If this is the case more attainable standards should be set and self-congratulation should occur for approximations to these goals. Also, specific pleasurable activities should be included in a day so as to have a behavioural base for building feelings of competence and self-satisfaction. Practicing these more positive ways of viewing oneself and one's world is emphasized as the key to modifying one's sense of wellbeing.

Finally, Part Six suggests a number of ways of maintaining one's progress. These include general techniques from telling others about your progress in order to obtain encouragement and support, to specific record keeping tasks designed to chart changes in sleep variables.

Enumerating helpful sleep habits is another means by which progress may be maintained.

#### Treatment Groups.

##### Self-administered manual (no contact).

Subjects in this group (N = 10) were mailed a copy of the self-help manual. They were told it was important to work on their own rather than relying on a therapist. Each subject was told to follow only the instructions included in designated chapters for each week of their program and not to do more. The outline they were given was the same one followed in the other two groups. This group had no further contact with the therapist until the post assessment telephone contact, two months later.

Self-administered manual with phone contact with the therapist.

This condition (N = 10) involved weekly phone contact with the therapist at prearranged times. Phone contact was supportive and directive in nature and served to check that the program was being correctly followed, to congratulate successes and to provide encouragement when subjects felt discouraged. The subjects were also given help resolving problems related to the program.

Self-administered manual with therapist contact.

In this condition (N = 10) subjects were seen by the therapist at a clinic. Subjects met together as a group once a week for about an hour over an eight week period. The therapist focused on reviewing subjects' programs, answering questions about the program and previewing procedures to be used during the coming week. The therapist and group members congratulated the successful completion of homework assignments and problem solved any difficulties. Brief practice in the techniques of the program (eg. relaxation, self-contracting, positive self-talk and imagery) was given.

All groups received the treatments in the following order: (1) General discussion of sleep (chapters 1 and 2); (2) Evaluating sleep behavior (chapter 3); (3) Cognitive modifications (chapter 4); (4) Progressive relaxation (chapter 5); (5) Positive imagery (chapters 6 and 7); (6) Stimulus control (chapters 8 and 9); (7) Daytime behavior a) applying relaxation; (8) Daytime behavior b) self-assertion.

Because of the limitations of subject availability and the established finding that no-treatment subjects do not improve (Bootzin and Nicassio, 1978), a no-treatment control group was not included in the design.



## Results

### Preliminary Analysis

The means and standard deviations for the Daily Sleep Questionnaire items (baseline and posttherapy periods) are presented in Table 1. These values were calculated on the basis of the second week of record keeping, when it was considered likely that a more stable baseline would have been reached and the effects of self monitoring (Kazdin, 1974) would have been minimized.

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Insert Table 1 about here

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The present study used four dependent measures which were found to be moderately correlated (Bartlett's test of Sphericity = 15.83,  $p < .02$ ). Multivariate Analysis of Variance (MANOVA) was therefore selected as the main initial technique of data analysis (Hummel & Sligo, 1971; Turner, 1978; Kaplan & Litrownik, 1977). Though these multivariate procedures have been shown to be robust under a wide variety of conditions (Olson, 1974; Harris, 1975), the assumption of homogeneity of variance was not found to be violated in the present study.

Table 1

Means and Standard Deviations for the Daily Sleep Questionnaire  
Measures for the Baseline and Posttherapy Periods

<u>Treatment</u>	Latency to Onset	Falling Asleep	Hours of	Level of
	<u>(minutes)**</u>	<u>Difficulty**</u>	<u>Sleep</u>	<u>Restedness**</u>
<u>Baseline</u>				
Manual & Therapist				
M	84.30	3.00	5.87	2.88
SD	53.85	.57	.68	.69
Manual & Therapist (phone)				
M	78.90	3.04	5.77	2.91
SD	47.67	.42	1.90	.78
Manual Alone				
M	75.00	2.77	6.41	2.93
SD	22.55	.67	1.24	.59
<u>Posttherapy</u>				
Manual & Therapist				
M	32.20	1.68	6.6	1.84
SD	21.28	.52	.63	.71
Manual & Therapist (phone)				
M	40.40	1.98	6.2	2.30
SD	37.85	.91	2.11	.84
Manual Alone				
M	61.70	2.56	6.52	2.62
SD	29.81	.74	6.52	2.62

\*Note All ns = 10

\*\*Decreased score indicates improvement

A multivariate analysis of variance conducted on the baseline values of the dependent variables was non-significant,  $F(8,50) = 413, p < .9$  (Pillais Trace Criterion). Thus the groups were initially similar. There was also no evidence of nonrandom assignment on the basis of frequency of nocturnal awakenings, age, sex, duration of insomnia or the measures of depression and anxiety.

After one week of therapy subjects were asked to complete two questions intended to assess their evaluation of the therapy rationale and aspects of the therapist. An analysis of variance performed on the mean group values of these variables indicated that there were no reliable group differences. All groups gave a moderately positive rating for the rationale ( $\bar{X} = 4.1$ ) and therapist ( $\bar{X} = .46$ ). These ratings by Group appear in Appendix N. It was therefore considered unlikely that any differences among the groups on the dependent measures were a function of either the subjects' initial assessment of the treatment rationale or the attributes of warmth and understanding demonstrated by the therapist. Since repeated measures analysis of variance failed to demonstrate any effect of the sex of the subject on the response to treatment for any dependent measure, the data were collapsed across this variable for the analysis reported below.

#### Initial Analysis

A multivariate analysis of variance was performed on the

dependent measures using one between subject (treatment group) and one within subject factor (baseline vs. posttherapy). This analysis revealed a significant effect of testing period (pre-post)  $F(4,24) = 21.63, p < .001$ . There was also a significant treatment  $\times$  testing period interaction  $F(8,50) = 2.25, p < .03$ . These effects were analysed in further detail.

#### Within Group Changes

Univariate repeated measures analysis of variance collapsing across conditions indicated significant improvements in response to treatment on all variables ( $p < .001$  for sleep onset, restedness, falling asleep difficulty, and hours of sleep per night). However, an analysis of pre-post changes within each group indicated that not all treatment conditions were successful in producing these improvements. The average reduction in sleep onset was 61.5% for the group which met with the therapist and 48.7% of the group receiving phone contact. A 17.3% reduction in sleep latency occurred in the no contact manual only group. Tukey's post hoc comparisons ( $K = 6, d.f.27$ ) revealed a significant pre-post difference for mean sleep onset latency for the group which met personally with the therapist ( $Q = 7.25, p < .05$ ) and the group which had telephone contact with the therapist ( $Q = 5.39, p < .05$ ). For the reported levels of morning restedness the group which met personally with the therapist exhibited a significant pre-post change ( $Q = 8.13, p < .05$ ) as did the group

which had telephone contact with the therapist ( $Q = 5.04, p < .05$ ). For the rated difficulty getting to sleep both the group which met personally with the therapist and the telephone contact groups had significant pre-post changes,  $Q = 10.2, p < .05$ ;  $Q = 7.84, p < .05$  respectively. However, only the group which met with the therapist in person significantly increased the number of hours they slept per night ( $Q = 5.65, p < .05$ ).

#### Between Group Comparisons

The univariate repeated measures analysis also revealed a significant group x period effect for onset  $F(2,27) = 3.75, p < .03$ ; reported morning restedness  $F(2,27) = 3.44, p < .05$ ; and rated difficulty getting to sleep  $F(2,27) = 9.01, p < .001$ . The number of hours slept per night was marginally significant  $F(2,27) = 2.90, p < .07$ .

Duncan's Multiple Range Comparisons using baseline to post therapy period changes were performed in order to locate group differences on the dependent variables. For sleep latency the results indicated that the two groups which had contact with the therapist were significantly different ( $p < .05$ ) from the group which only had the manual. However, the therapist groups did not differ from each other. This pattern also occurred for measures of

rated difficulty getting to sleep ( $p < .05$ ) and hours of sleep per night ( $p < .05$ ). Only the group which met personally with the therapist and the group which had no contact with the therapist significantly differed in levels of morning restedness.

#### Changes on Psychometric Measures

The psychometric measures (anxiety, depression and self-esteem) were submitted to separate repeated measures analysis of variance. Main effects of testing period were significant for anxiety  $F(1,27) = 4.59$ ,  $p < .04$  for depression,  $F(1,27) = 10.43$ ,  $p < .003$  and for self-esteem  $F(1,27) = 12.79$ ,  $p < .001$ . A group X period interaction occurred for self-esteem scores ( $F(2,27) = 7.83$ ,  $p < .002$ ). This finding was followed by a Duncan's Multiple Range Test on the mean change scores from baseline to posttherapy periods. This analysis indicated that the group which met with the therapist was significantly different from the no contact manual alone group,  $F < .01$ . Only the subjects in the group which had personal contact with the therapist significantly improved their levels of self-esteem over the course of therapy ( $p < .05$ ). The mean change in self-esteem for the group which met with the therapist was  $+10.1$ . The nonsignificant mean changes in the therapist telephone contact group and the no therapist contact, manual alone group were  $+4.5$  and  $+3$  respectively.

### Relationship Among Measures

Pearson Product-Moment Coefficients were obtained from correlations among baseline measures of the dependent, demographic, and psychometric variables (anxiety and depression). This analysis indicated that sleep-onset latency was according to Bonferroni (Lazalere & Mulaik, 1977), significantly correlated with the measure of anxiety  $r(28) = -0.36, p < .05$ . The duration of sleep was negatively correlated with the rated difficulty of falling asleep  $r(28) = 0.34, p < .05$  and levels of depression as indicated by the Beck Inventory  $r(28) = -0.40, p < .03$ . Morning feelings of restedness were related to the number of hours slept  $r(28) = -0.47, p < .009$ . The rated difficulty getting to sleep was moderately correlated with morning restedness  $r(28) = .37, p < .05$ .

### Individual Difference Measures, Subject Variables and Outcome

#### Minnesota Multiphasic Personality Inventory (MMPI)

Scoring. The MMPI IBM 805 answer sheet was scored by hand. Scoring templates were used to obtain the subject's raw score on the scales. This raw score was then K-corrected for the five appropriate scales. Scale 1 (Hypochondriasis) was K-corrected by .5, Scale 4 (Psychopathic Deviate) -as K-corrected by .4, Scale

7 (Psychasthenia) was K-corrected by 1, Scale 8 (Schizophrenia) was K-corrected by 1, Scale 9 (Hypomania) was K-corrected by .2. The K score used was the raw score of this validity scale. This scale is considered to reflect aspects of the subject's test-taking attitude such as the desire to appear either abnormally free of or unusually disturbed by psychological symptoms. The K-correction is part of the standard scoring procedure because the standard profile form is based on K-corrected scores and because virtually all of the data concerning interpretation of scores are derived from K-corrected scores.

After the appropriate scales were K-corrected, the raw scores were converted into standardized scale scores. This score was obtained by plotting the raw scores (K-corrected) on the male or female profile summary chart (Hathaway & McKinley, 1967). This standardized scale score was used in all MMPI analyses.

Profile Analysis. The MMPI profile for entire sample prior to treatment appears in Appendix K. None of the mean scale scores on the MMPI validity or clinical scales was elevated above T70. Scores above this level are frequently considered to be indicative of seriously disturbed functioning. The one validity scale which is often used as a general index of psychopathology, F, was however, somewhat high ( $T = 58.26$ ), but it



was within normal limits. In addition, it should be noted that three of the clinical scales which are typically among the four most elevated in an insomniac population (Kales, Caldwell, Preston, Healty & Kales, 1976), were also highest in the "profile" of the present sample. These peaks in the profile occurred for depression, conversion hysteria and psychopathic deviate. According to this criteria, therefore, the present sample may be considered similar to other clinical samples of insomniacs previously studied.

The MMPI profiles for each of the treatment groups separately appear in Appendix D. An analysis was performed to determine if the treatment groups differed on their MMPI profile elevations. A one-way analysis of variance performed on each of the validity and clinical scale scores indicated that the groups did not differ on any of these variables. The results of these analyses appear in Appendix E.

#### Measures of Anxiety and Self-Esteem

The mean pretreatment level of anxiety for this sample was also available; it was 6.2 (S.D. = 1.7). This value is slightly lower than the mean anxiety levels for a sample of clients at an Adult Guidance Center according to the norms reported by Krug, Sheier and Cattell (1963). In addition, the mean total score on the measure of self-esteem was 45.36 (S.D. = 11.54). This value indicated that, in general, the present

sample of insomniacs did not have a very favorable view of themselves.

#### Subject Variables and Outcome

Though some changes were evident in anxiety, depression and self-esteem over the course of treatment, none of these individual difference variables predicted improvement in sleep onset. Only one measure, the social introversion scale of the MMPI, was found to account for a significant amount of variance in the percent of improvement of sleep latency. A stepwise multiple regression analysis performed on all of the individual difference measures revealed that scores on this scale accounted for 19% of the variance in the percent improvement  $F(1,28) = 6.69, p < .03$ . No other variable could be added to it which significantly contributed to prediction of percent improvement.

Following on analysis of individual difference measures and outcome, a subsequent analysis was performed which added the three treatment groups to which the subjects had been assigned as a variable in the prediction analysis. It should be noted that for this analysis the "treatment group" variable was dummy coded as follows: "Treatment Group" Variable 1 - Group 1 coded 1; Group 2 coded - 1; Group 3 coded 0. "Treatment Group" Variable 2 - Group 1 coded 1; Group 2 coded 1; and Group 3 coded - 2. The sex variable was coded females 0, males 1.

When the "treatment group" variable was entered first before the psychometric and demographic variables the "treatment group" variable accounted for 46% of the variance in the outcome  $F(2, 27) = 11.68, p < .0002$ . No other demographic or psychometric variable could be added to this variable which would significantly improve the prediction of the outcome.

It would also be worthwhile to know whether the "treatment group" variable continued to account for a significant proportion of the variance when the variance due to the main demographic and psychometric variables had been removed. Therefore a multiple regression analysis which gave priority to these demographic and psychometric variables before entering the "treatment group" variable was performed. Results from this analysis appear in Appendix L.

This analysis indicated that the psychometric and demographic variables which were forced first into the analysis did not significantly predict the outcome  $F(7, 22) = 1.16, p < .36$ . However, once the "treatment group" variable was added to the prediction equation a significant 69% of the variance in the improvement to sleep onset was accounted for  $F(9, 20) = 5.14, p < .001$ . These results suggest that the most important variable in predicting the amount of improvement in the sleep problem is the treatment group to which the subject was assigned.

### Sex differences.

The primary dependent measures used in the present study were self-monitored time to sleep onset, rated level of difficulty getting to sleep, rated level of morning restedness and the number of hours of sleep per night. Using the sex of the subject as a grouping factor, a repeated measures analysis of variance was performed on these four primary dependent measures. The means and standard deviations for the primary dependent variables separated by the sex of the subject and the treatment group appear in Appendix O. The absence of any main effects or interactions of the sex of the subject on any dependent variables established that the sex of the subject did not have a differential effect on the response to treatment. The data therefore were collapsed across this variable for the analysis (p.77-78).

Percent improvement in sleep onset latency was used as the outcome measure in the regression analysis reported above. Though this measure was not one of the primary dependent measures, it would perhaps be worthwhile to determine if this measure correlated with the sex of the subject. After dummy coding the sex variable therefore, the correlation between the sex of the subject and the percent improvement in sleep onset latency for

the total sample was calculated; this correlation was  $r = .40$ ,  $p < .01$ . The latency to sleep onset for the males and females before and after therapy according to group membership appears in Appendix M. On the average the females exhibited a 56 percent improvement in sleep onset latency and the males exhibited a 28 percent improvement. These results suggest that, in general, for this outcome measure, females are more likely than males to benefit from a behavioural self-help manual approach to the treatment of their insomnia.

A closer inspection of the change in the latency to sleep onset for the males and females before and after therapy according to group membership (Appendix M) suggests that females appear to be showing greater improvement than the males in the "Therapist Contact" and "Therapist by Phone" groups. There does not, however, appear to be an effect of the sex of the subject on the outcome in the "No Therapist Contact" group. The group exhibiting the greatest improvement was the "Therapist Contact" group. There was a larger number of females in the "Therapist Contact" group (7/10) as compared to the "Therapist by Phone" group (4/10) or the "No Therapist Contact" group (5/10). From the present data therefore it is not possible to unambiguously determine whether the differential results obtained from the three groups were a function of the independent variable (Therapist Contact Condition) or the disproportionate number of men and women in the treatment groups.

### Subject's Satisfaction with the Program and Use of the Manual

A brief telephone interview was conducted after the post-therapy questionnaires were received. At that time the subject was requested to rate their satisfaction with the program (yes/no) and to indicate whether or not they had read at least parts of the manual (yes/no). If they said they had read the manual then they were asked to describe the extent to which they felt they had "put into practice" the material they had read.

All subjects said they were satisfied with the program and had read at least portions of the manual. Only two subjects (both from the totally self-administering group) said they had not consciously used the techniques they had read about. The other subjects variously attested to having used "some" "much" "alot" and sometimes "all" of the material they had read about. Unfortunately, these responses were too vague and inconsistently applied to be amenable to further analysis.

### Discussion

One of the major findings of Experiment 1 is that a commercially available self-help manual for the treatment of insomnia is a moderately effective treatment modality for moderately severe, chronic sleep-onset insomnia. This conclusion applied to subjects

who had at least some contact with a therapist. Clients had contact with a therapist and a group of insomniacs for an hour a week or received a brief telephone call of no more than ten minutes. Both of these means of delivering psychological health care would seem to be more efficient and economical than the standard clinic situation, where the client meets with the therapist on a one-to-one basis. Thus these findings would seem to suggest that Coates and Thorensen's manual may be usefully incorporated into a psychological clinic's treatment program for insomnia. It also indicates that a minimum of therapist time would be required in order to bring about quite substantial changes in moderately severe, chronic sleep problems. Furthermore, two subject variables which may be useful in increasing the likelihood of a successful outcome with this manual are female gender and the clients degree of interpersonal comfort, as measured by the Social Introversion Scale of the MMPI.

The other major finding from the present study was that clients who administered the manual without therapist contact did not exhibit statistically significant improvements in their sleep latency. This finding reinforces the observation from the study of other behavioural self-help manuals that it is not possible to automatically generalize the effects of the usage of self-help manuals under minimal therapist contact conditions, to a condition where the client has no contact with the therapist and must therefore totally self-administer it. Bellack, Schwartz and Rosensky (1974),

for example, found that obese subjects who were required to use a self-help manual on their own did not lose weight. The subjects, however, who used the manual in conjunction with minimal therapist contact through the mail successfully modified their problem. Matson and Ollendick (1977) found that mothers who were given a behavioural self-help manual designed to help them toilet train their children did not effectively use the manual without some contact with a therapist. Christensen, Johnson, Phillips and Glasgow (1980) studied parents who were attempting to use a self-help manual designed to help them alter their children's behaviour. These investigators found that parents required at least minimal contact with a therapist in a group setting in order to benefit from the usage of the manual.

The insomniacs in the totally self-administering condition of the present study obtained their copy of the self-help manual from a professional in a university setting. This group however in many other ways approximates the circumstances which an insomniac may confront after purchasing this commercially available book. This would suggest that possibly the moderately disturbed, chronic sleep-onset insomniacs in the general population who are using this behavioural self-help approach to their problem, are not obtaining significant benefit.

When considering these conclusions one must bear in



mind some important qualifications. As indicated in the results section, the group which exhibited the greatest improvement namely, the "Therapist Contact" condition, also contained the greatest number of females. In addition, the average age of subjects in this group was higher than the ages in the other two groups. It will be an important goal of future research to design factorial studies which will permit the independent evaluation of the age and sex of the subject on the outcome of the usage of a behavioural self-help manual for insomnia.

## Experiment 2

### Introduction

Self-report data has been the main source of information about the sleep problem for researchers investigating the behavioural treatment of insomnia (Borkovec & Fowles, 1973; Borkovec, 1979; Bootzin & Nicassio, 1977). Daily self-report measures have been found to be an efficient, practical and inexpensive means of obtaining reliable information (Bootzin & Engle-Friedman, 1981).

Relying solely on self-report data, however, is considered by some investigators to be a methodological limitation in the study of behavioural treatments of insomnia (Knapp, Downs & Alperson, 1976; Montgomery, Perkins & Wise, 1975; Ribordy & Denny 1977). Sleep charts may be intentionally or unintentionally completed in response to various demand characteristics of the situation. For various reasons the client may wish to appear either very debilitated or very improved after the treatment. Subjective estimates of latency of sleep onset do not appear to be influenced by the subject's intelligence or desire to give socially desirable responses (Turner & Ascher, Note 3). These subject response sets however may influence the completion

of other sleep chart items.

Self-report measures of sleep onset have been found to be correlated with the most widely accepted measure of sleep, EEG (Carskadon et al. 1976). The correlations between self-report and Stage 1 EEG measures of sleep onset have ranged from .64 (Carskadon et al., 1976) to .70 (Coates et al., Note 1). Some researchers have argued however that this less than perfect correlation suggests that subjective estimates of sleep variables are inaccurate (Lichstein & Kelley, 1980). They contend therefore that there is a need to incorporate into behavioural studies of insomnia, a measure which will provide an objective check on the subject's sleep problem. Obtaining EEG tracings was not considered a generally viable option as an objective measure in these studies. These measures require expensive equipment and elaborate sleep laboratories facilities.

Researchers investigating behavioural treatments of insomnia have therefore checked on the subject's self-report by obtaining ratings of sleep onset from the subject's spouse or roommate. These measures however have been found to be difficult and unreliable to collect. They have also been found to be susceptible to many of the same biases as the insomniac's self-report data (Coates et al., Note 1; Bootzin & Engel-Friedman, 1981).

A promising new approach to the objective assessment

of sleep has recently been developed by Kelley and Lichstein (1980). They describe a device which appears to overcome many of the limitations of other non-self-report means of obtaining information about the sleep problem. The instrument is portable and easy to install. Sleep data may therefore be collected in the environment of most concern - the subject's own bedroom. It is also comparatively inexpensive. Data are collected throughout the night thereby permitting the collection of information on several sleep variables. Ratings of the device's noxiousness indicates that it does not intrude or disrupt the sleep experience. Measures from the sleep device were also found to be highly correlated with EEG measures of sleep.

This device was developed for usage in research on behavioural treatments for insomnia. The initial validation study of it by Kelley and Lichstein (1980), however, used normal sleeping subjects. It remains unclear therefore the extent to which this instrument may be useful in measuring sleep functioning in an insomniac sample.

The purpose of Experiment 2 of the present study was to gather some information on the usage of this device with an insomniac sample. The sample studied was drawn from the group of subjects who participated in Experiment 1. Data generated from this subject sample would aid in determining the extent to which their subjective estimates

of sleep variables, which were used as the primary dependent measures in Experiment 1, corresponded to an objective estimate. This information would also permit an assessment of the impact of the treatment program on both the subjective and objective aspects of the sleep disturbance.

### Method

Subjects: Ten subjects from Experiment 1 were located who agreed to use the sleep assessment device. The subjects' group membership in Experiment 1 and demographic characteristics appear in Appendix E. Four subjects from each of the first two groups (i.e. manual and therapist; manual and therapist with phone contact) and two subjects from the no contact, manual alone condition agreed to participate in this experiment. Overall the average age of this group was 37.0, and the average duration of their problem with sleep-onset insomnia was 9.5 years. Five females and five males were included in Experiment 2.

### Procedure

Subjects were contacted by phone and asked if they would be willing to participate in a program to assess the present status of their sleep problem. They were told that their participation

would involve using a recently developed sleep assessment device.

A continued display of interest led to a brief description of the device (i.e. the device emitted soft tones throughout the night to which they were to verbally respond if they heard it).

Of the 14 subjects who were reached by phone, four declined to participate. Two of these subjects thought the tone may worsen their sleep problem and the other two subjects were not going to be available (leaving Montreal; going into hospital).

The subjects met individually with the experimenter.

A brief unstructured interview was conducted in order to obtain the subjects' impressions about the current status of their sleep problem and any changes which may have occurred in the sleep problem since the posttherapy assessment.

The operation of the sleep device was then demonstrated in detail to the subjects. The subjects were then required to demonstrate to the experimenter the steps that they were going to go through in order to install and to use the device. The tone on the sleep device was set to an audible but non-disruptive level. All subjects in this experiment found the tone, when set at its lowest level of loudness, was audible but not disruptive or intrusive.

Subjects were instructed to call the experimenter once the device was appropriately installed at home. Any malfunctions

of the device were to be reported immediately. They were told to use the device on four consecutive nights. In the morning, when the device was turned off, they were to complete the abbreviated form of the sleep chart. This chart recorded their subjective estimates of sleep onset latency, number of hours of sleep and number of awakenings.

#### The device

The sleep assessment device "consisted of a major and a minor timing circuit, a one-second tone generator, and a cassette tape recorder. All components were housed in a conventional attache case which opened for use. The device operated on regular house current and operated in the following manner. The major timing mechanism triggered a soft tone of one second duration every 10 minutes and 30 seconds throughout the night... Whenever the tone sounded, the tape recorder started recording until the minor timing circuit shut it off 10 seconds later (Kelley & Lichstein, 1980, p. 137)".

The device was placed on a table or chair at the head of the bed. The subject was instructed to make a verbal response immediately upon hearing the tone at any time throughout the night. In actuality, the recorder captured any verbal responses delayed up to even 10 seconds. The presence of a verbal response was taken

to be evidence of wakefulness. The absence of verbal responses was interpreted as evidence of sleep. "Hence, the cassette recording of an entire night produced dichotomous awake versus sleep data requiring about 1 minute of tape per hour in bed to reconstruct an individual's sleep pattern. Adjacent 10-second intervals on the tape were easy to discern since the recorder always captured the tone at the beginning of each interval (p. 137)".

Each night, prior to turning off the lights, the subject was to turn on the device and check that it was functioning. The device was considered to be functioning if, after pressing a "test" button the one second tone sounded and 10 seconds of recording tape was observed to have been activated. The cassette tape was housed in a chamber with a clear plastic cover. The subject therefore could readily observe the rotation of the cassette tape reels, which indicated the tape was operating appropriately. It should be noted however, that the subject did not have any direct access to the cassette tape itself. While it is true the subject could observe the total amount of tape which accumulated from one reel to the next, it is unlikely that this information would have aided the subject in any way in estimating the sleep variables. Moreover, the introduction in the present study, of having the subject test the equipment was considered to have two advantages. Firstly, it ensured the continuous and consecutive nightly collection



of data. Secondly, it also provided the subject with an opportunity for marking the beginning of each night's recording. By stating the date during the 10 seconds of "testing" recording tape, the subject clearly indicated the beginning of a night's recording. During the remainder of the night the subject was instructed to say "yes" immediately following any tone which was heard.

#### Scoring of the sleep device protocol

The recordings were scored according to the absence or presence of a verbal response between successive tones. The subject's statement of the date marked the beginning of that night's record. This scoring procedure yielded dichotomous sleep versus awake decisions. Thus each scored protocol then consisted of a non-continuous series of data points (i.e. ten-second sampling "points") spanning the subject's nighttime in bed.

Before comparing the estimates of sleep variables obtained from the sleep device and those obtained from the subjective measures, certain assumptions had to be made about the information obtained from the sleep device protocol. That is, it was assumed that each sampled data point best represented the time interval of which it was the midpoint. Hence, for a sleep device sampling period of ten minutes and 30 seconds, any data point indicating

sleep was assumed to have been preceded and succeeded by five minutes and 15 seconds of sleep.

### Outcome Measures

The dependent measures were the subjective estimates of sleep latency, hours of sleep and number of awakening and the objective estimates of these variables obtained from the sleep assessment device.

### Results and Discussion

Since it has been frequently documented that the presence of a sleep monitoring device (e.g. EEG) seems to have an initially disrupting effect on the subject's sleep pattern (de la Pêne, 1978; Kelly & Lichstein, 1980), only responses obtained on the second two nights were included in the following analysis.

Both overall and individual Pearson Product Moment reliability correlation coefficients were obtained on the following three variables: time to sleep onset, number of awakenings and total number of hours of sleep. The means and standard deviations of these variables ( $N=10$ ) for the sleep device and the sleep diary appear in table 2.

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Insert table 2 about here

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The overall correlation between the objective and subjective measures, (i.e. collapsing over the three variables) was  $r = .90$ ,  $p < .01$ . In addition, the individual correlations were also high. Of particular interest in the present study is the extent to which the subject's subjective impressions of sleep latency compared to the objective sleep device; this correlation was  $r = .90$ ,  $p < .01$ . The correlations for the other two variables were somewhat lower (hours sleep:  $r = .86$ ,  $p < .01$ ; awakenings:  $r = .89$ ,  $p < .01$ ).

These results suggest that that there is a clear correspondence between the subject's subjective estimate of the sleep disturbance, especially sleep onset latency, and an objective measure of the difficulties. It is likely therefore that the subjective changes in their sleep which they reported after treatment and at the time of their participation in Experiment 2, also occurred in the objective aspects of the problem.

The present study has extended the use of the sleep assessment device to subjects with self reported sleep problems. It is important to note however that though some of the subjects in this sample had severe sleep problems before the treatment, they

Table 2

## Means and Standard Deviations of the Sleep Device and Sleep Diary

	<u>Sleep Onset</u> (in minutes)	<u>Hours of</u> <u>Sleep</u>	<u>Number of</u> <u>Arousals</u>
Sleep Device			
M	36.5	6.1	3.0
SD	24.5	1.0	2.6
Sleep Diary			
M	38.6	6.6	3.3
SD	22.4	.9	2.9

had markedly improved after the program. This progress tended to have been maintained at the follow-up (i.e. Experiment 2). On the average then the sleep problem of this group could not be considered severe at the time of Experiment 2.

The current sleep functioning of this group then closely resembled the subjects pattern after therapy. This group's pre and posttherapy measures on criterion and dependent measures

appears in Appendix G. The subjective estimates of sleep onset for each of the ten subjects for the pre-and posttherapy and follow-up periods appears in Appendix H. The majority of subjects who participated in Experiment 2 reported that they felt their sleep problem had not appreciably changed since the posttherapy assessment. Inspection of the group averages and individual subject's data tends to substantiate this observation. Three subjects deviated from this pattern. One subject (S8) felt the sleep problem had completely returned after termination of therapy. Two subjects (S3 and S10) felt that the sleep problem was drifting back to pretherapy levels, though they felt it was still "much better" than before the treatment. All the subjects reported they had consulted the self-help manual at least once since the end of treatment.

The subjects who used the sleep device were therefore generally satisfied with the current status of their sleep problem. Overall the sleep functioning of this group at posttherapy and follow-up periods most closely resembles that of mild insomniacs. It will remain for future research to determine the extent to which the present findings apply to moderately and severely sleep disturbed insomniacs.

Another goal of future research will be to map out the correspondence among measures of self report, the sleep

device and EEG for insomniacs. The present study found a high correlation between the insomniac's self-reported estimate of sleep latency and measures taken from the sleep device. This result was found by Kelley and Lichstein (1980), in normal sleeping subjects. These investigators also found a substantial correlation between the sleep device and Stage 1 EEG Sleep ( $r = .83$ ). It is possible then that the estimates of sleep variables provided by the insomniacs in the present study also closely correspond to the most widely accepted measure of sleep, EEG. This conclusion however must be considered tentative until an EEG validation study of the sleep device is conducted using insomniac subjects.

There are therefore limitations involved in the conclusions which may be drawn from the present study. Incorporating an EEG measure of sleep and more severely sleep disturbed subjects into future research designs will enhance the generalizability of the results. On the basis of the present findings however the sleep device would appear to have potential for providing sleep researchers with a sturdy, compact and portable objective measure of sleep. Further suggestions for future research using the sleep device are discussed in the following section.

7

### General Discussion

The results of Experiment 1 suggest that sleep-onset insomnia may favorably respond to the usage of a commercially available self-instructional manual, provided the subject has some contact with a therapist. The average reduction in sleep onset latency for the two groups which had contact with the therapist compare favorably to previously reported treatment studies of insomnia conducted by a therapist. However, there was only a small average decrement in the sleep onset of the group which totally self-administered the manual. This finding is in contrast with the statistically significant though moderate improvements reported by Alperson and Biglan (1979). In their study, a group receiving a booklet containing meditation exercises and stimulus control instructions was able to reduce their sleep latency by an average of 59%. The possibility that the dissimilar types of manuals used in these two studies is partly responsible for these differences is perhaps worthy of some consideration.

Self-help treatment manuals may differ in length, detail, content and complexity. For instance, the manual put together by Alperson and Biglan was simple and straightforward.

It included descriptions of two techniques which were readily amenable to a written format. Meanwhile, the commercially available multicomponent treatment manual used in Experiment 1 was considerably more detailed and indepth. This text contained general information about sleep and suggestions about ways to sustain motivation while learning the many treatment methods. The reader was also instructed in some approaches to improving self-esteem.

However, many facets of self-administering a complex treatment manual may block or impede progress. For example, the technique descriptions may require clarification or demonstration. It is also possible that a new technique may be introduced before the subject feels competent in using the ones he has recently acquired. Either of these difficulties may erode the subject's confidence in the manual or in his ability to modify his problem. These issues point to the need for studies examining the relative effectiveness of self-administering manuals which vary in complexity. It also suggests that it may be necessary for the insomniac to consult a therapist who can aid in tailoring the information in the manual to suit his individual needs.

Though contact with a therapist was important in successfully using Coates and Thoresen's manual, differences were not statistically significant whether the contact involved a brief



(6-10 minute) weekly telephone conversation or an approximately one hour group meeting. Subjects in both groups were given support, encouragement and help in solving specific problems with the program. The present design therefore does not allow for conclusions about the relative merits of therapist support or direction. It does however suggest that considerable therapeutic gains may be made with a minimum of therapist time. This would reduce the costs and improve the efficiency involved in the standard clinic setting, where the therapist meets on a one-to-one basis with the client.

These generalizations to a clinical setting seem well founded given the characteristics of the insomniac sample selected. The subjects included in this study were adults, drawn from the community. All of them felt strongly enough about the debilitating effects of their insomnia to seek the treatment. In addition, the subjects had fairly severe sleep disturbances which had existed for several years.

Because of the intense, chronic nature of the sleep problem exhibited by many subjects in the present study, it was not surprising that the average sleep onset after treatment, for the groups which improved, was still longer than normal. However, other ancillary measures of sleep quality (i.e. difficulty getting to sleep and morning restedness) also significantly improved, implying that

the treatment favorably affected the subject's overall satisfaction with the sleep experience. In addition, the reductions in neurotic symptomatology and elevations in self-esteem suggest that subjects in these groups may have also experienced an increase in their general personal life satisfaction.

Furthermore, several other measures suggest that the observed improvement is not likely to be a function of differential subject characteristics, expectations or aspects of therapeutic demand. Firstly, subjects were carefully screened in order to minimize the possibility that their sleep problem was secondary to medication or a physical disorder. In addition, attempts were made to select insomniacs whose primary complaint was difficulty getting to sleep. Secondly, subject ratings of the therapist and the treatment logic showed no between group differences. This finding implies that group differences are unlikely to be attributable to aspects of the therapist's relationship with the client or the client's treatment expectancies. Lastly, there was a high correspondence between subjective reports and the sleep device, an objective measure of sleep. This result reduces the likelihood that the observed changes were a function of therapeutic demands. Furthermore, it would seem to provide some limited support for the validity of the self-report data, on which Experiment 1 of the present study was based.

This conclusion, however, must be tempered by an awareness of several factors in Experiment 2 of the present study which limit the generalizability of these results. In particular, the sleep device was used with only a small sample of subjects. Moreover, this objective data was collected several months after the end of the program. Many events could have occurred during treatment or in the follow up period which affected the subject's reactions to the device. For example, enhanced levels of physical and mental relaxation may have improved time estimation (Borkovec & Hennings, 1978). Finally, the average levels of sleep onset for those who volunteered to use the device indicated that the sleep problem was mild. Future research will have to determine the extent to which the present findings apply to subjects with moderate to severe insomnia. Results from this device may also vary according to whether the insomnia is primarily one of sleep onset difficulties or is typified by nocturnal arousals. In the meantime, the device seems to have some promise in providing sleep researchers with a moderately priced, minimally disruptive portable objective measure of sleep.

There are other difficulties in the present study, besides those already highlighted, regarding the sleep device. It should be noted that conclusions drawn from this study are somewhat limited due to the absence of adequate control groups. Such control groups

would have ruled out the possibilities that treatment effects observed were due to the passage of time (waiting list), nonspecific treatment variables (placebo) or self-monitoring of the sleep disturbance. The difficulty of obtaining suitable subjects created practical limitations to the size of the project. This consideration, in conjunction with the minimal changes observed in control conditions of other studies (Bootzin & Nicassio, 1978), favored the assignment of available subjects to treatment rather than control conditions.

Using a single therapist to conduct all the treatment conditions could be considered another methodological flaw of the present study. There are two aspects of the therapist who conducted the present study which may have contributed to the outcome. Firstly, the therapist has extensive clinical experience with the behavioural-cognitive techniques outlined in the manual. It is possible that a more experienced therapist may be better able to advise clients about the most effective ways to use the treatment techniques. The present therapist's experience may thus have contributed to the effective usage of the manual by the subjects who had contact with her. Secondly, the therapist was aware of the previous literature evaluating the usage of self-help manuals for the treatment of various problems, including insomnia. This knowledge may have generated differential expectancies about the effects of the treatment conditions. The therapist, for example, for example, may have thought that the group which met personally

with her would exhibit greater improvements than the telephone contact group. If such a belief was associated with differential treatment of the groups, then the therapist's expectancies may be one factor influencing the outcome.

Knowledge of the previous literature, however, would not have fostered differential expectancies about the outcome of the treatment conditions in the present study. The only previous study evaluating a self-help manual approach to insomnia had not assessed the effects of therapist contact. The therapist in the present study therefore did not feel there was an empirical basis upon which to expect one group to improve more than another. Further fortifying this belief was an awareness that experiments evaluating behavioural treatments for insomnia had not demonstrated a therapist effect (Nicassio & Bootzin, 1974; Steinmarke & Borkovec, 1974; Tokarz & Lawrence, 1979). No significant effect for the therapist factor had been detected either for treatment type or sex of the client. The previous relevant research suggests therefore that aspects of the therapist are not likely to contribute to the outcome in the present study.

A further limitation in the design of the present study is the absence of any systematic evaluation of the long term effects of the program. The unequal and limited number of subjects per group who volunteered to use the sleep device, make this an

unacceptable sample on which to form any firm or meaningful conclusions about follow-up changes. Four subjects from each of the two therapist contact groups and two subjects from the no contact manual alone group were included in Experiment 2. It will remain for future research therefore to establish the persistence, both subjectively and objectively of the posttherapy changes observed in Experiment 1 of the present study. Incidentally, one variable which may enhance the likelihood of obtaining follow-up information is a strong monetary contingency. It is possible this factor was important in the lack of dropouts from this study. In particular, the attrition rate in the strictly self-administered (i.e. no therapist contact) condition of this study was quite impressive when compared to the dropout problems reported with self-directed treatments in other areas such as weight reduction (Mahoney, Moura & Wade, 1973), study skills training (Beneke & Harris, 1972) and fear reduction (Clarke, 1973). The reason for these differences is difficult to pinpoint. Unstructured posttherapy responses from the subjects in this condition suggested that they enjoyed reading the manual, though it was unclear how much of what they read was put into practice. Nevertheless, in general, apparently having a greater understanding of their problem and access to a compendium of techniques for its amelioration was reassuring. Satisfaction from these aspects of their program in conjunction with the monetary incentive, may have aided in their completion of the study.

The completion of the program requirements for the group which self-administered the manual without contact with the therapist is perhaps significant for other reasons. In particular, it is noteworthy that though the average change in their sleep onset was not statistically significant, all members of this group voiced a satisfaction with the program. Comments from these subjects indicated that they found the book "informative", "interesting" as well as "supportive" and "reassuring". As a consequence these subjects described feeling much less agitated at the prospect of lying awake, in bed prior to falling asleep. Meanwhile, a few subjects indicated they had had some difficulties in understanding or applying some of the techniques. However, generally they added that their experiences with the text had not discouraged them. If anything, having read such an informative and encouraging account of their problem had stimulated their interest in resolving their problems by possibly consulting one of the sleep clinics listed in the book.

These unsystematic observations are perhaps important insofar as they suggest that the subjects who used the manual on their own did not seem to suffer from any psychological setbacks as a result of the program. On the contrary, it appears that their experiences with the manual may have reduced their anxiety about their difficulties, motivated them to continue to work on their problem and possibly encouraged some of them to seek treatment from a professional. These would seem to be very valuable experiences

towards constructively dealing with a sleep problem. Perhaps in assessing the utility of self-help books therefore attention should not be restricted to changes in the target problem. Future research could perhaps more systematically explore these indirect, but possibly important positive consequences on the subject's outlook and motivation of using self-help materials.

While the group which self-administered the manual without therapist contact did not seem to be negatively affected by the experience, they also did not improve their levels of sleep onset. Either the quality or quantity of this group usage of the manual when compared to the therapist contact groups, may have contributed to this outcome. Only very suggestive evidence relevant to this hypothesis is available from the present study. In particular, only two subjects said they had not used the techniques they had read about; they were both members of the manual alone group. However, more detailed and discriminating measures of manual usage will have to be incorporated into future research, before any definitive statements about the relationship of this variable to outcome can be made. In the meantime it is encouraging to note that commensurate with the recent interest in the empirical evaluation of self-help manuals, has come refinements in the measurement of manual usage. For example, recently Glasgow, Schafer and O'Neil (1981) obtained two measures of usage from subjects who had used a self-help manual for smoking reduction namely (1) the percent units in the book which had been



read and (2) a check list of behaviours they had used in each phase of the program (e.g., relaxation exercises, stimulus control instructions etc). Other useful suggestions have included having the subject list the techniques they have attempted and/or take a test assessing their knowledge of the manual's principles (Frankel & Merbaum, 1982).

Besides examining the utility of a behavioural self-help manual, the present study was designed to locate factors which would possibly aid in prognosis. Results from this portion of the analysis were, aside from female gender, dissimilar to favorable prognostic signs identified in other treatment studies of insomnia. Borkovec, Steinmark and Nau (1973) found that high validity (F) scores and low Hysteria (Hy) scores on the MMPI were associated with improvement in sleep onset latency after relaxation training. Shealy, Lowe and Ritzler (1980) found that improvement in a relaxation plus stimulus control treatment was related to initially higher scores on the Test taking Attitude (K), Depression (D), and Psychopathic Deviate (Pd) Scales and low scores on the Hypomania (Hy), Schizophrenic (Sc) and F scales. In the present study, the only variable which predicted a significant amount of the variance in sleep improvement was the Social Introversion (Si) Scale of the MMPI. The relatively small amount of variance which it accounted for in improvement implies that it may have only limited utility in screening out insomniacs who may not profit from a self-help manual approach to insomnia.

In general, however, it appeared that insomniacs who improved were initially more sociable, warm, active and involved and tended to have fewer difficulties establishing relationships of real intimacy than insomniacs who showed less improvement (Newmarke, 1979).

This finding will obviously have to be cross-validated in future research.

One goal of future research would be the replication of the findings of the present study. In addition, it is possible to make several suggestions for other studies which may clarify the issues raised and generated by the present research. That is, there are a number of parameters of the present study which could perhaps be more fully and systematically explored in other research programs. For example, many aspects of the sleep problem could be explored in greater detail. The subjects studied in the present project suffered from moderately severe, chronic sleep-onset insomnia problems. It would be worthwhile in the future to gather information on the response to the present treatment approach, of subjects troubled by other types of insomnia (e.g. frequent nocturnal awakenings), by other severities of the disturbance (i.e. mild or severe), and by more acute durations of the problem.

Furthermore, subjects varying in the severity, type and duration of the sleep problem may be assigned to groups which differ

in terms of the type of self-help manual being used. The present study examined the utility of a complex, commercially available self-help treatment manual. Alperson and Biglan (1979) examined the utility of a straight forward, non-commercially available self-help manual. Incorporating both simple and complex types of manuals into a study may clarify some of the issues associated with the usage of these different types of material. Glasgow et al. (1981) found that, using a self-help manual treatment approach to smoking, a simple manual was more effective when administered without contact with a therapist. On the other hand, the complex manuals were significantly more beneficial when used with the guidance and supervision of a therapist. It would be interesting to determine whether similar conclusions were reached when simple and complex manuals for insomnia were compared under both therapist and self-administered conditions. In addition, it would also be enlightening to compare the utility of Coates and Thoresen's manual with other commercially available self-help treatment manuals for insomnia. For the treatment of problems such as obesity, smoking or nailbiting there are several behaviourally based books which are commercially available. The relative merits of these texts has been the subject of some recent research (e.g. Glasgow et al., 1981). Unfortunately, however, to the author's knowledge, there are no other commercially available self-help books for the treatment of insomnia which like Coates and Thoresen's manual, are based directly on present day

behaviour and cognitive therapy principles and treatment techniques.

Therefore, there are aspects of the sleep problem and the manual which perhaps merit further research. Another variable which could be examined in greater detail is the type of therapist contact which is involved in the usage of self-help texts. The present study found that a brief, weekly telephone interview with a therapist was comparable to a weekly group meeting in improving the time taken to fall asleep. Other types of therapist contact conditions may involve communicating with the therapist by mail or leaving regular reports on the therapist's phone answering machine. Another approach to therapist contact might be to include, along with the manual, a cassette tape of a therapist describing the techniques in the book, and encouraging the client to have patience in resolving their problem. Furthermore, it would be helpful to know whether, to be effective, the contact with the therapist must include both support and specific advice. Zeiss, (1979), studying the utility of a self-help manual for the treatment of a sexual problem, found that a brief weekly supportive phone conversation lead to improvement in the problem. It may be possible therefore to train volunteers to provide this kind of nonspecific feedback to the client, thereby permitting the therapist to concentrate his/her time and skill on the more intractable cases of insomnia.

Finally, one component of the present study which is

perhaps worthy of more attention in future research is the sleep assessment device which was studied in Experiment 2. The present findings extend the work of Kelly and Lichstein (1980) by using insomniac, rather than normal, subjects. The high correlation which they found between the sleep device and subjective estimates of sleep onset was also found with the present insomniac sample. However, their device requires further evaluation as a means of gauging the relative efficacies of behaviour treatment programs. Future studies may obtain estimates of sleep variables from the sleep device both before and at various stages throughout a treatment program. These objective estimates may then be compared with subjective measures. This would indicate whether the present finding of a high correspondence between the sleep device and self-report observed for the treated insomniacs of this study, apply to insomniacs before treatment and over the course of their therapy.

In addition to generating objective sleep information for the purpose of comparing treatment efficacies, the sleep device may also be useful in gaining a better understanding of the nature of the insomnia. It may be possible to design research which uses the sleep device to both signal wakefulness and to indicate the kind of cognitive or physiological activity occurring at the time of the response. For example, after having the subject signal wakefulness by responding to the tone, it may be possible to have him specify

the nature of his cognitive activity by using a simple code system. Such a code system may involve responding "A" if he is worried about falling asleep, "B" if he is ruminating about personal problems, "C" if he is not worried and just allowing his thoughts to float etc. In this way, the sleep device may provide information about the pattern of the sleep disturbance and clues to the kinds of cognitive events contributing to the sleep disorder. Nevertheless, it is obvious that much more research using the sleep assessment device will have to be done before any firm statements about the full extent of its utility for the study of insomnia can be made. However, the compact and portable design of the device, the ease with which one may score and interpret its data and the close correspondence it has been shown to have with EEG measures of sleep make the study of its use a potentially important area of future research.

#### Concluding Summary

In conclusion then, results from the present study indicate that usage of Coates and Thoresen's self-help manual for insomnia may produce substantial, though less than complete, decreases in sleep-onset insomnia. A major qualification, however, is that the insomniac have contact, either personally or over the telephone, with a therapist who provides support and guidance throughout the program. That is, the manual was not effective when used on a totally self-administered basis. Moreover, the high correspondence observed in

this study between the subjective and objective indices of sleep disturbance implies that these conclusions may be generalized to both of these aspects of the problem. The group which self-administered the manual without having contact with the therapist most approximates the circumstance which an insomniac may confront after purchasing this commercially available book. It is possible therefore that moderately disturbed, chronic sleep-onset insomniacs in the general population who are using this behavioural self-help approach to their problem, are not obtaining significant benefit.

Reference Notes

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APPENDICES



Demographic Characteristics  
of Subjects by Group

Treatment Group

		Group 1 (Therapist)		Group 2 (Therapist by phone)		Group 3 (No contact)	
		<u>Males / Females</u>		<u>Males / Females</u>		<u>Males / Females</u>	
N -		3	7	6	4	5	5
<u>Variable</u>							
<u>Age</u>	M	51.3	42.6	35.5	40.7	39.6	42.6
	SD	1.5	7.0	8.7	11.1	11.1	10.8
<u>Duration</u>							
<u>of Problem</u>	M	17.6	6.4	10.1	12.0	9.0	10.2
<u>(in years)</u>	SD	6.6	3.7	12.6	6.3	6.4	3.6

Daily Sleep Chart

Name: \_\_\_\_\_

Date: \_\_\_\_\_

1) How many minutes did  
it take you to fall  
asleep last night?

\_\_\_\_\_

2) How many times did  
you awaken in the  
night?

\_\_\_\_\_

3) What is the total  
number of hours and  
minutes you slept  
last night?

\_\_\_\_\_

4) Please rate how difficult  
it was for you to fall  
asleep last night.

1...2...3...4...5...

not very  
difficult

extremely  
difficult

\_\_\_\_\_

5) Please rate how rested  
you feel this morning.

1...2...3...4...5...

very  
rested

poorly  
rested

\_\_\_\_\_

Manova Results and Univariate Tests for Latency  
to Sleep Onset, Falling Asleep Difficulty, Hours of Sleep  
and Level of Morning Restedness

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Manova Tests

Effects	df. hyp.	df. err.	F	p
Group	8.0	50.0	.76	ns
Pre-Post	8.0	50.0	2.25	< .04
Interaction	4.0	24.0	21.62	< .0001

Univariate Tests for Group

Variable	df	F	p
Latency to Sleep Onset	2,27	.26	ns
Falling Asleep Difficulty	2,27	.76	ns
Hours of Sleep	2,27	.24	ns
Level of Morning Restedness	2,27	1.23	ns

Univariate Tests for Pre-Post

Variable	df	F	p
Latency to Sleep Onset	1,27	34.85	< .0001
Falling Asleep Difficulty	1,27	59.84	< .0001
Hours of Sleep	1,27	18.04	< .0001
Level of Morning Restedness	1,27	36.77	< .0001

Univariate Tests for Group X Pre-Post

Variable	df	F	p
Latency to Sleep Onset	2,27	3.75	< .04
Falling Asleep Difficulty	2,27	9.01	< .001
Hours of Sleep	2,27	2.90	< .07
Level of Morning Restedness	2,27	3.44	< .04

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Means and Standard Deviations of Ratings  
on the MMPI Validity and Clinical Scales  
for each Treatment Group

<u>MMPI Scale</u>	<u>Treatment Group</u>		
	Group 1 (Therapist)	Group 2 (Therapist by phone)	Group 3 (No contact)
<u>Validity</u>			
L Scale	51.1(7.8)	49.4(6.7)	51.6(8.7)
F Scale	58.6(6.0)	57.9(5.1)	58.3(7.7)
K Scale	51.3(8.5)	56.0(9.3)	55.6(11.6)
<u>Clinical</u>			
<u>Scale 1: Hypochondriasis</u>	43.6(17.9)	45.1(9.7)	49.6(19.4)
<u>Scale 2: Depression</u>	69.9(12.)	68.2(10.0)	55.4(20.5)
<u>Scale 3: Hysteria</u>	64.0(12.4)	59.1(9.8)	63.8(9.7)
<u>Scale 4: Psychopathic Deviate</u>	65.8(17.9)	54.1(28.0)	57.0(19.7)
<u>Scale 5: Masculinity-Femininity</u>	51.2(14.0)	48.6(10.8)	60.8(19.1)
<u>Scale 6: Paranoia</u>	59.4(10.4)	56.0(9.2)	55.1(19.0)
<u>Scale 7: Psychasthenia</u>	63.5(26.7)	52.4(22.5)	47.2(25.5)
<u>Scale 8: Schizophrenia</u>	58.0(27.2)	47.9(24.9)	49.6(27.7)
<u>Scale 9: Hypomania</u>	54.5(11.6)	53.8(16.6)	47.8(10.6)
<u>Scale 10: Social Introversion</u>	60.0(9.0)	55.4(10.7)	50.8(11.3)

APPENDIX E:

Results of Univariate  
F-Tests for the MMPI  
Validity and Clinical  
Scales

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<u>Variable</u>	<u>df</u>	<u>F</u>	<u>p</u>
MMPI L Scale	2,24	.21	ns
MMPI F Scale	2,24	.03	ns
MMPI K Scale	2,24	.67	ns
MMPI Scale 1	2,24	.35	ns
MMPI Scale 2	2,24	2.87	ns
MMPI Scale 3	2,24	.64	ns
MMPI Scale 4	2,24	.69	ns
MMPI Scale 5	2,24	2.11	ns
MMPI Scale 6	2,24	.30	ns
MMPI Scale 7	2,24	1.17	ns
MMPI Scale 8	2,24	.42	ns
MMPI Scale 9	2,24	.92	ns
MMPI Scale 10	2,24	2.25	ns

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Group Membership and  
Demographic Information  
on Subjects who Used  
the Sleep Assessment  
Device

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		<u>Treatment Condition</u>					
		Group 1 (Therapist)		Group 2 (Therapist by phone)		Group 3 (No therapist Contact)	
		Males/Females		Males/Females		Males/Females	
N =		1	3	3	1	1	1
<u>Variable</u>							
Age	$\bar{X}$	53.0	38.6	30.6	28.0	33.0	48.0
	SD	0.0	6.4	7.2	0.0	0.0	0.0
Duration of Insomnia	$\bar{X}$	25.0	5.0	5.6	14.0	13.0	11.0
	SD	0.0	2.6	5.5	0.0	0.0	0.0

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Criterion and Dependent  
Measures of the Subjects  
who Used the Sleep  
Assessment Device for  
the Pre and Posttherapy  
Periods

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<u>Criterion Measure</u>		<u>Pretherapy</u>	<u>Posttherapy</u>
Anxiety	$\bar{X}$	6.5	6.4
	SD	1.7	1.2
Depression	$\bar{X}$	5.8	5.3
	SD	5.8	3.9
Self-Esteem	$\bar{X}$	47.6	49.8
	SD	14.6	16.4
<u>Dependent Measures</u>			
Sleep Onset Latency	$\bar{X}$	85.0	34.2
	SD	55.3	14.5
Hours of Sleep	$\bar{X}$	6.2	6.6
	SD	3.8	4.0
Difficulty Falling Asleep	$\bar{X}$	2.8	1.7
	SD	.5	.4
Morning Restedness	$\bar{X}$	2.8	2.1
	SD	.6	.7

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Subjective Estimates  
of Sleep-Onset Latency (in minutes)  
of Subjects Who Used the  
Sleep Assessment Device  
for the Pre, Posttherapy  
Periods and Follow-Up  
(EXPERIMENT 2) Assessment

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<u>Subject</u>	<u>Treatment</u> <u>Group</u> <u>Membership*</u>	<u>Pretherapy</u>	<u>Posttherapy</u>	<u>Follow-up</u>
1	1	124	56	63
2	1	222	55	45
3	1	53	17	40
4	1	70	27	10
5	2	66	31	16
6	2	119	40	45
7	2	33	15	2
8	2	39	14	45
9	3	64	55	70
10	3	60	32	50

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\* Group Membership

- 1 = Therapist  
2 = Therapist by phone  
3 = No therapist contact



Means and Standard Deviations  
of Anxiety, Depression and  
Self-Esteem Before and  
After Treatment by Treatment  
Condition

Treatment Groups

	Group 1 (Therapist	Group 2 (Therapist by phone)	Group 3 (No therapist contact)
<u>Measures</u>			
Anxiety	Pre 6.6(1.5) Post 5.5(2.0)	6.0(2.3) 6.9(1.4)	6.0(1.4) 5.6(1.5)
Depression	Pre 6.8(6.4) Post 2.8(3.8)	6.9(5.6) 5.4(5.0)	7.3(5.2) 5.7(5.3)
Self-Esteem	Pre 42.4(7.7) Post 52.5(12.3)	48.3(15.8) 52.8(13.1)	45.4(11.1) 45.7(13.1)

Average Ratings of Anxiety, Depression  
and Self-Esteem Before and After Treatment

	Pre	Post	df	F
Anxiety Mean, SD	6.2 1.7	5.6 1.6	1,27	4.59*
Depression Mean SD	7.0 5.7	4.6 4.7	1,27	10.43*
Self-Esteem Mean SD	45.3 11.5	50.3 12.8	1,27	12.29*

\*  $p < .05$

Mean Ratings on MMPI Validity and  
Clinical Scales

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<u>Scale Validity</u>	<u>Mean</u>
L Scale	50.7
F Scale	58.3
K Scale	54.3

Clinical

Scale 1 (Hypochondriasis)	46.1
Scale 2 (Depression)	64.5
Scale 3 (Hysteria)	62.3
Scale 4 (Psychopathic Deviate)	58.9
Scale 5 (Masculinity/Femininity)	53.3
Scale 6 (Paranoia)	56.8
Scale 7 (Psychasthenia)	54.4
Scale 8 (Schizophrenia)	51.8
Scale 9 (Hypomania)	52.0
Scale 10 (Social Introversion)	55.4

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Multiple Regression of Demographic,  
Psychometric and Treatment Group  
Variables

Outcome Measure: Percent Improvement in Sleep Onset

A. Summary Statistics after entering psychometric and demographic variables and before entering "treatment.group" variable.

Analysis of Variance

	DF	SS	MS
Regression	7	5325.25	760.78
Residual	27	14335.27	651.60

F = 1.16

SIG F. = 0.36

Multiple R	.52	R SQ CHANGE	0.27
R Square	.27	F CHANGE	1.16
Adjusted R SQ.	.03	SIGNIF F CHANGE	0.36
Standard Error	25.52		

Variables in Equation

<u>Variable</u>	<u>Beta</u>	<u>F</u>	<u>SIG. OF RSQ CHANGE</u>
Self-Concept Scale	-0.14	.44	ns
Depression Scale	0.06	.10	ns
Age	-0.08	.12	ns
MMPI 2 (F Scale)	.23	.33	ns
Anxiety Score	0.08	.16	ns
Duration of Insomnia	0.07	.08	ns
Sex of Subject	0.40	4.56	ns

## B. Summary Statistics after entering the "treatment group" variable.

Analysis of Variance

	DF	SS	MS
Regression	9	13729.17	1525.46
Residual	20	5931.62	296.58

F = 5.14

SIG. F = 0.001

Multiple R	.83	R SQ CHANGE	0.42
R Square	.69	F CHANGE	14.16
Adjusted R	.56	SIG F CHANGE	0.0001
Standard Error	77.22		

Variables in the Equation

<u>Variable</u>	<u>Beta</u>	<u>F</u>	<u>SIG. OF R<sup>2</sup> CHANGE</u>
Self-Concept Scale	-0.14	.95	ns
Depression Scale	.12	.82	ns
Age	-0.04	.05	ns
MMPI (2)	-0.24	.329	ns
Anxiety Scale	-0.00	.001	ns
Duration of Insomnia	-0.00	.002	ns
*Sex of Subject	.36	7.54	p < .01
*Treatment Group Variable			
Contrast 1	.00	.00	ns
Contrast 2	.66	28.33	p < .000

\* These variables were dummy coded as follows:

1. Sex of Subject: males 0; females 1.
2. Treatment Groups: Contrast 1 = 1,-1,0  
Contrast 2 = 1,1,-2

Mean Latency to Sleep Onset Before and  
After Treatment for the Males and Females  
by Treatment Groups

		<u>Treatment Condition</u>		
		Group 1, (Therapist)	Group 2 (Therapist by phone)	Group 3 (No Therapist Contact)
		N= 3 males; 7 females    6 males; 4 females    5 males; 5 females		
<u>Sex of Subject</u>				
Males	Pre	87 (36.5)	75.5 (53.5)	83.0 (27.2)
	Post	48 (29.6)	51.0 (46.0)	74.6 (34.3)
Females	Pre	83 (62.4)	84.0 (44.4)	67.0 (15.6)
	Post	25 (13.8)	24.5 (14.2)	48.8 (20.0)

APPENDIX N:

Means and Standard Deviations of  
Ratings of Therapist Warmth  
and Treatment Rationale by  
Treatment Groups

		<u>Treatment Conditions</u>		
		Group 1 (Therapist)	Group 2 (Therapist by phone)	Group 3 (No Therapist Contact)
<u>Variable</u>				
Rating of Therapist Warmth	M	4.5	4.4	4.5
	SD	1.0	.8	.9
Rating of Treatment Rationale	M	4.3	4.1	4.0
	SD	.6	.7	.8

APPENDIX O:

Means and Standard Deviations  
for the Primary Dependent  
Measures Separated by Treat-  
ment Condition and Subject  
Gender

<u>Group Membership</u>							
		Group 1 (Therapist)		Group 2 (Therapist by Phone)		Group 3 (No Therapist Contact)	
N =		3	7	6	4	5	5
<u>Sex of Subject</u>		Male	Female	Male	Female	Male	Female
<u>Testing Period</u>		Pre/Post	Pre/Post	Pre/Post	Pre/Post	Pre/Post	Pre/Post
<u>Dependent Measure</u>							
A) Sleep Onset	$\bar{X}$	87/48	83/25	75/51	84/24	83/74	57/48
(in minutes)	SD	36/29	62/13	53/46	44/14	27/34	15/20
B) Hours of Sleep	$\bar{X}$	6.5/6.9	5.5/6.4	6.2/6.6	5.0/5.7	6.7/6.8	6.0/6.1
	SD	.6/ .9	.4/ .4	.7/ .8	2.9/3.3	1.4/1.7	1.0/1.0
C) Difficulty Falling Asleep	$\bar{X}$	3.0/2.0	2.9/1.5	3.0/1.9	3.1/2.0	.8/ .9	.3/ .4
	SD	.6/ .3	.5/ .5	.4/ .9	.5/ .9	.8/ .9	.3/ .4
D) Morning Restedness	$\bar{X}$	2.4/1.9	3.0/1.8	2.9/2.5	2.8/1.9	2.7/2.5	3.1/2.6
	SD	.8/ .9	.5/ .6	.7/ .7	.9/ .9	.6/ .5	.4/ .4