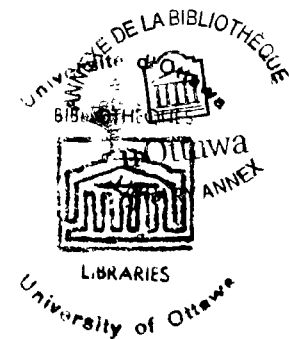


**GENERALIZED ANXIETY AND SLEEP-ONSET INSOMNIA :
EVALUATION OF TREATMENT USING ANXIETY MANAGEMENT
TRAINING**

by Marcel J. Viens

**Thesis Submitted as a Partial Requirement
for the Degree of Doctor in Philosophy**



University of Ottawa

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TO B.F. SKINNER,
WITHOUT WHOM PSYCHOLOGY WOULD NOT HAVE THE SAME
MEANING.

PREFACE

SLEEP

To thieves your wealth is beyond reason
To kill for you is really no treason
The gifts for those you so freely hand
Changes sparkling diamonds into piles of sand

To lovers your touch is so ever caring
Nestled in your arms their hearts never mending
Longing for love of many times gone by
You give them refuge within bitter sweet lies

To sinners your threat is always foreboding
Unearthing vile demons even time has forgotten
Image flashing, a mere ghost in your grasp
The executioner simply burns them from dust into ash

To dreamers euphoria is all they can seek
The colors of living make them mortal and weak
So they hide from the light, in a world of delusions
Conquering far kingdoms, mere shadows of illusions

To philosophers your secrets are but great mysteries
The search for your truth has left them so weary
You laugh at their trials, you dangle the key
How many one day will you really set free?

Sleep my friend, please hear what I say
No time for your lies, no time for your play
Wherever you hide, be it near or so far
I'll find you some day, I'll know who you are...

I wrote this little poem amidst the stench of collodion and acetone, under a dim light which barely enabled me to witness the polygraph's interpretation of the subject's EEG. As I was watching over one of my motionless subjects, this creative pastime was a personal way of coping with my self-imposed sleep deprivation.

As a group, sleep researchers are a special breed of scientists. Even though they love their work, their task is often tedious, slow going and at times downright unstimulating. But the rewards are beyond compare. The men and women who probe sleep are, in fact, reaching towards our body's innermost boundaries. I can only say that I am proud to be part of such an elite group of individuals.

As a young Ph.D. candidate and a novel sleep researcher, I fondly remember approaching my supervisor, Dr. Joseph De Koninck, with a skeleton-like proposal of this thesis. I can still picture his frown as he warned me of how insomniacs were difficult to work with. Now, when I reflect back on that particular moment, even though I know my supervisor was right, I am glad I did not listen to him.

But at the time, my youthfulness was also my greatest enemy, for youth and inexperience too often go hand in hand. What I first envisioned as being "a nice little project" became an undertaking at times too complex to handle all by myself. This section is reserved for those individuals who perseverated with me through it all.

Foremost, I feel deeply indebted towards Dr. Joseph De Koninck. The originality of his feedback, his support and his total commitment to the completion of this project has shown me that he is not only an excellent supervisor but a dear friend also.

This research would have never lifted off the ground without the invaluable help of many of my colleagues. Here is my chance, finally, to thank all of them personally.

First, I would like to extend my deepest gratitude to a special friend of mine, Dr. Dominique Lorrain, for her friendship and her precious work as a co-therapist in this project. Second, I would also wish to thank Gilles Hébert, Jean Grenier, Louise Couture, Jean-Louis Beaulé, Marie Taillon, Bervely Leblanc, and Maryse Paré, all of whom gave many hours of their time in preparing and watching over the numerous subjects who came and went through our laboratory doors. Finally, my deepest appreciation to Dr. Pierre Mercier for his advice concerning computer programming matters and statistical analysis, as well as to Gregory Christ for his friendship, his good humor and his insightful comments in proofreading this document.

Last, but not least, I would like to extend my warm gratitude to Liette, Brenda and especially my beloved wife Marie, all of whom offered me their special moral support through the long years it took me to finalize this thesis.

TABLE OF CONTENTS

	Dedication :	I
	Preface :	II
	Table of Contents :	V
	List of Tables :	VIII
	List of Figures :	IX
	Abstract :	X
	<u>CHAPTER 1 : THE LITERATURE</u> :	1
1.	INTRODUCTION :	1
2.	BEHAVIORAL TREATMENTS OF INSOMNIA :	5
	Relaxation Techniques :	5
	Progressive Relaxation :	5
	Autogenic Training :	7
	Hypnosis :	8
	Systematic Desensitization :	9
	Stimulus Control :	10
	Biofeedback :	14
	Paradoxical Intention :	16
	Sleep Restriction Therapy :	18
	Sleep Hygiene :	20
3.	CONCLUSION :	21
	Review of Success Rates :	21
	Objective versus Subjective Measures :	21
	Do we need Cognitions ? :	23
4.	COGNITIONS AND INSOMNIA :	25
	Insomnia and the Attribution Process :	25
	Somatic versus Cognitive Arousal :	29
	Somatic Arousal + Cognitive Arousal =	
	Anxiety :	32

5. THE ROLE OF ANXIETY IN THE MAINTENANCE OF	
SLEEP-ONSET INSOMNIA :	35
Anxiety and Insomnia :	35
State versus Trait Anxiety :	37
Trait Anxiety and Insomnia :	38
Insomnia and Vigilance :	41
Hypotheses :	44
<u>CHAPTER 2 : THE STUDY</u> :	46
METHOD :	46
Subjects :	46
Design and Procedure :	48
Treatments :	51
Progressive Relaxation :	52
Anxiety Management Training : ...	52
Measurements :	53
1) Polysomnography :	53
2) SOL Monitor :	53
3) Sleep Positions :	54
4) Psychological measures :	54
Personality :	54
Anxiety :	54
Depression :	55
Vigilance :	55
RESULTS :	57
The Measurement of Sleep-Onset Latency : .	57
AMT versus PR :	57
High Anxiety (HI-A) versus Low Anxiety	
(LO-A) :	66
Correlates of Improvements in Sleep-Onset	

The Chicken or the Egg :	
A Behavioral Model :	84
On the Measures Used :	87
1) The Psychological Tests :	87
2) Polysomnographic measures : ..	88
3) The SOL Monitor :	89
4) The Four-Choice Reaction Time Test :	92
On Improving this Study :	94
CONCLUSION :	97
REFERENCES :	98
APPENDIX A : CONSENT FORM :	111
APPENDIX B : QUESTIONNAIRES USED :	114

LIST OF TABLES

<u>Table</u>	<u>Title</u>	<u>Page</u>
1	Number of Subjects & Age Range (AMT versus PR). . . .	50
2	Means & S.E.M. for the Personality and Performance Tests (PR & AMT)	59
3	Means & S.E.M. for the Different Sleep Parameters (PR & AMT)	60
4	Number of Subjects & Age Range (HI-A versus LO-A) . .	68
5	Means & S.E.M. for the Personality and Performance Tests (HI-A & LO-A).	69
6	Means & S.E.M. for the Different Sleep Parameters (HI-A & LO-A).	70
7	Correlation Coefficients Between SOL Improvements at Home and Changes in Psychological Measures	75
8	Correlation Coefficients Between SOL Improvements at Home and Changes in Laboratory Sleep Parameters, Sleep Satisfaction and Vigilance.	76

LIST OF FIGURES

<u>Figure</u>	<u>Title</u>	<u>Page</u>
1	Experimental Design	49
2	Before and After Treatment Scores on the MMPI for All the Subjects	61
3	Before and After Treatment Scores on the MMPI for the AMT Group.	62
4	Before and After Treatment Scores on the MMPI for the PR Group	64
5	Before and After Treatment Scores on the MMPI for the LO-A Group.	71
6	Before and After Treatment Scores on the MMPI for the HI-A Group.	73
7	A Behavioral Model of the Effects of AMT on Trait-A and Depression.	85

ABSTRACT

In this study, we tested the notion that generalized anxiety is a predominant factor in the maintenance of psychologically related sleep-onset insomnia. This was achieved by monitoring trait anxiety, as defined by Spielberger (1966), in sleep-onset insomniac subjects who received either Anxiety Management Training (AMT), which has been shown to reduce trait anxiety, or progressive relaxation (PR), one of the most popular treatments for sleep-onset insomnia.

Twenty subjects (14 females and 6 males, age 19 to 63), screened by an interview, the MMPI, and one night of polysomnography to present moderate to severe sleep-onset chronic insomnia (taking at least 45 minutes to fall asleep, four nights per week), were first asked to monitor their Sleep-Onset Latency (SOL) for a three week baseline period at home using our SOL clock device. Then, ten received AMT for nine weeks while the remaining ten were trained in the use of PR. All subjects were measured before and after therapy using sleep laboratory recordings (three nights each), the Spielberger Trait Anxiety Inventory, the MMPI, the Beck Depression Inventory, and the Four-Choice Reaction Time test. Daily home sleep-onset measures with the SOL clock device were also taken during therapy.

Results showed that no real distinction could be made between the two therapies, i.e., AMT was at least as effective in reducing sleep-onset insomnia as PR. However, insomniacs receiving AMT appeared to significantly improve on a greater number of monitored psychological and physiological correlates of sleep-onset insomnia than subjects receiving PR. This showed that to a certain extent, AMT would be the better choice over PR.

The main hypothesis of a relationship between trait anxiety and sleep-onset insomnia has been generally supported in this study. However, the reasons for this improvement are still somewhat unclear. Our results demonstrated that, in addition to trait anxiety being one of the principle etiological factors responsible for the maintenance of sleep-onset insomnia, a reduction in the overall reported level of depression was closely associated with a significant improvement in SOL, sleep satisfaction and other psychological measures.

To explain this, a behavioral model was proposed the intended usefulness of which will be to guide future research in understanding the interactional role played by both depression and anxiety in the maintenance of sleep-onset insomnia.

CHAPTER 1 : THE LITERATURE

1. INTRODUCTION

For an organism to survive, one of the basic requirements other than food and water is rest. In many species, including humans, rest often takes the form of sleep. In fact, an individual sleeping an average of eight hours per day spends about a third of his life asleep. Any disorder affecting this function is not to be taken lightly. One of the side effects resulting from the pressures that our societies impose on us is often, among other things, one form or another of sleep disorder. A recent survey (Bixler et al., 1979) conducted on a sample population from Los Angeles (N= 1006) suggests that about 50% of the individuals living in this large metropolitan area have suffered or are suffering from a sleep disorder, the most common of which is insomnia (40%). More recently, Walsh et al., (1986) reported that "about 50 percent of Americans over 15 years of age have difficulty sleeping at some point in their lives, and over 35 million individuals report chronic insomnia" (p.185). Such observations stress the necessity for researchers and specialists to identify the causes and develop efficient treatment methods to deal with this problem. Early studies of insomnia generally revolved around the faulty premise that only one type of sleep disorder existed: a bad night's sleep. This philosophy was reflected in such diagnostic classifications as the DSM-II, which provided only one category for all sleep disorders (306.4) .

As research progressed in this field, it became apparent that a redefinition of insomnia into sub-categories was needed. Price (1974) suggested 5 types of insomnia according to their accompanying behaviors: 1- difficulty in falling asleep or taking a long time to fall asleep, which is the behavior most researched in the literature; 2- early awakenings; 3- frequent

nocturnal awakenings; 4- feelings of fatigue and dissatisfaction with sleep upon awakening; and, 5- a combination of the above. It has been shown that when overnight electroencephalographic recordings are made, self-reported insomniacs in fact may yield normal sleep patterns (Dement, 1972). This suggests that a distinction should be made between those sleeping normally but reporting sleep disturbances and those in whom physiological measures indicate the existence of a true sleep disorder.

More recently, a very valuable tool for sleep researchers has been published through the Association of Sleep Disorders Centers (ASDC). The "Diagnostic Classification of Sleep and Arousal Disorders" (Sleep, Vol. 2, No. 1, 1979) separates insomnias (disorders of initiating and maintaining sleep or DIMS) from the other three major sleep disorder categories: disorders of excessive somnolence (DOES), such as narcolepsy and hypersomnolence; disorders of sleep-wake schedules; and finally, parasomnias which include sleep disturbances such as sleep-walking, sleep terrors, and sleep-related enuresis. In addition, other nomenclatures stress that insomnia can also be classified as being transient (situational) or persistent (chronic).

For a condition to earn the label of insomnia, it must be based on a complaint from the client (fatigue and/or impairment in daily functioning). Thus, insomniacs must be distinguished from short sleepers who are satisfied with a very limited amount of sleep. Since the causes of insomnia can vary from strictly organic to physiological or psychophysiological, a multidisciplinary diagnostic and treatment approach is important (De Koninck and Godbout, 1985).

According to Montgomery and al. (1975), insomnia is increasingly treated with hypnotic and sedative drugs in our western culture. During the year 1977, physicians, gave nearly 26 million prescriptions for hypnotic medications (Institute of Medicine, 1979). This has prompted many investigations into the use of drugs for the treatment of insomnia. Kripke (1983)

estimated current usage of hypnotics in the U.S. at about 40 million yearly prescriptions. Mellinger et al. (1985) found that in 1979, 2.6% of the general population had used hypnotics. Ribordy and Denney (1977), after conducting a thorough review of the literature, arrived at five criticisms against the use of drugs in the treatment of insomnia: A- Extended use of drugs leads to tolerance effects; B- There is a danger of carry-over effects, thus morning drowsiness, nausea, headaches and other symptoms may occur from drug build up and/or slow metabolic elimination; C- There is a proven alteration in sleep patterns; D- Rebound effects are to be expected; and, E- Possible attributional effects are not uncommon. For example, a psychological dependency on the drug may develop even though it often proves itself ineffective in the long run. These conclusions were later generalized across a wide array of hypnotic drugs (Kales et al., 1982 A; 1982 B). To this list, still another criticism can be added. Scharf and Brown (1986), as cited by Lacks (1987), suggest that "Because the elderly consume a variety of drugs, there are problems with toxic interaction. Changes take place in the older adult's ability to absorb, metabolize, and excrete drugs. Consequently, the toxicity profile of a particular hypnotic may be very different for the elderly than for younger adults" (Lacks, 1987, pp. 13). Kales et al. (1983) presented a recent extended review of sleep-drug side-effects especially in the area of rebound insomnia and rebound anxiety, restating the need for reservation in prescribing this type of medication. Walsh et al. (1986) suggest that the best use of hypnotics may be with healthy individuals experiencing transient or short-term sleep problems. Still, the preferred treatment approaches, according to Montplaisir (1984), are nonpharmacological.

Because of these criticisms, several researchers in psychology and psychiatry have been interested in an increasingly popular alternative: cognitive and/or behavior therapy. Over the years, many learning principles and variations of these have been applied to the treatment of sleep disorders. Before discussing the research literature on the various components of insomnia, we will present a brief review of the different behavioral therapies applied over the last

two decades. Since each technique aims at alleviating what their proponents claim to be the key contributing factors of insomnia, separate assessment of the individual effectiveness of these techniques in actual therapy, will facilitate the discussion surrounding the potential causes which have been researched.

2. BEHAVIORAL TREATMENTS OF INSOMNIA

Relaxation techniques

Progressive Relaxation

One of the most widely used techniques in the behavioral treatment of insomnia is relaxation. The first report of muscle relaxation as a treatment procedure was made by Jacobson (1938). His own technique involved the systematic tensing and releasing of progressively more muscle groups until all the body muscles could be relaxed simultaneously at will without any further muscle tensing.

In a study by Borkovec and Fowles (1973), progressive relaxation was as successful as hypnotic relaxation in treating female college students. In the same study, self-relaxation produced nearly equal improvement as progressive and hypnotic relaxation and all three approaches were superior to giving no treatment. The authors attributed their results to one or a combination of three specific factors: demand characteristics, placebo effects, and attention focusing. Weil and Goldfried (1973) were successful in treating an 11 year old girl using taped instructions for relaxation which were listened to just before going to sleep. These taped directions were gradually shortened until the girl could fall asleep within 5 minutes without any help whatsoever. This case report was a successful example of an "in vivo" treatment of insomnia using a mechanical device (taped instructions) as an aid. This eliminated the need for the therapist to be present thus saving time and money for both the therapist and the client. Haynes et al. (1974), using fourteen insomniac university students, showed that significant improvement in sleep patterns was attained in the relaxation group following six one-half hour

sessions when compared to a placebo therapy group which demonstrated only slight overall improvement. Even though the effectiveness of relaxation was apparent, the authors warned that it could be due in part to contributing factors such as expectation and demand characteristics. In addition, Lick and Heffler (1977) further demonstrated that relaxation training was significantly more effective in treating their group of 40 insomniacs than placebo and no-treatment control.

Anderson (1979) reported successfully treating a 13 year old boy with a four month history of insomnia by combining relaxation training, to reduce the patient's level of tension, with a behavioral program aimed at reducing parental attention surrounding the boy's symptoms. Turner and Ascher (1979 A) further supported the effectiveness of progressive relaxation by comparing it to two other promising therapies, stimulus control and paradoxical intention. All three techniques were equal but significantly superior to a waiting list control group in reducing sleep complaints.

Finally, Cannici, Malcolm and Peek (1983) used a 3 day muscle relaxation program in treating 30 cancer patients suffering from insomnia secondary to their cancerous condition. Of the 26 subjects available for follow-up 3 months later, it was found that the pre-therapy sleep onset latency mean of 124 minutes, was down and maintained to a new mean of about 29 minutes. The authors attributed their success to the secondary nature of the insomnia to a medical disease as compared to primary insomnia which they speculate is the principle problem in itself.

More recently, certain authors have reviewed past studies and attempted to evaluate the overall effectiveness of progressive relaxation. Ladouceur and Gros-Louis (1984) and Lacks (1987) both report treatment efficacy lasting up to one year after its termination. As reported by Borkovec (1982), only four of the 17 studies reviewed used EEG measures (i.e., Borkovec and Weerts, 1976). All the other studies relied heavily on data collected through self-reports. The percentage of improvement averaged 45% in the self-report studies and 59% in the remaining

research employing EEG recordings. An average of these percentages is in agreement with averages estimated by Bootzin and Nicassio (1978; 50%) and Lichstein and Fisher (1985; 43%). Finally, progressive relaxation "may be the treatment of choice where qualitative improvement is perceived by the patient to be more important than quantitative change" (Espie et al., 1989, pp. 87).

Autogenic Training

A variant of relaxation training termed autogenic training, developed by Schultz and Luthe (1959), consists of suggesting feelings of warmth and heaviness to the patient until he/she is completely relaxed. Kahn, Baker and Weiss (1968) used autogenic training in treating 16 students in a special 2 week training program. Out of 13 students reached after treatment, 11 reported improvement after a 1 year follow-up. Of those responding, a significant majority reported being less nervous, tense and anxious while being more efficient in their studies and their interpersonal relationships. In another study, Nicassio and Bootzin (1974) compared autogenic training to progressive relaxation in 30 adults reporting difficulties falling asleep. Both techniques were equally successful in improving the time to fall asleep as indicated by global measures of improvement and by reduction in time to fall asleep. Nevertheless, at a six-month follow-up, subjective self-report showed less improvement in both cases.

In an attempt to study the effects of relaxation on two postulated types of insomnia, Borkovec et al. (1979) compared the influence of relaxation with tension (progressive relaxation) to relaxation without any tension (autogenic training). In order to do this, they divided a group of 29 subjects into either "idiopathic" insomniacs or "pseudoinсомniacs". "Pseudoinсомniacs" show little evidence of sleep deficit according to EEG criteria. These individuals are in clear contrast to "idiopathic" insomniacs who show all of the abnormal EEG criteria expected in

insomnia. Results showed that "as predicted, tension-release relaxation was significantly more effective than the other two conditions (no-tension-release relaxation or no-treatment) on subjective sleep measures, regardless of insomnia subtypes and on objective sleep measures only for idiopathic insomniacs." (Borkovec et al., 1979, p. 37).¹

Coursey et al. (1980) using 22 chronic sleep-onset insomniacs, compared autogenic relaxation to electrosleep therapy. This last technique is a physiological procedure where electrodes (which transmit intermittent impulses to the brain) are placed at strategic areas on the scalp. These impulses have been shown to induce to a certain degree sleep in various populations such as normal adults, insomniacs and hypertensive patients (Nagata et al., 1981). Coursey and his colleagues found that while none of the individuals treated with electrosleep therapy improved on all-night laboratory electroencephalographic measures, half of the relaxation group improved significantly. Those who responded to treatment were found to be different from non-responders in sleep habits, psychological make-up, in motivation to meet treatment demands, and finally, in the amount of external stress they were enduring.

The success rate of autogenic training can at best be described as moderate (Ladouceur and Gros-Louis, 1984). Even though Nicassio and Bootzin (1974) rated the technique as quite good, other positive outcome studies (Graham et al., 1975; Johnson, 1975; and Kahn, Baker and Weiss, 1968) are believed to contain methodological weaknesses which in turn shed doubts on their validity.

Hypnosis

Others have concentrated their efforts on hypnosis as a possible tool for inducing

¹It is important to add that even though 25% of all insomnia patients are nowadays labeled "pseudoinsomniacs", some researchers still firmly believe that this "disorder" in fact does not exist (Trinder, 1988).

relaxation and thus treating insomnia. In this procedure, subjects are hypnotized and given post-hypnotic suggestions of increased relaxation and well being at bedtime. To this date, promising research has been mostly limited to single case reports (Tiller, 1967; Tood and Kelley, 1970). However, hypnotically produced relaxation was compared to a proven technique (progressive relaxation) and shown to produce significant improvements (Borkovec and Fowles, 1973). Fry (1973), using 28 patients, showed overall improved sleep when hypnotic induction was used.

Finally, because of the restricted number of studies and their questionable results, authors like Stanton (1975) rate the success of hypnosis with insomniacs as moderate at best.

Systematic Desensitization

Systematic desensitization is another well-known behavioral procedure where relaxation is a component. The individual is first instructed on how to relax properly. Then, using a hierarchy of anxiety producing scenes, all related to sleep, the client is asked to imagine the least anxiety provoking scene in the hierarchy. When anxiety is experienced, the client is instructed to switch to relaxation. This procedure is repeated until the most anxiety producing scene related to sleep loses its potential to elicit an anxiety response. At that point, the client imagines the next to last scene in the list and so on until the last and most anxiety provoking scene has been successfully mastered.

Geer and Katkin (1966) successfully treated a 29 year old female suffering from severe insomnia using a variant of systematic desensitization where no fear hierarchy was established. An 8 month follow-up showed that improvement was still maintained. This same technique unfortunately was reported unsuccessful by Evans and Bond (1969) who later opted for a classical conditioning procedure in treating their patient. Borkovec, Steinmark and Nau (1973),

using twenty-four sleep-disturbed subjects, compared relaxation-alone, desensitization with relaxation, and desensitization without relaxation. After a total of three sessions for each group, the researchers discovered that all three techniques were equally successful in treating mild insomnia. For more severe cases, it was found that desensitization plus relaxation was the most promising therapy. In a similar study, Gershman and Clouser (1974) used an automated approach to compare systematic desensitization to muscle relaxation and again both were equally effective in treating insomnia.

Most of the studies cited above suffer from methodological weaknesses (i.e., small sample sizes, strong reliance on subjective measures of sleep). Therefore, their results should be interpreted with caution (Ladouceur and Gros-Louis, 1984). In its actual form, systematic desensitization offers only moderate improvement of insomnia. According to some authors, the standardized application of the technique may be too rigid, and therefore hardly adaptable to individual differences found across insomniacs (Bootzin and Nicassio, 1978; Ribordy and Denney, 1977; Steinmark and Borkovec, 1974).

Stimulus Control

As reported earlier, classical conditioning has also proven effective in treating certain types of sleep disorders. The most commonly used version of the procedure is to instruct clients to cease all behaviors which are known to interfere with the "behavior of sleep". Thus, in addition to not being allowed to read, write or engage in any activity but sleeping in their bed, clients, if not able to fall asleep, have to get out of bed and return only if they feel they can resume their night's sleep.

In the Evans and Bond (1969) experiment mentioned earlier, the authors successfully paired counting episodes with drug-induced sleep onset. The subject was asked to count while

slowly being injected with a sleep inducing drug. It was hoped that successive pairing of that sort would help the subject to fall asleep by simply counting in bed. In fact, towards the end of therapy, when the subject counted in bed, he/she fell asleep much faster, with no further need for drugs.

By ceasing all sleep incompatible behaviors in the bedroom, Bootzin (1972) was successful in treating one of his clients. The same experiment was later repeated by Haynes, Price and Simons (1975) using an ABAB design on four volunteer subjects. All of them showed marked decreases in time to sleep onset thus validating the usefulness of the technique. In another study, Pendleton and Tasto (1976) were able to condition relaxation to the regular beat of a metronome which in turn allowed their subject to fall asleep faster. In fact, their technique proved to be as successful as progressive relaxation in treating sleep-onset insomnia. More recently, Norton and DeLucas (1979) treated a 22 year old male with a 6 year history of insomnia. For this, they used a combination of relaxation training and stimulus control. The former reduced sleep onset while the latter, combined with self-punishment ("filling out job application forms"), dealt with nocturnal awakenings quite effectively.

Zwart and Lisman (1979), in an attempt to examine the components responsible for the efficacy of the stimulus control treatment of sleep-onset insomnia, have also generated additional data supporting the treatment's success. These authors postulated three hypotheses for the success of the stimulus control techniques, namely 1- contingent disruption of bed and bedtime as cues for the arousal which is possibly associated with worrying, tossing and turning in bed; 2- the fact of getting out of bed if unable to sleep may be seen as quite aversive; and 3- engaging in an unfamiliar activity, such as getting out of bed and doing something, may decrease the apparent duration of the interval (thus time awakened seem less longer). Turner and Ascher (1979B) gave further credit to the technique by showing that it was also quite effective in treating subjects with severe sleep-onset insomnia. In addition, the same authors showed that a stimulus

control therapy also greatly reduced the usage of sedative-hypnotic drugs by the subjects. Shealy (1979) succeeded in showing that, for a sample of 14 college female undergraduates suffering from mild insomnia, a combination of stimulus control and relaxation could yield better results in treating their mild insomnia than relaxation alone.

Focusing on a different population, Puder et al. (1983) demonstrated that a short-term stimulus control treatment of insomnia could be efficacious in treating 16 subjects aged 60 and over. At the end of the four week treatment program, 10 of 16 insomniacs reduced their sleep latency by 50% or more. In addition, half of the subjects reported at post-treatment that their sleep latency reached or exceeded 30 minutes not more than once a week.

More recently, Lacks et al. (1983A) compared the effectiveness of stimulus control, progressive relaxation and paradoxical intention techniques in treating several individuals suffering from one of three different degrees of sleep-onset insomnias (mild, moderate, or severe). Overall, the authors reported that the stimulus control approach yielded the most rapid and pronounced results, and this for all three levels of severity. Of importance was the use of subjective sleep-onset latency reports which lasted all through the study.

The same year, Lacks et Al. (1983B) were successful in demonstrating that stimulus control was not only efficacious in treating sleep-onset insomnia, but sleep-maintenance insomnia as well. This last finding has later been supported by Davies et al. (1986) using 34 sleep-maintenance insomniacs ranging in age from 35 to 78 years. The treatment used, called countercontrol (a variant of stimulus control), helped reduce the sleep complaints of the overall group by about 30%.

Haynes et al. (1982), being somewhat more critical of the stimulus control procedure, attempted to assess this behavioral conceptualization of sleep-onset insomnia. In a series of 3 successive studies using varying numbers of insomniacs and non-insomniacs, they concluded that : 1- insomniacs and non-insomniacs do not significantly vary on reported frequency of

sleep-incompatible behaviors, nap frequency or location, or variability in sleep. In addition, laboratory measures of sleep-onset latency did not concur with the above mentioned variables; 2- no relationship has been found between sleep incompatible behaviors (as self-monitored at home) and sleep-onset insomnia; and 3- nevertheless, as predicted by the stimulus control paradigm, insomniacs in the sleep laboratory took less time to fall asleep than the usual latencies reported at home. This is attributed to the fact that stimuli associated with wakefulness at home are no longer present in the sleep laboratory.

Finally, insomniacs suffering from somatized tension and negative conditioning are now said to have developed persistent psychophysiological (learned) insomnia (Hauri and Fisher, 1986). These individuals are known to have difficulties initiating and maintaining sleep but nevertheless show normal sleep staging. Jacobs et al. (1988), in reviewing the role of polysomnography in the differential diagnosis of chronic insomnia, stated that "Of the 23 patients who were diagnosed as suffering from persistent psychophysiological disorders of initiating and maintaining sleep, nine (39%) were found to sleep better in the laboratory than they reported sleeping at home, supporting the clinical impression that patients have conditioned insomnia in their home environments" (pp. 348).

Overall, stimulus control is the second most studied technique in the insomnia literature. In addition, its success rate is quite impressive. Borkovec (1982), based on self-report ratings of improvement, evaluated the success rate at 70%, while Lichstein and Fisher (1985), after reviewing 13 studies in all, rated the technique's efficacy at 58%. Of interest is the fact that the stimulus control approach has been proven effective with many different age groups and various levels of severity of sleep problems.

More recently, Espie et al. (1989), in a replication study of Turner and Ascher's (1979) original design in which they compared stimulus control, progressive relaxation and paradoxical intention, concluded that "Stimulus Control appears to be effective in habit-restructuring but

patients will not necessarily become more contented with their sleep" (pp. 87).

With the exception of Morin et al. (1986) who showed improvements in an elderly population by using an electromechanical timer, most studies which have reported positive outcomes using stimulus control may be criticized for not including objective measures of sleep (Lacks, 1987). There has been no study of stimulus control in the laboratory. Probably, this would present a difficulty since by itself, the laboratory is a comprehensive controlling stimulus.

Biofeedback

Other behavioral techniques have also proven useful in the treatment of insomnia but, because of their limitations (applicability to a small sample of the population, monetary costs, etc.), up to now they have been less popular. Biofeedback is a good example. Through this technique, an attempt is made to increase the client's awareness of some otherwise unperceivable physiological function and then to control it. Research has focused on three specific feedback modalities: EMG (mostly from the frontalis muscle), EEG (alpha and theta waves), and SMR (sensorimotor rhythm, i.e.; breathing, heartbeat).

Raskin, Johnson and Rondestvedt (1973) were able to improve the sleep patterns of 5 of their 6 subjects using biofeedback of the frontalis muscle as a way of learning to relax and fall asleep. Nicassio et al. (1976) obtained similar results in their own study. Haynes, Sides and Lockwood (1977) compared biofeedback of the frontalis muscle to relaxation instructions as intervention methods with insomnia in twenty-four subjects who were assigned to one of 3 groups: biofeedback, relaxation or control. Six bi-weekly sessions of the assigned therapy was received by each subject. It was found that both relaxation and biofeedback were equally effective and superior to the control group receiving no treatment.

As reported by Ribordy and Denney (1977), other researchers such as Budzynski and

Stoyva (1969), using alpha waves and muscle feedback, have had some success in inducing sleep but further investigation using this possible combination (EMG and EEG (alpha waves)) is still needed. The use of EEG theta biofeedback was successful in treating a 42 year old female complaining of chronic sleep-onset insomnia. This improvement was maintained after a three month follow-up (Bell, 1979).

Hauri (1978), in an attempt to evaluate the efficacy of various biofeedback techniques, compared frontalis EMG biofeedback, EMG/theta feedback and SMR (sensorimotor rhythm) feedback to a non-feedback control group. He concluded that: 1- both EMG and SMR feedback were successful in treating some forms of serious insomnia; 2- EMG biofeedback was superior to other feedback techniques in relieving complaints about insomnia; and 3- in the laboratory, using polygraphic measures of sleep, SMR feedback yielded the best results. Thus specific biofeedback techniques are useful with certain forms of insomnia highlighting the need for proper assessment prior to treatment.

In sum, biofeedback is not the ideal choice of treatment with insomniacs. Results from different studies are quite inconsistent. In addition, it is not yet clear what feedback modality is the most appropriate. The reduction of frontalis EMG does not mean that the whole body has reached a relaxed state. This has to be further assessed. As well, EEG (alpha waves) are difficult to control, thus severely limiting clinical use of this measure. Finally, little research has been conducted using SMR (sensorimotor rhythm) feedback. Thus, considering its high cost and low applicability, it is ill-advised at this point in time to use biofeedback to treat insomniac clients.

Paradoxical Intention

Paradoxical intention, a procedure developed by Frankl (1960, 1975) functions on the principle that exaggerating a behavior, instead of opposing it, will in fact lead to its cessation. Thus instructing the subjects to stay awake as long as possible relieves the patient's anxiety of performing well and going to sleep as quickly as possible. This technique was shown to be successful in treating five cases where muscle relaxation and systematic desensitization yielded no results (Asher and Efran, 1978). Additional studies have also supported the efficacy of paradoxical intention in treating mild to chronic insomnia. Relinger, Bornstein and Mungas (1978) used paradoxical intention in treating an individual suffering from chronic insomnia. To control for improvement attributable to demand characteristics alone, counterdemand manipulations were utilized during the treatment period. Significant improvement on five out of eight self-recorded sleep dimensions were reported. These gains were maintained over a 12 month follow-up period. The authors explain their results in terms of successfully breaking the "exacerbation cycle" where one's attempts to fall asleep faster only evokes more anxiety thus not allowing the individual to rest before falling asleep.

In another study, Turner and Ascher (1979) compared a paradoxical intention group to a progressive relaxation and a stimulus control group. These three active techniques were all superior to a standard placebo and a waiting list group. Compared to each other, they were all equally effective in treating the insomnia problem.

The same authors (Ascher and Turner, 1980) compared two methods for the administration of paradoxical intention. The straight-forward explanation (labeled type A administration) was compared to a procedure explaining the need of paradoxical intention in a manner best suited to the understanding of the client (type B administration). Thus type B administration attempted to deceive the client in performing the paradoxical behavior. These two groups were compared to a placebo control and a no-treatment control group. Results showed

that type A administration was superior to type B and that the type B administration was not significantly different than either of the two control groups. These results support a straightforward explanation of paradoxical intention without the use of deception.

Relinger and Bornstein (1979) used paradoxical intention in treating four severe, chronic sleep-onset insomniacs. They each received a five-session treatment program. Results indicated significant improvement on five of eight daily sleep chart measures. These gains were still apparent at a 12 week follow-up.

More recently, Fogle and Dyal (1983) compared paradoxical intention (a method they relabeled "try giving up" (TGU)) to a variation of paradoxical intention where insomniac subjects were instructed to "give up trying" (GUT) falling asleep. These two groups were compared to a placebo-attention (self-monitoring) treatment group. After a 3 week treatment period, it was found that all 3 groups improved in daily sleep estimates ("sleep efficiency") but only the two giving-up groups improved on a sleep-report measure of sleep performance anxiety.

Finally, Espie and Lindsay (1985) attempted to assess the efficacy of paradoxical intention by treating a small sample of 6 chronic insomniacs. After treatment, which lasted roughly 8 weeks, "Considerable variability in response to therapy was observed, with 3 patients obtaining a rapid reduction in sleep onset latency while the sleep pattern of the 3 other S's was significantly exacerbated" (Espie and Lindsay, 1985, pp. 703). The authors add that one of the three resistant patients subsequently improved following additional sessions. As for the remaining two, follow-up treatment using relaxation training yielded successful results.

Reports on the success of paradoxical intention are mixed. Some researchers claim improvement rates averaging up to 70% (Relinger and Bornstein, 1979; Relinger, Bornstein, and Mungas, 1978). On the other hand, Others are more conservative in their ratings, reporting amelioration rates of 58% (Borkovec, 1982), 46% (Lichstein and Fisher, 1985), and even rates as low as 18% (Lacks et al., 1983). Espie et al. (1989), demonstrated that, out of 15 patients

treated with paradoxical intention, 5 experienced an increase in sleep-onset latency (SOL) of at least 33% during the first week of treatment. They concluded with this statement: "Paradox may offer some improvement in both spheres (qualitative and quantitative improvements), however, it appears to have the unique capacity of producing, albeit temporary, exacerbation of sleep problems" (Espie et al., 1989, pp. 87).

Individual differences in interpreting the set of instructions given at the beginning of treatment may be responsible for treatment response variability across studies. Until this mechanism is better understood, treatment efficacy will remain difficult to predict.

Sleep Restriction Therapy

One very promising procedure called sleep restriction therapy (Spielman et al., 1983, 1987) has been only recently added to the already extensive behavioral arsenal just reviewed. The reasoning behind this treatment approach stems from the fact that most insomniacs spend a lot of time in their bed at night without actually sleeping. This in turn, makes for very poor sleep efficiency (estimated total sleep time/time in bed x 100%). The treatment attempts to forcefully reduce the TIB (total time in bed) to a certain amount of hours per night, until the sleep efficiency increases to above 90%. Once this new sleep efficiency is maintained for a certain number of days (5 in all), the TIB is increased 15 min. and the procedure starts all over again. "For example, if a patient reported a nightly average of 5h of sleep for a 2-week baseline period, although having spent an average of 8h in bed, then the nightly TIB prescribed at the start of treatment was 5h" (Spielman et al., 1987, p.47).

In their most recent study, Spielman et al., (1987) treated 35 patients with a mean history of insomnia of 15.4 years. Restriction of time available for sleep was first set for each

individual in a similar fashion as the example cited above. Then, an extension of time in bed was made contingent upon improvement in their respective sleep efficiencies. After 8 weeks of treatment, there was a significant overall reported increase in total sleep time, combined with improvement in sleep latency, total wake time, sleep efficiency and subjective assessment of their insomnia. Of the 23 subjects contacted 36 weeks after treatment, most reported a maintenance of the improvement first noted during the treatment phase.

Theorizing as to the effectiveness of such an approach, the authors remarked: "Most current theories of insomnia emphasize factors that precipitate insomnia and characteristics that predispose individuals to develop a sleep disturbance. In contrast, the present approach is guided by the idea that addressing the factors that perpetuate chronic insomnia is essential for therapeutic success. Sleep restriction therapy assumes that excessive TIB is one important factor that sustains insomnia although it may not have initiated the sleep disturbance" (Spielman et al., 1987, p. 53). They add that the crucial ingredient necessary for the effectiveness of their approach might be the initial mild sleep deprivation imposed by the treatment. According to the same authors, this similar mild sleep loss would also account for the apparent success of the stimulus control procedure.

Finally, the authors point out research supporting the notion of sleep fragmentation and not sleep loss as the possible source of complaints behind insomnia (Carkabon et al., 1976, Rosenthal et al., 1984, Bonnet, 1985). "Therefore, the clinical efficacy of the current approach may be more related to the reduction of nocturnal wakefulness, which was considerable, than the increase in total sleep time, which was modest" (Spielman et al., 1987).

In sum, Spielman and his colleagues report a success rate of 60% with polysomnographic measures to substantiate their results. This promising approach obviously needs to be further assessed with special emphasis placed on reducing the drop-out rate, which appears to be due to the technique's apparent "harshness".

Sleep hygiene

Although not defined as a technique per se, the practice of sleep hygiene is still considered by some as a healthy and therapeutic approach towards controlling sleep-onset insomnia. In general, sleep hygiene includes the assessment/control of sleep scheduling, presleep activities, attitudes about sleep, daytime behavior and the sleep environment (Nau and Walsh, 1983). Other authors are willing to extend this list even further, adding additional factors such as noise, light, temperature, hunger, naps, sleep scheduling, sleep medication, attitude, worry and bedpartner (Hauri, 1982).

Lacks and Rotert (1986), in an attempt to understand the role of sleep hygiene in the maintenance of certain forms of insomnias, surveyed 44 sleep-onset insomniacs, 49 sleep-maintenance insomniacs and 50 good sleepers. Using an instrument developed especially for their research, they showed that insomniacs were more knowledgeable about sleep hygiene than good sleepers, but practiced it less often. Their results also suggested "that poor sleep hygiene is not a primary cause of insomnia; however, behavior therapists should continue to include this element in treatment to help insomniacs avoid exacerbation cycles" (Lacks and Rotert, 1986, pp. 365).

Finally, most therapeutic interventions, from biofeedback and stimulus control to relaxation and hypnosis, contain inherent procedures demanding some alterations in sleep hygiene. As an example, for some individuals, practicing relaxation at bed time, in addition to controlling muscle tension, can also affect other components such as attitudes, worry, presleep activities, sleep scheduling, and bedpartner, to name just a few. If prior to or during treatment certain crucial sleep hygiene components are absent, then they should be added to the overall treatment package offered.

CONCLUSION

Review of success rates

In reviewing the various behavioral approaches to the treatment of insomnia, it is possible to sum up our findings as follows: 1) relaxation therapy, and more specifically, progressive relaxation, repeatedly rates at about a 50% level of success without much variation across studies; 2) most of the EEG studies reported attempted to assess, or at least used as a comparison treatment, progressive relaxation; 3) stimulus control also produces elevated success ratings but its overdependence on subjective sleep measures casts some doubt on its otherwise promising usefulness; 4) a summary of the extensive literature on paradoxical intention reveals profound discrepancies in success rates across studies. The cause of this remains to be investigated; 5) sleep restriction therapy is a sound and promising technique but its novelty is, at the present time, its greatest hindrance; and finally, 6) systematic desensitization, as well as biofeedback, offer at best only moderate success rates thus making their usefulness in a clinical setting questionable.

Objective versus subjective measures

Given the repeated criticisms based on the lack of objective outcome measures, a recent trend in sleep research has developed which attempted to combine physiological measures and self-report to assure an accurate measurement of sleep-onset latency (Coursey and Frankel, 1977; Coursey et al.; 1980; Freedman and Sattler, 1982). Montgomery, Perkin and Wise (1975), after a thorough review of the already existing research concerning behavioral treatment for insomnia,

came to the conclusion that "both subjective and objective sleep measures in future studies would provide more information about the management of insomnia" (p. 97). This same conclusion was later stressed by Knapp, Downs and Alpers (1976) and Ribordy and Denney (1977). Given that most subjects tend to chronically overestimate their sleep-onset latencies (Freedman and Papsdorf, 1976), it is of the utmost importance to objectively monitor sleep-onset latencies (SOL) on a regular basis.

However, polysomnographic recording in the laboratory, the most reliable means of accurately recording SOL, is cumbersome, intrusive, and not practical in ordinary clinical settings. For this reason, many investigators have relied on self-reports as an alternative in monitoring SOL. In an attempt to resolve the problem of the unreliability of self-reports for the measurement of SOL in the home environment, a special switch-activated clock was introduced by Franklin (1981). This device is essentially a clock, activated at bedtime by pressing a thumb button, which ceases to record time when the button is released (presumably when the subject falls asleep). We have pointed out that "While going a long way in providing a simple and objective measure of SOL in the home environment, Franklin's device presents the following limitations. Firstly, the procedure requires that a partner set the clock to a predetermined time to avoid any data contamination arising from the S's own awareness of the clock's preset time. Secondly, the clock stops with any deactivation (thumb release) of the switch and thus is subject to premature SOL recordings in cases of accidental or brief releases" (Viens et al., 1988, pp. 271).

In order to avoid these pitfalls, we have developed an improved version of Franklin's clock. The SOL monitor consists of a time-based counter and display module. The subject is required to press the button of a hand-held switch with the thumb. The initial pressure on the button causes the digital display to go blank. This does not allow any type of visual feedback to the subject. If the button is released then repressed within a preset interval (5 min. in this study),

this will be ignored by the device. However, a depression of the button which exceeds the preset interval stops the counter, reactivates the display, and shows the SOL time in a coded format. "SOL is therefore measured from the moment the button is pressed to the time when the button has been released for a period exceeding 5 min. In the morning, the S simply writes down the coded number on a special form" (Viens et al., 1988, pp. 271).

Preliminary results suggest that the SOL device is a reliable measure of sleep-onset latency. The monitor was validated against electrophysiological measures of sleep-onset latency with 4 female students (3 normal sleepers and 1 insomniac). For the four subjects, all correlations between latency to stage 2 and the monitor's SOL were significant. With the exception of the insomniac subject, no significant correlations were found between latency to stage 1 and the monitor's readings.

These preliminary results warranted the use of the SOL monitor as an effective apparatus in measuring sleep-onset latencies where they are most important, in the home environment.

Do we need cognitions?

To sum up this section on various behavioral treatments of insomnia, Borkovec (1982), in one of the most extensive reviews in the area concluded: "If we limit our discussion to sleep-onset insomnia of the psychophysiological and subjective subtypes, which we must do given the extant literature (.....), then some meaningful (although not definitive) statements are possible. Such insomniacs fail to achieve, either objectively or subjectively, sufficient sleep to feel refreshed during the day. They show highly variable sleep, overestimate sleep latency, tend to present a psychological picture of mild depression, anxiety, and worry, report excessive and often negative cognitive intrusions prior to falling asleep, and will often report being awake when

aroused from Stage 2 sleep. Sometimes, they have been found to be physiologically hyperaroused, and suggestive evidence indicates the possibility of dampened average-evoked-response potentials and, among psychophysiological insomniacs, deficient sensorimotor rhythm. The subjective sleep disturbance of sleep-onset insomniacs can often be successfully reduced by unspecified but clearly active ingredients contained in the behavioral treatments of relaxation, biofeedback, paradoxical intention, and stimulus control; preliminary EEG evidence indicates that objective improvement also occurs under relaxation and biofeedback regimes. It is quite likely that these techniques are effective for both psychophysiological and subjective insomnias, with laboratory support for this conclusion in the case of progressive relaxation." (Borkovec, 1982 p. 891).

This last statement accurately reflects our present behavioral/physiological knowledge of sleep-onset insomnia. Nevertheless, some would argue that an important component is missing, the discovery of which would help unravel some of the inconsistencies existing in the present literature. In fact, many have turned to cognitive theories in the hope of shedding some light on their continuing dilemma. Authors such as Lacks(1987), Cook and Lacks (1986), and Schoicket et al. (1987), have stressed the importance of the role of cognitions in the maintenance of insomnia.

Our present review of sleep-onset insomnia and behavioral techniques leads us now to draw attention to the body of theoretical research on the role of cognitions in causing and maintaining insomnia. Given the significant contribution of cognitive psychology to research on sleep disorders, it is only fitting here to briefly review some of their main findings.

4. COGNITIONS AND INSOMNIA

An overview of the studies investigating the possible relationship between cognitions and insomnia may be viewed from two different angles. On the one hand, many investigations have probed the theory of attribution (based on Schachter and Singer's (1962) classic study) and how it could, in turn, help us better understand its role in relation to insomnia. On the other hand, others have preferred to investigate the possible connection between cognitive arousal, a complaint often reported by insomniacs, and insomnia.

Insomnia and the attribution process

The role of cognitions in affecting emotions and behavior has always been the main investigation pillar supporting cognitive psychology. The landmark experiment on emotions published by Schachter and Singer in 1962 opened what was believed to be a whole new area in psychology.

In their experiment, Schachter and Singer (1962) exposed subjects to situations designed to elicit an emotional response, either anger or euphoria. Before this exposure took place, however, subjects were injected with adrenaline, a drug producing autonomic arousal. While certain individuals were informed of the effects the drug would produce on them, other subjects received no information at all. It was later found that these latter subjects became far more emotional, either euphoric or angry, depending on the experimental condition, than their well informed counterparts. Thus it appeared that not only "what" one felt but "how" one interpreted the situation (with cognitions attending the arousal) were important factors in producing the final emotional response.

Storms and Nisbett (1970) carried their interpretation of these results even further: "A perhaps equally important implication of the experiment has received little attention. Not only were informed subjects less emotional than uninformed subjects, they were also less emotional than control subjects who received a placebo. This trend, though statistically not significant, suggests that informed subjects overcompensated for the injection. They perhaps attributed not only adrenaline-producing arousal to the injection, but naturally occurring arousal as well. As a consequence, informed subjects were less emotional than they "should" have been, given the emotion-eliciting situation in which they were placed." (Storms and Nisbett, 1970, p.319).

This interpretation is a direct application of Kelley's attribution theory (1967). In this theory, Kelley proposed that our perception of causes for inner psychological effects results in many cognitive and motivational phenomena. However, in this operation called causal attribution, one can make errors, that is, attribute an effect to the wrong cause, thus changing motives and beliefs in the process. "Thus, the subjects in Schachter and Singer's (1962) experiment may be viewed as victims of an experimentally produced attribution error. Uninformed subjects in that experiment who were injected with adrenalin mistakenly attributed their arousal to the situation in which they found themselves, rather than to the injection. As a consequence, they became emotional" (Storms and Nisbett, 1970, p.320).

Storms and Nisbett (1970) believed that the manipulation of the perceived source of autonomic arousal may become a therapeutic procedure in itself. They decided to test this hypothesis on a sample of sleep-onset insomniacs. Forty-two subjects were assigned to either of two conditions, the arousal condition or the relaxation condition. In the arousal condition, subjects were given placebo pills and were told that these would "increase their bodily activity". In the relaxation condition, the exact same pills were used, but here subjects were told that the drug would "lower their bodily activity". Results showed that subjects in the arousal condition fell asleep more quickly than they had before the experimental manipulation. Similarly, subjects

in the relaxation condition took more time to get to sleep after taking the pills than before.

According to the authors, the "arousal" subjects attributed their autonomic arousal to their pills rather than to their emotions, thus making them less emotional. As for the relaxation group, being still aroused after taking their pills led them to believe that they must have been "very" aroused thus further heightening the intensity of their emotions. Supported by these results, the authors suggested the feasibility of a new therapy based on reattribution of symptoms.

Unfortunately, since then efforts to replicate Storms and Nisbett's results have been futile. Kellogg and Baron (1975), in an attempt to understand the "reverse placebo" effect, replicated portions of the Storms and Nisbett (1970) experiment (i.e. number of subjects and their recruitment procedures) in addition to adding other experimental manipulations. Half the subjects were given placebo pills with accompanying arousal side effects instructions. Whereas the other half received no pills. In each of these groups, half the subjects were given a high justification rationale for the study, while the other half was given none. So of these four conditions, only one resembled Storms and Nisbett's arousal group, that is, the pill / no justification group. Kellogg and Baron found that this last group, in addition to not improving in their reported sleep-onset, reacted like any placebo group would, that is, their condition in fact worsened under the belief that they were affected by the placebo drug.

Bootzin, Herman and Nicassio (1976) attempted to replicate Storms and Nisbett's (1970) experiment while at the same time delimiting its effects. In order to do this, they used forty-eight subjects which were assigned to one of 5 different groups. Two of these groups were subjected to the exact experimental conditions employed by Storms and Nisbett (1970), that is, arousal and relaxation manipulations. Two additional groups also took the placebo pills but each received a differing set of instructions as to the effects of the pills on their sleep (pill will help you sleep / pill will keep you awake). As the authors themselves explained: "The purpose

of these conditions was to determine the effect of direct sleep-related suggestions accompanying the placebo on the reporting of sleeping difficulty on the experimental nights." (Bootzin, Herman and Nicassio, 1976, p. 676). Finally, the fifth group (a control group) was added in order to measure differences in sleep-onset reports which could not be attributed to any experimental manipulations.

The results were inconsistent with Storms and Nisbett's (1970) predictions. The arousal group was in no way affected by the experimental manipulation. Interestingly, a straight placebo effect was reported for the relaxation group.

More recently, Heffler and Lisman's (1978) attempt to replicate the original placebo effect experiment first described by Storms and Nisbett (1970) again ended in failure. Even with added "improvements" to the original design, they were unsuccessful in replicating their original results.

In sum, it appears that Storms and Nisbett's (1970) original findings opened up a new avenue that ended up to be a dead end street. Heffler and Lisman (1978) summarized it well in writing : "These successive (replication) failures would suggest that the growing excitement about therapeutic application of attribution theory should be tempered with caution. We would agree with Storms and Nisbett's expressed concern that, despite the outcome of their own work, pills and deception may not be the most reliable techniques for achieving reattribution." (Heffler and Lisman, 1978, p.128).

As this series of unsuccessful attempts to replicate Storms and Nisbett's (1970) results was coming to a grinding halt, other investigators started following a different avenue in an attempt to understand the relationship between cognitions and insomnia. The theory of attribution was simply brushed aside, replaced by the study of what is commonly referred to as "racing thoughts", "worries", and "mental tension". In other words, cognitive arousal became the new focus of investigators' attention.

Somatic versus Cognitive arousal

Ever since Monroe's (1967) and Freedman and Sattler's (1982) findings indicating that "poor" sleepers showed heightened sympathetic arousal when compared to "good" sleepers, therapeutic endeavors have to a large extent focused on ways of reducing the physical tension associated with poor sleep. This was made possible by training clients either to relax their muscles before going to bed or to focus their attention on relaxation. However, as Haynes, Follingstad and McGowan (1974) have pointed out, successful reduction of physical tension was not necessarily correlated with a return to a "normal" latency to sleep onset. This observation prompted Borkovec et al. (1975) to suggest "that in addition to physical and mental tension, pre-sleep intrusive cognitions such as "racing thoughts", worry and specific anxieties may also contribute to insomnia as measured by latency to sleep onset" (Mitchell and White, 1977, p.57).

Mitchell and White (1977) set out to clarify the role of pre-sleep intrusive cognitions in the maintenance of sleep-onset insomnia. They used 13 subjects assigned to one of 3 group conditions, i.e., 1- progressive relaxation (5 weeks), mental relaxation (5 weeks) and cognitive training (5 weeks); 2- combined muscle and mental relaxation (10 weeks) and cognitive training (5 weeks); or 3- self-monitoring (10 weeks) and cognitive training (5 weeks). Their findings indicated that muscle relaxation reduced the pre-sleep tension targeted but had no effect on pre-sleep intrusive cognitions. On the other hand, when combined with mental relaxation, not only was pre-sleep tension was further reduced, but intrusive cognitions were reduced as well. Cognitive training alone was successful, as was mental relaxation, in decreasing pre-sleep tension and intrusive cognitions.

As a follow-up, Mitchell (1979) compared the efficacy of three treatment techniques focusing either on: 1- presleep tension alone; 2- presleep tension and intrusive cognitions; or, 3-

perception of sleep. The author found that reducing presleep tension and intrusive cognitions led to greater reductions in both latency to sleep-onset and daytime impairment. Reduction of "presleep" tension alone had similar effects but not as pronounced. Interestingly, giving information about sleep and usual sleep habits (which the author labelled "perception of sleep") improved subject's condition as much as reducing "presleep" tension alone. Mitchell (1979) concluded : "The results of the present study and the findings of Mitchell and White (1977) support this conclusion (the importance of cognitive components) because they clearly suggest that the control of covert cognitive stimuli such as "racing thoughts" and worries is particularly relevant as a target in predormital insomnia" (Mitchell, 1979, p. 68). It is important to add at this point that Mitchell and White's (1977) and Mitchell's (1979) experiments relied solely on their subject's subjective estimates of sleep-onset. No objective measures were reported. As will be explained later, this in itself, is a serious methodological drawback and any conclusions from these studies should be interpreted with caution.

At about the same time that Mitchell (1979) was demonstrating the importance of intrusive cognitions in the etiology of sleep-onset insomnia, Lichstein and Rosenthal (1980) arrived at similar conclusions using a somewhat different perspective. Surveying 296 insomniacs (209 women and 87 men), the authors attempted to clarify the subjective influence of somatic versus cognitive arousal on the reported sleep disorder. As their results indicated, cognitive factors were cited by most insomniacs surveyed as being the primary cause of the insomnia (and this more often than somatic ones). When both factors were cited, more importance was assigned to cognitive factors than to somatic ones. The authors suggested these "modalities" (cognitive versus somatic) should be taken into consideration in determining an effective treatment plan.

At this point, the next logical step was to assess the actual (measurable) impact of cognitive stress on sleep-onset insomnia. To do this, Haynes et al. (1981) exposed 10 sleep-

onset insomniacs and 11 non-insomniacs to brief cognitive stressors before sleep-onset for the last two nights of five consecutive nights in the sleep laboratory. The results were somewhat unexpected. On stress nights, while non-insomniacs increased their latency to sleep-onset as would normally be predicted, insomniacs actually decreased in sleep-onset latency. In explaining these results, the authors postulated : "If ruminative cognitive activity, sleep-related thoughts, or attributions of internal causality for sleeping difficulties serve etiological functions in sleep-onset insomnia, disruption of those cognitive events will result in reduced sleep-onset latencies" (Haynes et al., 1981, p. 604). But as it was argued earlier in this discussion, reattribution therapy has repeatedly met with replication failure (Kellogg and Baron, 1975, Bootzin et al., 1976, Heffler and Lisman, 1978). Haynes et al. (1981) concluded by adding that "additional research on the comparative effects of cognitive and noncognitive stressful and nonstressful presleep stimuli will help identify mediating factors and mechanisms"(p. 604).

Van Egeren et al. (1983) conducted a study examining the role of cognitive factors (attributions about the causes of sleep difficulties and presleep cognitive activity) in the maintenance of sleep-onset insomnia. Thirty-four sleep-onset insomniacs slept for 5 consecutive nights in the sleep laboratory. Presleep cognitions, as well as three different sleep-onset latency measures (laboratory-measured subjective sleep-onset latency, laboratory-measured objective sleep-onset latency and interview-measured subjective sleep-onset latency), were monitored. The authors reported a significant positive correlation between attribution of sleep-onset difficulties and interview-reported subjective sleep-onset latency. In addition, and of relevance to this discussion, "cognitive variables were not significantly associated with the objective measure of sleep-onset latency" (Van Egeren et al., 1983, p. 229). In other words, the more an insomniac reports presleep cognitions, the greater the probability he/she will overestimate his/her latency to sleep-onset, but this does not significantly affect objective (physiological) measures of sleep-onset latency.

In concluding this section, it is safe to state that at the present time the role and the effects of cognitions on sleep-onset insomnia remain unclear. Nevertheless, there appears to be some empirical support for the following statements : 1- mental or cognitive relaxation apparently increases the efficacy of progressive relaxation (Mitchell and White, 1977; Mitchell, 1979); 2- most insomniacs tend to believe that their condition is precipitated by cognitive rather than somatic arousal (Lichstein and Rosenthal, 1980); 3- when exposed to brief cognitive stressors, insomniacs actually decreased their sleep-onset latency as measured in a sleep laboratory (Haynes et al., 1981); and, 4- there appears to be no association between cognitive variables and objective measures of sleep-onset latency (Van Egeren et al., 1983). These conclusions reinforce the notion that cognitive factors are poor predictors in the assessment and treatment of sleep-onset insomnia as measured objectively in a sleep laboratory. For this reason, other etiological factors have to be contemplated.

Somatic arousal + Cognitive arousal = Anxiety

As the above review indicates, it appears hazardous to attempt to separate arousal into somatic and cognitive subclasses. An interesting observation can be made if we parallel somatic/cognitive arousal with the operant conditioning definitions of overt and covert behaviors. Somatic arousals are, in general, behavior changes which are overt in nature. Most of the time they are observable, measurable and often lend themselves to empirical manipulation. On the other hand, cognitive arousals can also be interpreted as covert behaviors. This interpretation "arises from the fact that the behavior may actually occur but on such a reduced scale that it cannot be observed by others at least without instrumentation. This is often expressed by saying that the behavior is 'covert'" (Skinner, 1953, pp. 263). This definition helps us understand the apparent contradictions across studies stemming from the cognitive field. Covert

behaviors (known as cognitions by some) are not well understood. They seldomly lend themselves to standard measurement procedures and thus cannot be scientifically investigated at this time. Yet, some studies are reported in which cognitions are not only identified and isolated, but more surprisingly measured by often obscure processes. This is the basic flaw that has contaminated many of the studies in the cognitive literature which has resulted in an endless series of contradictory outcomes.

Measurement of covert behaviors will eventually be possible, but for now it is a premature and often fruitless endeavour simply due to our still neonatal measurement procedures. Fortunately, there is a solution to this problem. Somatic arousals (overt behaviors which to a certain extent can be measured objectively) and cognitive arousals (which lend themselves to more introspective types of investigations) are phenomena used to describe what is better known as anxiety. Many definitions of anxiety exist. To avoid any confusion, anxiety in this discussion will be behaviorally defined as an agglomerate of behaviors following an aversive stimulus or many aversive stimuli. This steers readers away from the suggestion that anxiety may be an "entity" functioning independently from the environment. "Anxiety, as a special case of emotion, should be interpreted with the usual caution. When we speak of the effects of anxiety, we imply that the state itself is a cause, but so far as we are concerned here, the term merely classifies behavior. It indicates a set of emotional predispositions attributed to a special kind of circumstance. Any therapeutic attempt to reduce the 'effects of anxiety' must operate upon these circumstances, not upon any intervening state. The middle term is of no functional significance, either in a theoretical analysis or in the practical control of behavior" (Skinner, 1953, pp. 180-181).

This appears to be the common thread joining all of the behavioral techniques reviewed earlier in this paper. As a consequence of using any one of them, there is apparently a change in overt and possibly in covert behaviors. This change is labelled by many as a reduction in

anxiety. To investigate this possibility more extensively, let us now review some of the important studies which have investigated the connection between anxiety and insomnia.

5. THE ROLE OF ANXIETY IN THE MAINTENANCE OF SLEEP-ONSET INSOMNIA

Anxiety and Insomnia

In researching this topic, the question most often asked is: "Does an increased amount of anxiety-related behaviors lead to an inability to fall asleep?" This is obviously what Haynes, Follingsted and McGowan (1974) had in mind when they wrote "the use of these behavioral techniques in the treatment of insomnia is based on the premise that anxiety or heightened autonomic arousal is an etiological or maintained factor. If heightened physiological arousal or anxiety inhibits sleeping, reduction of anxiety or autonomic activity levels would facilitate sleep" (p. 69-70). In fact this claim was partially supported several years ago by Monroe (1967) when he conducted his study comparing poor and good sleepers and concluded that the former group showed signs of elevated physiological arousal during sleep. This was indicated by their increased heart rate, respiration and electrodermal activity.

It would seem logical to find many other such studies linking insomnia and anxiety in the literature, but in fact, very little research in the area has been conducted. "The physician's common sense justified the routine assumption that his anxious patients sleep poorly. A nervous, tense, and apprehensive patient scarcely seems a likely candidate for a normal night's sleep, and our hypothetical clinician assumes that this notion is adequately supported by laboratory sleep research. Unfortunately, a perusal of the pertinent sleep studies yields a conclusion rather more tentative than that which guides clinical practice" (Williams et al. , 1979, p. 211). Nevertheless, indirect information gathered from various sources points to the fact that there clearly seems to be a viable connection between insomnia and anxiety.

Cohen and White (1950) as cited by Williams et al. (1979), have reported that 53% of

patients diagnosed as anxiety neurotics also reported insomnia as one of their symptoms. This was compared to 4% in a normal control population. As mentioned earlier, Monroe (1967), and a later study by Freedman and Sattler (1982), supported this by showing that poor sleepers (many of them insomniacs) also exhibited increased physiological activity. It was postulated by the same authors that this increase must have been the result of some underlying anxiety. In other words, the personality of poor sleepers makes them more likely to internalize rather than outwardly express their own emotions. This internalization of emotions results in physiological activation and subsequent insomnia. Goodenough et al. (1975) have shown that subjects viewing a stressful film obtained higher test scores on measures such as anxiety, hostility and depression among other things, than a control group viewing a neutral film. In addition, the subjects who viewed the stressful film subsequently took more time to fall asleep than the time taken by the control group. This again would support the notion that some form of increased anxiety could lead to sleep-onset insomnia.

More recently, Shealy, Lowe, and Ritzler (1980) confirmed that sleep-onset insomniacs scored somewhat higher on scales indicating neuroticism. Freedman and Sattler (1982), in addition to replicating these findings, added that insomniacs also obtained high scores on scales indicating anxiety and worry (derived from the Hysteria, Psychasthenia, and Schizophrenia scales).

Healey et al. (1981) demonstrated that patients who reported suffering from insomnia also reported that they were at a so-called peak in their own life-stress events. Kales, Kales and Soldatos (1982), referring to the same study, wrote : "A summary of these data suggests that chronic insomnia usually develops at a time when life-stress factors are prevalent and in those individuals who are predisposed by existing mental health problems to have less-than-adequate coping mechanisms" (Healey, Kales and Monroe, 1981, as cited by Kales, Kales, and Soldatos, 1982).

Finally, from an experimental point of view, a host of laboratory studies have shown that subjects viewing a stressful film or placed in a stressful presleep situation obtained higher test scores on measures of anxiety, hostility, depression, etc. than controls and subsequently took more time to fall asleep and had disrupted sleep patterns (De Koninck and Koulack, 1975; Koulack, Prevost and De Koninck, 1985). These results again support the notion that some already present form of generalized increased anxiety may be triggered, thus precipitating the sleep-onset insomnia.

State versus Trait Anxiety

It is now necessary to further elaborate on the definition of anxiety presented earlier. A more thorough operational definition of anxiety is in order using Spielberger's differentiation between "state" and "trait" anxiety (Spielberger, 1966). State anxiety (A-State) refers to an individual's momentary or situational anxiety, varying over time and across different settings. This is closer to a transient behavioral experience. On the other hand, "Trait anxiety (A-Trait) refers to relatively stable individual differences in anxiety proneness, i.e., to differences among people in the disposition or tendency to perceive a wide range of situations as threatening and to respond to these situations with differential elevations in state anxiety. A-Trait dispositions are reactive and remain latent until activated by stress associated with a specific danger situation (...). Persons who are high in anxiety proneness are disposed to perceive greater danger in relationships with other people that involve threats to self-esteem and to respond to these threats with greater elevations in state anxiety (...) than do people low in anxiety proneness. High and low A-Trait persons do not appear to differ in their reactions to threats posed by physical dangers" (Spielberger, 1975, p.137).

As reported by Levitt (1980), the use of the Spielberger's State-Trait dichotomy has

been fairly well documented and researched. More recently, Endler and Edwards (1982) reviewed the previous literature on the topic and concluded that the State-Trait distinction is in fact well supported and constitutes a valid tool in assessing various levels of anxiety.

In light of this additional definition, it is obvious that most previous studies in the area of sleep-onset insomnia have neglected to specify what type of anxiety they were measuring and subsequently treating. In reviewing the literature on the various techniques used to alleviate sleep-onset insomnia, it is clear, given the temporal focus of the various therapies (to be practiced at sleep-onset), that the type of anxiety most focused on as the potential source of the sleep disorder is state anxiety. Thus the premise is that if situational anxiety surrounding the act of going to sleep and staying asleep can be decreased, time to sleep-onset would follow the same trend. Up to now, this approach has had a reasonable amount of success. Nevertheless, the present study stresses that in order to understand psychophysiological sleep-onset insomnia more clearly, Spielberger's contribution (State vs Trait anxiety) must not remain overlooked.

Trait Anxiety and Insomnia

We have only started to realize the full extent of the mechanisms underlying the sleep-onset insomnia problem. Even with our present understanding of the problem, there are many questions still left unanswered. For example, what if, as first suggested by De Koninck and Godbout (1985), not state but trait anxiety is the real cause of insomnia? Reynolds et al. (1984) hinted at this when they reported that their group of insomniacs was not significantly different than a group of patients with a history of generalized anxiety disorder, but quite different from a group of depressed patients.

Thus this new question opens up an area not yet explored in the field of sleep research. Using the current behavioral techniques at our disposal, are we simply teaching patients to

somewhat improve their sleep (decrease in state anxiety) without really getting at the root of their underlying problem (i.e. trait anxiety)? In other words, current behavioral techniques which have yielded encouraging results may be treating the problem only partially (possibly reducing state anxiety) without acknowledging the potentially true etiology of the disorder (i.e., trait anxiety). So the question is : "would the reduction of trait anxiety be superior in alleviating sleep-onset insomnia relative to the lowering of state anxiety alone?(which apparently the other therapies are doing to a certain extent)"

There is at least partial support from previously conducted studies for the notion that treatment of generalized anxiety would be more efficient than the treatment of situational anxiety. As reported earlier, Healey et al. (1981) were among the first to postulate that insomnia could be caused, not by situational, but rather by chronic anxiety. In fact, they have shown that sleep-onset insomniacs most often reported their condition when they were at a so called peak or maximum in their personal life-stress events. In another study, Hauri (1968) found no difference in the latencies of his normal subjects after relaxation or strenuous physical exercise, even though the latter group was "physiologically" aroused as indicated by the increased heart rate, respiration rate and rectal temperature. This would indicate that "short-term physiological arousal" because of its "short-term" effects may not be at the core of most sleep-onset insomnias. Therefore, a possible postulation for the lack of correlation between high physiological arousal and sleep-onset insomnia in this study is the absence of "trait" or "chronic" anxiety. In a more recent study on the effects of Transcendental Meditation (TM) on personality dimensions and dream content (Busby and De Koninck, 1980), a progressive relaxation group was used to control for the relaxation component of TM. After five weeks of relaxation training, there was a significant reduction in anxiety as measured by the 16 PF and a nearly significant decrease on the trait anxiety inventory for both groups, suggesting that relaxation alone was not sufficiently potent in reducing generalized anxiety.

Finally, Carr-Kaffashan and Woolfolk (1979) treated both severe and moderate sleep-onset insomniacs using relaxation training or a highly credible placebo. Even though subjects significantly decreased their self-reported state anxiety, relaxation only led to a trend towards reduction of trait anxiety.

An important question which remains to be answered at this point is: "Are there any behavioral techniques which are effective in reducing generalized anxiety?" In fact, studies by Suinn and Richardson (1971) and Jannoun et al. (1982) support the notion that the technique called "Anxiety Management Training" (AMT) which uses progressive relaxation plus additional cognitive rehearsals, does have an effect on generalized anxiety. This relatively new technique needs to be detailed because of its important contribution to this thesis.

According to Suinn, "Anxiety Management Training" (AMT) may be described as "a conditioning technique involving: 1) the use of instructions and cues to arouse anxiety responses; and, 2) the training of the client in developing competing responses, such as relaxation or competency" (Suinn, 1975). AMT was initially developed as an alternative to desensitization. In some respects, both techniques share many similarities. However, crucial variations allow AMT to be used for problems for which desensitization has been proven ineffective. One basic difference between these two techniques is that in AMT, there is no hierarchy of anxiety provoking stimuli and/or events. Instead, one clearly defined anxiety provoking situation is used. First, the subject is trained to relax. Then, he/she is instructed to evoke anxiety by concentrating on the selected situation. Finally, the client is instructed to relax again while imagining pleasant scenes and scenes of success. This procedure is repeated several times during a single session. The aim of the technique is to pair the anxious reactions with bodily relaxation and pleasant thoughts .

This technique has a distinct advantage over desensitization. It was discovered that it

could be used successfully with individuals suffering from what is commonly known as "free-floating" or "generalized" anxiety. "Desensitization cannot be used with such clients in as much as no anxiety hierarchies can be constructed. On the other hand, since AMT focuses on the anxiety responses and not the anxiety stimuli, it should prove to be a valuable treatment method for free-floating anxiety" (Suinn, 1975, p. 66). Subsequent research listed by Suinn (1977) has proven that AMT is not only effective with generalized anxiety but is also very useful in dealing with test anxiety, public speaking anxiety, trait and state anxiety, heart disease, and hypertension. It is the goal of this thesis to determine whether sleep-onset insomnia could be added to this already extensive list.

More recently, a self-help AMT program has been developed where standard audio-cassettes are used as instructional tools. Jannoun et al. (1982) have successfully used this variant in treating twenty-seven of their patients suffering from generalized anxiety states. Given the success of AMT in its audio-cassettes format, this technique was selected for this research in order to treat generalized anxiety conditions.

Insomnia and Vigilance

So far, we have focused on the causes of insomnia and the treatment approaches. However, one important aspect remaining to be explored concerns the consequences of insomnia. In fact, a necessary criterion for insomnia is impairment in daily functioning. One of the common complaints reported by insomniacs is that during the day they lack attention and efficiency. Surprisingly the study of differential vigilance levels in insomniacs is recent. Even though Monroe's (1967) classic experiment identified physiological differences between poor and good sleepers, many years went by before appropriate investigations in this area were finally conducted.

"Vigilance" is defined in the New Lexicon Webster's Dictionary as "watchfulness, a being on the alert..."(pp. 1097), which is not to be confused with "performance" which has to do with what is accomplished during a certain task. Certain performance tasks will demand high levels of vigilance (i.e., discrimination between different auditory tones). Other performance tasks will require very little vigilance (i.e., a simple reaction time test). An air traffic controller will have a much different on the job level of vigilance than an assembly line worker. Nevertheless, vigilance cannot be assessed without a performance output thus making the separation of the two components very difficult. In the studies reviewed below, this distinction is not always apparent.

Research combining vigilance and sleep (or more precisely, the lack of sleep) appears to fall into two distinct categories: artificial sleep deprivation and its effects on vigilance and how vigilance is affected by lack of sleep secondary to a specific sleep disorder such as insomnia.

Horne and Pettit (1983) reported that depriving 8 subjects of sleep by keeping them awake 60 consecutive hours led to a significant decrease in auditory vigilance. On the other hand, Webb and Levy (1984) arrived at contradictory results with their own sample. Even after two nights of sleep deprivation, no significant reduction in visual vigilance was detected. It appears that Wilkinson (1964, 1968) was right in stating that the nature of the task itself determines if vigilance is affected or not by some lack of sleep. Sander et al's. (1982) own conclusions might have been correct in that the effects of sleep deprivation are more closely related to the nature of the cognitive operations to be performed than vigilance per se.

Several studies have focused more specifically on insomnia and its effects on vigilance. This disorder, in its chronic phase, is considered by many as a form of sleep-deprivation. Mendelson et al. (1984), comparing ten insomniacs to an equal group of normal sleepers, found no significant difference on the Multiple Sleep Latency Test (MSLT) and on a test measuring auditory vigilance. That same year, Seidel et al. (1984) showed that on the MSLT and on a card

sorting task, 138 objective insomniacs (those whose insomnia had been electrophysiologically assessed) fared as well as 89 asymptomatic sleepers. "In fact, the authors report that 14% of the DIMS (Disorder of Initiating and Maintaining Sleep) group did not fall asleep on any of the MSLT naps and hypothesize that a subset of DIMS subjects may respond abnormally to sleep loss. That is, DIMS subjects do not show the characteristic decline in daytime sleep latency seen in normal subjects following sleep loss" (Stepanski et al., 1988, pp. 54-55).

The above mentioned results corroborated Surgarman et al.'s (1985) findings. In that study, insomniacs were compared to normal individuals and were found to be as vigilant as their counterparts on an auditory task. More recently, Stepanski et al. (1988) reported results that were somewhat unexpected. Comparing 70 clinic patients seeking evaluation for chronic insomnia to a group of 45 sleepers with no symptoms, they found that the insomniacs were significantly more alert than the control group, alertness being measured by the MSLT.

So it appears that at least on the MSLT, insomniacs are indeed as alert if not more so than normal sleepers. Unfortunately, there are many problems in using the MSLT with insomniacs. First, you are asking insomniacs (many of them with severe difficulties falling asleep) to try to fall asleep, under observation, during the day. Logically, their difficulties with sleep-onset will be reflected in the test. But does this mean that they are more alert? Or that they are still having great difficulties falling asleep during the test? Second, it appears quite unnatural to have to go to bed at 10.00, 12.00, 14.00, and 16.00h for periods of about 20 minutes when it sometimes take you longer, if you are an insomniac, to simply relax. Finally, it appears that for many insomniacs, awakening in the morning is even harder than getting through the day (Seidel et al., 1984, Schneider-Helmert, 1987). This has also been observed with the many insomniacs interviewed for this research. Thus, once their peak in alertness has been reached, most feel awake enough to go on with their day. This entails that insomniacs should be assessed when they first rise in the morning and not necessarily during the day. As the MSLT and other results

point out for insomniacs, they are as alert and vigilant, if not more so, than non-symptomatic sleepers during the day. (Mendelson et al., 1984, Seidel et al., 1984, Stepanski et al., 1988).

Thus in conclusion, using the MSLT, it has been shown that there are clear results pointing to similar levels of vigilance between insomniacs and normal sleepers. It was also suggested that early morning assessments would be more sensitive to possible variations in these measures than all day assessments such as the ones done with the MSLT. Finally, as suggested by Wilkinson (1968) and later supported by Herscovich and Broughton (1981), an unstimulating performance task, such as the proposed Four-Choice Reaction Time test, could be sensitive to the effects of sleep loss.

Hypotheses

The purpose of this study was to compare the effects of the reduction of generalized (trait) anxiety on sleep-onset insomnia as compared to the reduction of presleep tension alone (state anxiety). To do this, Anxiety Management Training (AMT) was used to reduce trait anxiety and was compared to progressive relaxation (PR). A crucial feature of this research is the use of direct behavioral and physiological measures at the expense of less reliable subjective methods, such as self-reports. From the review and rationale presented above, many workable hypotheses were formulated.

1) Anxiety Management Training will lead to a greater reduction in Trait anxiety than would Progressive Relaxation. In other words, while it was expected that PR would significantly reduce trait anxiety, AMT was expected to have a larger effect. Even though there were some indications that PR somewhat affects trait anxiety (Busby and De Koninck, 1980), the bulk of the research on this topic indicated that AMT would be superior in affecting trait anxiety (Suinn and Richardson, 1971; Jannoun et al., 1982).

2) **Both PR and AMT are expected to reduce sleep-onset latencies in the laboratory and the home environment.** As was shown earlier, studies which have assessed PR frequently report steady success rates of about 50% (Lacks et al., 1987; Ladouceur and Gros-Louis, 1984). However, as also explained earlier, reductions in SOL, through the reduction of trait anxiety via the use of AMT, should also be observed.

3) **Congruent with reductions in trait anxiety, AMT will lead to greater SOL reductions in the laboratory than PR.** This stems from the speculated connection between trait anxiety and sleep-onset insomnia and is consistent with hypothesis 1.

4) **AMT will also be more efficacious in reducing SOL in the home environment when compared to PR.**

5) **AMT will significantly reduce the negative psychological correlates of insomnia such as depression (as measured by the Beck Depression Inventory and certain MMPI scales i.e., Hs, D, Hy, and Pt).** This is of some importance since, notwithstanding the Montplaisir et al. (1986) study, no other studies have reported significant personality changes following therapy for sleep-onset insomnia, including PR.

6) **Finally, AMT will have greater positive effects on morning vigilance than PR.** The improvement in sleep and personality structure will hopefully translate into increased post-treatment performance, which in turn, will be expressed as better vigilance relative to pre-treatment.

CHAPTER 2 : THE STUDY

METHOD

Subjects

In the present study, it was important to screen out individuals whose insomnia was not strictly psychologically related. As a first step, a thorough sleep behavior questionnaire combined with an in-depth interview was used in an attempt to assess the specific cause of the sleep disorder. Past and present history of the problem, influencing factors, accompanying physical and psychological factors known to influence sleep, brief personal history and other areas were covered. This allowed a first screening out of many types of sleep-onset problems caused by physiological deficiencies (i.e., sleep apneas, periodic movements during sleep (PMS), and so on).

It was hoped to detect as well persons presenting signs of severe psychological and psychiatric disorders (psychotism and severe depression) as evidenced in the interview and/or through the MMPI (see Appendix B). Those exhibiting overuse of drugs and/or alcohol were not retained. Potential candidates using sleeping medication were required to withdraw from their medication for at least one month prior to the experiment.

To assess the degree of insomnia, an approach similar to the one suggested by Nicolas and Silvestri (1967) and subsequently by Shealy (1979) was used. According to these researchers, insomnia is considered at least moderate if there is a sleep-onset latency greater than 45 minutes at least four nights per week. To provide a preliminary assessment of sleep latency, a special switch-activated clock originally developed by Franklin (1981) and improved by this

author and his colleagues (Viens et al., 1986; see Appendix B) was used by the subjects at home for one week prior to final selection. Each daily sleep-onset latency (SOL) was recorded and returned to the experimenter for analysis. It was expected that subjects with moderate to severe insomnia would present not only sleep-onset delays but also frequent nocturnal awakenings. This, in turn, warranted the use of polysomnographic measures to assess the level of sleep efficiency at the different stages of the study.

Finally, to ensure that subjects were experiencing a relatively high level of trait anxiety, a score above the 60th percentile level on the trait dimension of the STAI (see Appendix B) was required.

The selection criteria can thus be summarized as followed:

Inclusion :

- Sleep-onset insomnia with a minimum of 45 minutes sleep-latency at least 4 times/week.
- Minimum percentile score of 60 on the trait dimension of the STAI.

Exclusion :

- Physiological causes of insomnia (i.e. sleep apneas, myoclonus, sleep schedule disorders).
- Severe psychopathology.
- Heavy use of alcohol and/or drugs, including hypnotics.

In all, over 200 individuals responded to our call for participants in the study. They were recruited in the Ottawa area using local newspapers, T.V. and radio advertisement, as well as public billboards. Of these, 47 fulfilled the preliminary requirements (as determined in a phone interview) and were subsequently invited for a personal assessment interview. Of these,

27 were selected as suitable candidates. All selected subjects were asked to complete the MMPI (Minnesota Multiphasic Personality Inventory) and the STAI (State-Trait Anxiety Inventory) as preliminary psychological baseline measures.

Shortly thereafter these subjects slept for one night in the laboratory for polysomnographic screening. Of these 27 individuals, 23 met the inclusion criteria. The other 4 were found to suffer from PMS (Periodic Movements during Sleep). Finally, 2 dropped out and one was later found to be using hypnotics. The twenty remaining subjects ranged in age between 19 and 63 years old. When asked during their personal interview: "how long does it take you to fall asleep?", the average response was 96 minutes. In addition, these insomniacs averaged around 5.5 hours in total sleeping time as assessed subjectively at home. Finally, when queried about the last time when they had a good night's sleep, eight respondents simply answered "do not remember", while the remaining subjects' estimates averaged 20 days.

Design and Procedure

A schematic representation of the research design is presented in Figure 1. Participants were assigned in a non-random fashion to one of two treatment groups (10 in each) in order to minimize a priori differences between the groups. We attempted to match the groups approximately for sex, age, level of insomnia and anxiety (see Table 1). This assignment was carried through in a blind fashion by an independent specialist using basic information (subject characteristics). Then they were brought for one night to the sleep laboratory for polysomnographic and psychological screening measures. The MMPI and the STAI were again completed to serve as comparative baseline measures.

As explained earlier, subjects presenting signs of sleep disorders which may be linked to insomnia-like symptoms were not maintained in the study protocol. Following this procedure,

FIGURE 1

EXPERIMENTAL DESIGN

GROUPS		SELECTION (1 night/lab)	"BASELINE" (21 days/ home)	PRE-TREATMENT (3 nights/lab)	TREATMENT (63 days/ home)	POST-TREATMENT (3 nights/lab)
AMT(@) (n=10)	Measures	polygraph SOL(#) STAI MMPI F-CHOICE	SOL	polygraph SOL	SOL	polygraph SOL STAI MMPI F-CHOICE
	Therapy					AMT
RP(*) (n=10)	Measures	polygraph SOL STAI MMPI F-CHOICE	SOL	polygraph SOL	SOL	polygraph SOL STAI MMPI F-CHOICE
	Therapy					RP

*RP=Progressive Relaxation @AMT= Anxiety Management Training #SOL= Sleep Onset Latency

TABLE 1

**NUMBER OF SUBJECTS, SEX & AGE RANGE
(AMT* VERSUS PR#)**

	AMT (n=10)	PR (n=10)
AGE RANGE:	19 58 (mean=35.7)	20 63 (mean=36.1)
SEX:	M=4 F=6	M=2 F=8

*AMT= Anxiety Management Training

#PR= Progressive Relaxation

subjects were told that a waiting period of approximately 3 weeks was necessary. During this period, they were instructed to keep a daily log of their sleep activities and their varying levels of sleep satisfaction using a short self-report questionnaire (see Appendix B) in addition to the SOL clock. After the three week period, subjects returned to the lab for psychological and polysomnographic baseline measures (3 consecutive nights). Here, one night of habituation and two nights of data collection were used. Following this, the two experimental groups received their respective treatment package (details presented below).

Throughout the baseline and treatment period, the subjects were seen once every two weeks to encourage compliance and collect the data. Finally, the two groups returned to the laboratory for final psychological and polysomnographic measures (3 nights).

Treatments

Two specially trained doctoral students (one male and one female, both in their late twenties and nearing the end of their Ph.D. program), acted as therapists in this study. They were trained in the administration of the two therapeutic approaches and were randomly assigned subjects to whom they applied the treatment. Thirteen (13) subjects (7 PR and 6 AMT) were treated by the male therapist while the remaining seven (7) subjects (3 PR, 4 AMT) were seen by the female therapist. The therapists had strict instructions not to overstep the boundaries prescribed by the technique itself. Thus their role was to simply reiterate the instructions contained in the original cassette used during the initial session.

Progressive Relaxation

The members of the first treatment group were individually taught PR using live instructions from one of two participating therapists. Then, for a period of about nine weeks, they were asked to use a tape recorded version of the first relaxation session which lasted roughly 30 minutes. This tape was to be practiced twice daily, once during the day and the second time while the subject was in bed and ready to fall asleep. During this home treatment period, subjects were asked to fill out and submit a special sleep questionnaire on a biweekly basis to allow monitoring of treatment effectiveness and compliance. In addition, the SOL clock was used at home to provide continuous daily measures.

Anxiety Management Training

The second treatment group was taught Anxiety Management Training. This procedure requires three basic steps : 1) A half hour training in deep muscle relaxation using tape-recorded instruction; 2) A one hour training session (with a therapist) in visualization of an anxiety-arousing scene, then visualization of a scene reinstalling competency or success response, and finally visualization of a scene associated with relaxation; and 3) A one hour take home tape-recorded version of the second step where anxiety is aroused, followed by either competency or relaxation. This tape was listened to at least once at the end of each day. As with the PR treatment, the AMT treatment lasted approximately nine weeks. The same measures of anxiety and SOL were used for both the PR group and the AMT group.

Finally, to avoid any bias in treatment implementation, a second therapist was employed. No therapeutic effect was observed which might have been due to differential treatment from the two therapists.

Measurements

1) Polysomnography : The subjects slept alone in a relatively soundproof room where temperature was maintained between 21 and 22 degrees celcius. Standard electroencephalographic (EEG) (C4/A1, C3/A1), electrooculographic (EOG) and electromyographic (EMG) activities were monitored as prescribed by Rechtschaffen and Kales (1968). In addition, respiration and lower limb muscle activity were monitored for the first baseline period to detect sleep apnea and PMS. To ensure comparability with previous studies, two measures of sleep onset latency (SOL) were calculated: I) 2 minutes of stage 1; and, II) 5 minutes of stage 2. In the case of female subjects, attention was given not to confound recording nights within the menstrual cycle. Recordings were scheduled during intermediate phases. The collected polysomnographic data was subsequently scored in an epoch by epoch fashion by this author and two independent judges. Interscorer reliability ratings between the author and each scorer was above 80% of epochs for every night compared. This reliability rating was calculated by randomly selecting three nights and subsequently comparing the sleep stages scored by the author, on an epoch by epoch basis, to the scoring of the other judges. If reliability for any night compared between the author and the two judges did not exceed 80% of total epochs, differences were discussed with the author. Afterwards the judge was required to rescore the night's recording until the desired reliability criteria was reached.

2) SOL monitor: Recently, an improved version of Franklin's (1981) sleep-onset latency (SOL) monitor was introduced the main purpose of which was to accurately measure sleep-onset in the home environment (Viens et al., 1986, 1988). The monitor, an electronic thumb controlled timing device, was validated in our laboratory against electrophysiological measures of SOL. Results showed a high correlation between onset of stage 2 (as measured polysomnographically) and the SOL monitor's final readings. It was concluded that the SOL monitor would be a very useful tool in this study, providing a reliable yet inexpensive measure of

SOL in the home environment.

In addition, a daily sleep satisfaction self-report questionnaire was included in order to compare variations in self ratings with possible variations in SOL.

3) Sleep positions: Since it has been observed in the laboratory that poor sleepers have a different postural preference during sleep (De Koninck et al., 1983), we used an infrared continuous video recording system to monitor the subjects' postural activity. It is also useful to detect abnormal body movements and PMS (Gagnon and De Koninck, 1985). The analysis of postural activity was not part of the present thesis.

4) Psychological measures

Personality : Given the extensive use of the MMPI in assessing and monitoring personality changes in insomniacs (Kales et al., 1976), this test was selected as one of the important instruments to detect not only severe pathologies commonly associated with insomnia but also any significant changes in personality induced by our treatment conditions.

Anxiety : It was paramount that generalized anxiety, in addition to other important psychological measures, be measured in this thesis. For this reason, the well-recognized State-Trait Anxiety Inventory (STAI) developed by Spielberger, Gorsuch, and Lushene (1970) was used. The Minnesota Multiphasic Personality Inventory scales of hysteria, hypochondriasis, depression, and psychastenia which have been shown to be sensitive in subjects suffering from sleep-onset insomnia (Coursey et al., 1975; Haynes et al., 1974; Johns et al., 1971; Kazarian, Howe, Mersky and Deinum, 1978), were used as secondary, but important, measures of anxiety and neurotism.

Physiological measures of anxiety have been considered. These include for example skin resistance and potential, heart rate, blood pressure, blood volume, respiratory rate, volume of saliva, gastric motility, pupillary diameter, muscular tension, EEG, and adrenal hormones. We agreed with Endler and Edwards (1982) who concluded that "problems encountered in the

interpretation of physiological measures and in relating them to each other and to reported levels of anxiety have limited their usefulness" (p.41) and thus have rejected that overall option.

Depression : The Beck Depression Inventory, in addition to the depression scale of the MMPI, was chosen as a quick and efficient instrument for measuring varying levels of depression in our subjects.

Vigilance : The Four-Choice Reaction Time test, which has been postulated to be directly related to varying levels of vigilance, was employed to allow us to measure possible variations in performance. Two decades ago, Wilkinson (1968) proposed that unstimulating performance tasks could be quite sensitive to the effects of sleep loss. This was later substantiated in a cumulative partial sleep deprivation experiment (Herscovitch and Broughton, 1981). One of the instruments used to measure vigilance was a portable Four-Choice Reaction Time (R.T.) test (Wilkinson and Houghton, 1975). The original form of the test used a magnetic tape recording device. Test information recorded on the tape must later be transferred to a computer for final data analysis. This whole procedure is in itself quite time consuming.

To overcome these drawbacks, a totally computerized version of the Four Choice R. T. test was developed in our laboratory. After selecting the stimulus size and the duration of the test, the subject was simply instructed to press one of four keys on the computer keyboard which corresponds in placement (one of four corners) to the stimulus displayed on the monitor. In our experiment this stimulus was a small square of $6,25 \text{ cm}^2$, that appeared in one of four corners of a $5.5\text{cm} \times 5.5\text{cm}$ square. When a response occurred (pressing any of the four designated keys), the square disappeared for .125 second and reappeared randomly in one of the square's four corners. The object of the test was to emit as many responses as possible without making any errors.

In addition to ease of administration, this computer version of the test allowed the researcher to immediately obtain a detailed descriptive analysis of the responses and errors that

were recorded during the trial run completed beforehand.

To ascertain the similarity in function between the original Four-Choice R. T. test and its computerized counterpart, nine individuals participated in a short experiment to make this comparison. All were subjected to a 10 minute trial on each of the two Four-Choice tests. While five subjects started with the original version first, the remaining four started with the computer version. The Pearson correlation comparing total number of responses on both tests was .73 ($df=7$, $p < .02$). A simple t-test showed that the performance on either test was comparable ($t=1.042$, $df=8$, $p < .31$). Thus it is apparent that the few modifications brought forth have not substantially altered the test's reliability and validity.

RESULTS

The Measurement of Sleep-Onset Latency

The principle dependent measure of this study was sleep-onset latency (SOL). It was thus important to determine the reliability between measures obtained with the SOL clock device and the polygraph.

Analyses comparing the SOL device's measures to those of the polygraph essentially gave results which corroborated those found in our pilot study. Notwithstanding the initial screening night, all subjects slept for a total of 6 nights in the laboratory (2 adaptation and 4 experimental nights). Data was therefore potentially available from 120 nights (20 subjects x 6 nights) of recording. In actuality, SOL measurements were compared on 103 nights. During initial recordings, the SOL clock device was not used for 17 nights.

Given the small number of nights for each subjects, the data was pooled and each night was treated independently. The Pearson correlation coefficient between latency to stage 2 (as measured with the polygraph) and the SOL monitor was $r = 0.72$ ($df=101$, $p < 0.001$). However a t-test comparing the stage 2 onset and the SOL device latencies revealed that the latter systematically overestimated SOL by about 8 minutes (means 34.8 min. and 42.8 min., respectively $t=2.97$, $p < 0.004$).

AMT versus PR

The means and standard errors of the mean (SEM) for the various personality and performance tests and the different sleep parameters for both groups are summarized in

Table 2 and 3. Two way repeated measures ANOVAS were performed on the data to compare the two therapy groups, and the efficacy of each one of them taken separately. For overall changes induced by treatments, within-group main effects were expected whereas the superiority of AMT over PR would be obtained through significant interactions and appropriate simple main effects.

Hypothesis 1 stated that AMT would lead to a greater reduction in trait anxiety than would PR. This was not confirmed. A within-group main effect, indicating a significant reduction in trait anxiety from pre-treatment to post-treatment, was observed ($F=14.45$, $df=18$, $p<0.001$), but no interaction was present ($F=0.32$, $p<0.57$). This indicated that both AMT and PR were equivalent in reducing trait anxiety.

Hypothesis 2 stated that a significant decrease in trait anxiety in both PR and AMT would lead to a significant decrease in SOL (at home and in the laboratory). In fact, this was shown to be true only for sleep-onset latencies measured at home ($F=11.07$, $p<0.004$).

Hypothesis 3 stated that AMT would lead to a greater reduction in SOL measured in the laboratory than PR. This was not the case. No significant decline in SOL was manifested in the laboratory ($F=1.71$, $p<0.20$).

As predicted by hypothesis 4, a significant within-group difference was observed ($F=11.07$, $p<0.004$) indicating that both groups improved in their sleep-onset latencies at home. On the other hand, the absence of an interaction ($F=0.29$, $p<0.59$) did not support the claim that AMT would be superior to PR in reducing home SOL.

Hypothesis 5, which predicted that AMT would significantly reduce the negative psychological correlates of insomnia, was confirmed. Within-group differences were present on such MMPI scales as Hs ($F=7.76$, $p<0.01$), D ($F=8.21$, $p<0.01$), Hy ($F=4.08$, $p<0.05$), and Pa ($F=11.92$, $p<0.003$) (see Figure 2 for the pooled MMPI scores of all subjects before and after treatment and Figure 3 for AMT's MMPI group profile) as well as the Beck Depression

TABLE 2

**MEANS & S.E.M.@ FOR THE PERSONALITY AND
PERFORMANCE TESTS (PR & AMT)**

	Group	Before		After		T.E.#	INTER.#	S.M.E.#
		Mean	S.E.M.	Mean	S.E.M.			
STAI	PR	88.2	3.11	70.0	5.90	* *	ns	* *
	AMT	89.8	3.01	65.2	10.56			*
Beck	PR	8.77	1.45	6.44	1.19	* *	ns	ns
	AMT	12.5	2.91	6.70	2.65			* *
MMPI:								
Hs	PR	62.7	3.86	59.0	4.17	* *	ns	ns
	AMT	66.6	3.54	57.5	3.02			*
D	PR	70.3	4.84	65.3	3.88	* *	ns	ns
	AMT	70.1	5.29	62.3	3.47			* *
Hy	PR	65.8	2.69	64.2	3.79	*	ns	ns
	AMT	67.9	2.15	61.6	2.84			*
Pd	PR	57.8	2.35	60.9	2.92	ns	ns	ns
	AMT	66.9	5.40	65.7	3.18			ns
Mf	PR	51.5	4.73	48.5	5.43	ns	ns	ns
	AMT	53.3	5.15	51.7	5.40			ns
Pa	PR	61.7	1.90	55.0	3.43	* *	ns	*
	AMT	66.8	2.94	63.1	2.96			*
Pt	PR	62.2	3.02	60.8	3.76	ns	ns	ns
	AMT	62.1	4.33	59.6	4.07			ns
Sc	PR	59.6	1.90	58.1	4.34	ns	ns	ns
	AMT	63.5	4.86	61.8	3.81			ns
Ma	PR	51.1	4.72	53.5	4.04	ns	ns	ns
	AMT	59.0	3.66	64.1	3.55			ns
Si	PR	52.7	2.61	51.4	2.53	ns	ns	ns
	AMT	52.7	4.42	51.2	3.14			ns
4-Ch.	PR	950	28.42	1016	23.73	* *	ns	* *
	AMT	962	35.26	1001	30.46			*

** : $p < 0.01$; * : $p < 0.05$; ns : $p > 0.05$

: T.E.= Within group(Treatment Main Effects); Inter.= Interaction; S.M.E.= Simple Main Effects

@= Standard Error of the Mean

TABLE 3

MEANS & S.E.M.@ FOR THE DIFFERENT SLEEP
PARAMETERS (PR & AMT)

	Group	Before		After		T.E.#	Inter.#	S.M.E.#
		Mean	S.E.M.	Mean	S.E.M.			
SOL Dev.	PR	68.25	13.71	42.85	10.80	**	ns	**
	AMT	81.06	8.26	45.72	13.58			*
SOL Poly.	PR	37.75	7.02	34.78	11.86	ns	ns	ns
	AMT	47.80	22.28	21.22	3.54			ns
% Sleep Stages								
1	PR	0.06	0.01	0.03	0.01	*	ns	ns
	AMT	0.13	0.05	0.04	0.01			ns
2	PR	0.52	0.02	0.52	0.04	ns	ns	ns
	AMT	0.56	0.04	0.57	0.03			ns
3	PR	0.09	0.01	0.10	0.02	ns	ns	ns
	AMT	0.08	0.01	0.08	0.02			ns
4	PR	0.08	0.02	0.11	0.02	ns	ns	ns
	AMT	0.02	0.01	0.06	0.02			ns
Delta	PR	0.18	0.02	0.22	0.03	*	ns	ns
	AMT	0.10	0.02	0.14	0.03			ns
REM	PR	0.23	0.01	0.21	0.02	ns	ns	ns
	AMT	0.18	0.02	0.23	0.02			*
Sleep Effic.	PR	0.94	0.02	0.94	0.01	ns	ns	ns
	AMT	0.89	0.06	0.91	0.03			ns
Sleep Satisf.	PR	2.32	0.21	2.10	0.22	**	ns	ns
	AMT	2.59	0.13	2.12	0.18			**

** : $p < 0.01$; * : $p < 0.05$; ns : $p > 0.05$

: T.E.= Within group (Treatment Main Effects); Inter.= Interaction; S.M.E.= Simple Main Effects

@ = Standard Error of the Mean

FIGURE 2

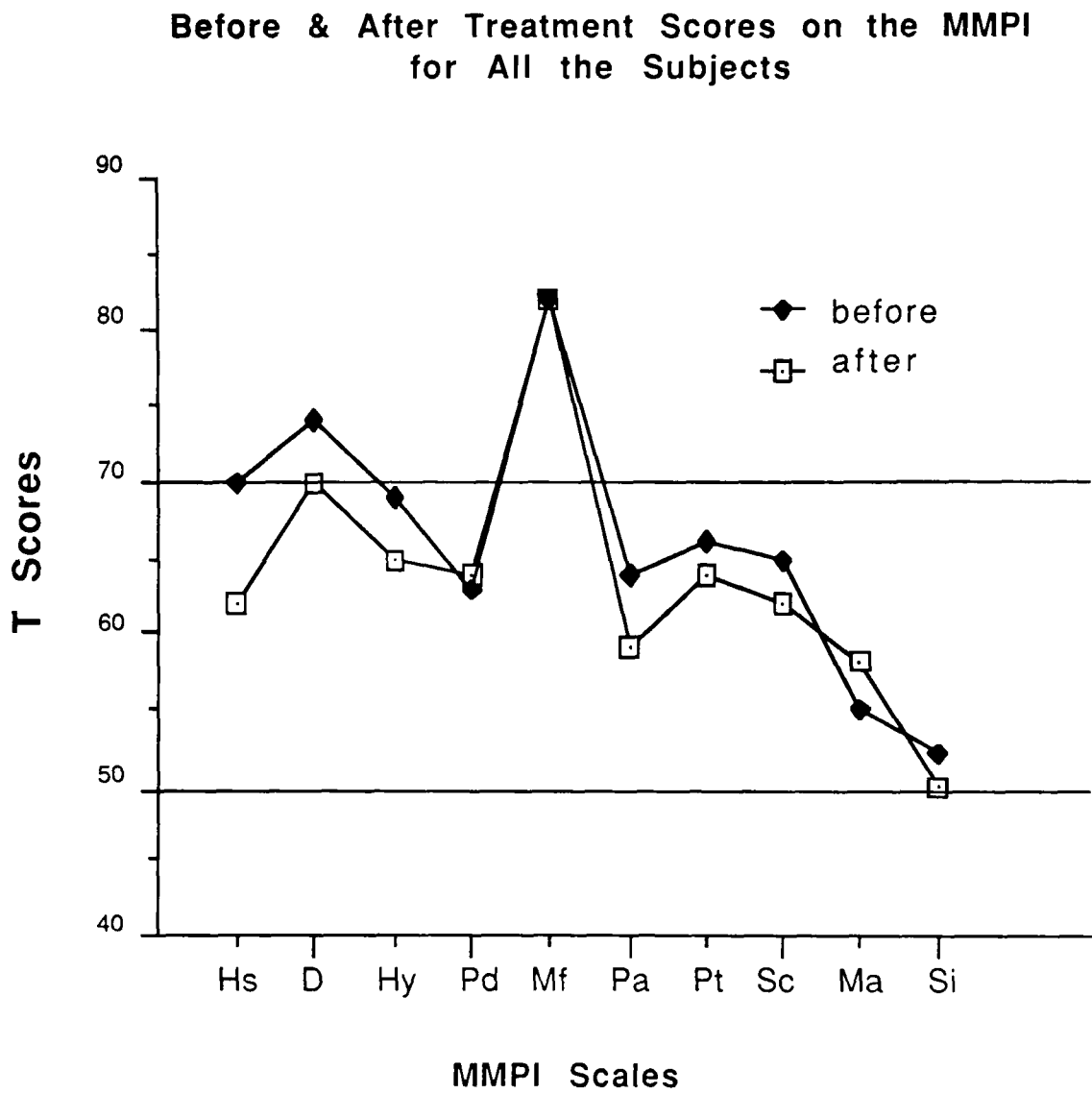
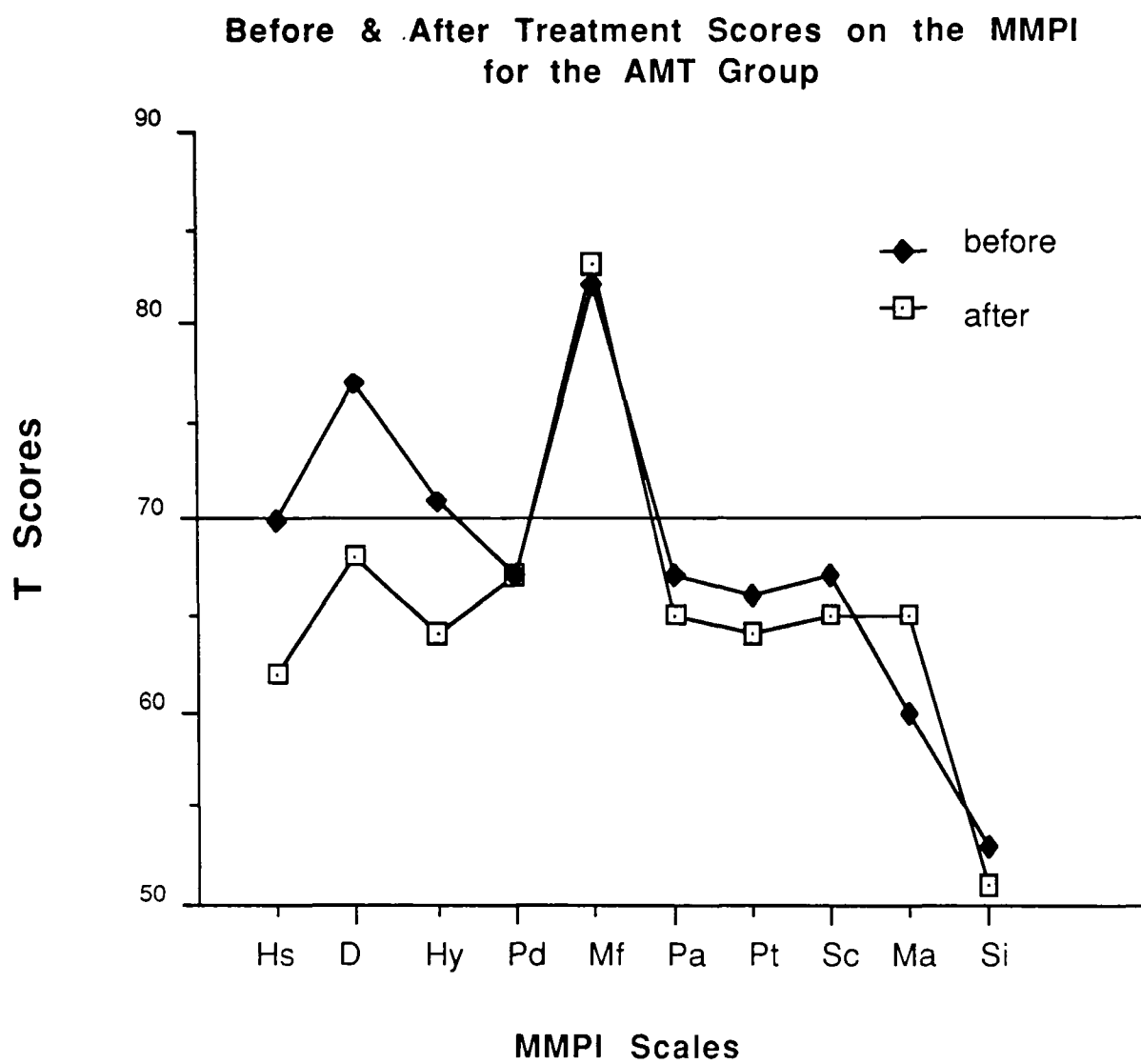


FIGURE 3



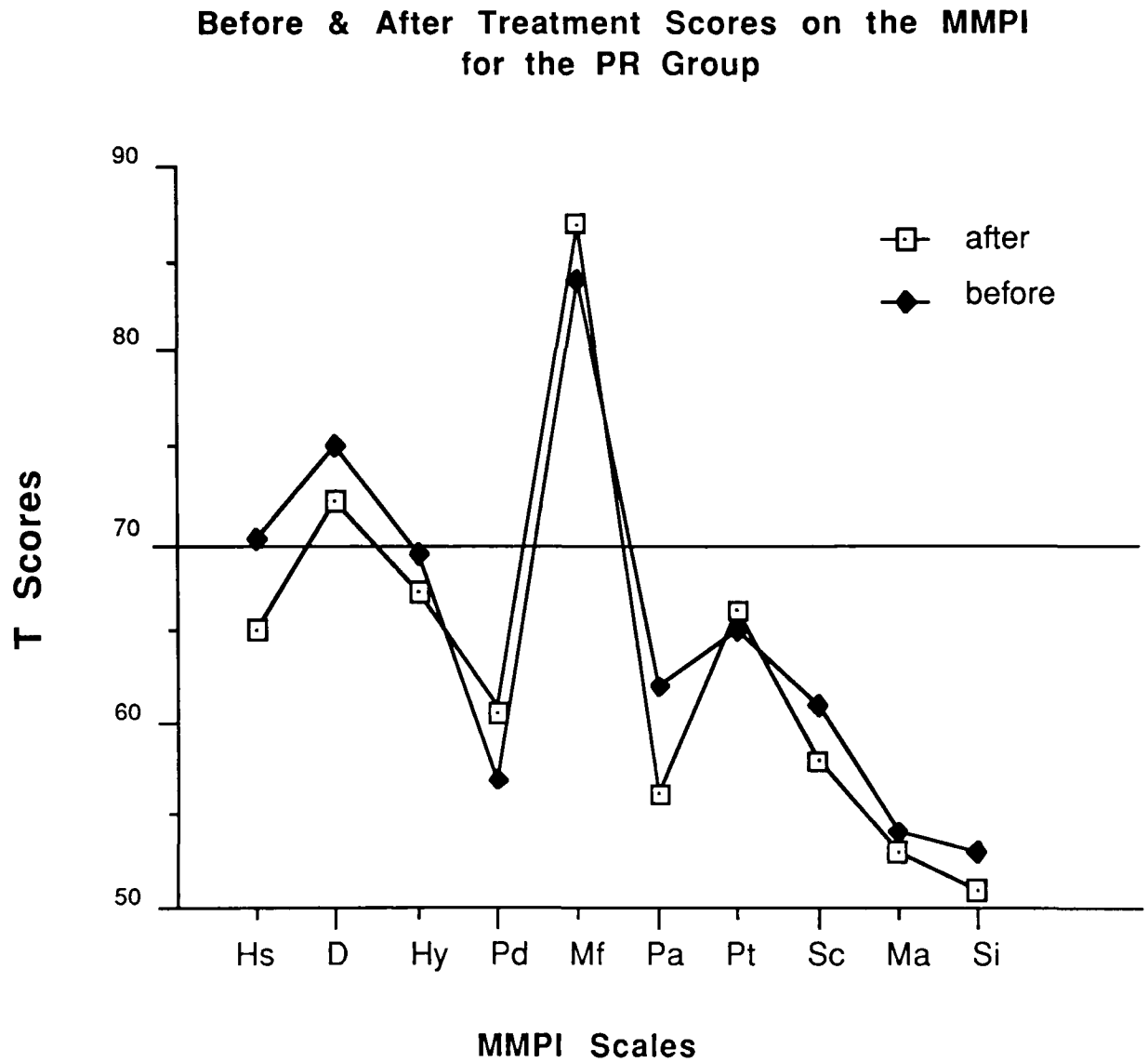
Inventory ($F=10.53$, $p < 0.001$). This indicates that not only AMT but also PR (see Figure 4 for PR's MMPI group profile) were successful in affecting the above mentioned psychological measures. However, no interactions on any of these measures were present, pointing to the fact that AMT is not superior to PR in reducing these psychological indices.

Finally, AMT did not have greater positive effects on morning vigilance (as measured by an increased number of responses on the Four-Choice R. T. test) when compared to PR. Both groups were comparable in performance in this measure as supported by the presence of a within-group difference ($F=19.57$, $p < 0.001$). On the other hand, the absence of any Group x Treatment interaction ($F=1.29$, $p < 0.27$) showed that both AMT and PR were equally effective in increasing vigilance.

Even though predictions have not been formulated as to the effects of therapy on the different sleep stages, repeated measures ANOVAS comparing AMT and PR on these stages showed within-group differences indicating significant decreases in percentage of stage 1 ($F=6.38$, $p < 0.02$) and increases in delta sleep percentage ($F=4.87$, $p < 0.04$). In addition, a within group difference was also observed on self-rated sleep satisfaction ($F=11.53$, $p < 0.001$). However, no interactions were noticeable for these sleep measures. Thus, both treatments affected the sleep stages similarly. The only measure in which an interaction showed a tendency toward significance was the percentage of REM increase after treatment ($F=3.97$, $p < 0.06$). Simple main effects indicated that AMT tended to increase the percentage of REM sleep more ($F=5.37$, $p < 0.04$) than PR ($F=0.57$, $p < 0.47$).

Given the nature of this study, that is assessing the usefulness of a recently developed technique (AMT) with sleep-onset insomniacs by comparing it to an already effective treatment (PR), it was crucial to fully explore the particular impact of that technique on the different psychological and physiological measures monitored. For this reason, simple main effects were

FIGURE 4



calculated, even when there were no interactions, to detect possible tendencies which might clarify the potential usefulness of AMT in actual practice.

Analyses comparing pre to post treatment showed that the AMT group significantly improved on many of the psychological variables. Specifically, significant reductions in trait anxiety ($F=6.39$, $p < 0.03$), depression (as measured by the Beck Depression Inventory) ($F=9.18$, $p < 0.01$), and several personality scales of the MMPI (Hs : $F=5.00$, $p < 0.05$; D : $F=10.70$, $p < 0.01$; Hy : $F=5.25$, $p < 0.04$; and, Pa : $F=5.70$, $p < 0.04$) were observed.

Similar improvements after treatment were also observed on home SOL decrease in latencies ($F=4.66$, $p < 0.05$). Other post-AMT improvements, such as REM % increase ($F=5.37$, $p < 0.04$), a marginal decrease in stage 1 ($F=4.56$, $p < 0.06$), and a marginal increase in responses on the Four-Choice R.T. test ($F=3.90$, $p < 0.08$), were also noted. Finally, subjects in the AMT group expressed more satisfaction with their sleep after treatment ($F=8.73$, $p < 0.01$).

By way of contrast, only a few variables monitored in the PR group improved enough to attain significance. These variables were trait anxiety ($F=10.33$, $p < 0.01$), Paranoia (on the MMPI; $F=6.73$, $p < 0.03$), home SOL ($F=9.85$, $p < 0.01$), and finally, increased number of responses on the Four-Choice R.T. test ($F=30.65$, $p < 0.001$).

In sum, there was a complete absence of interaction between AMT and PR, which indicated that the effects of both therapies were in essence similar. Nevertheless, closer look at the various simple main effects showed that the AMT group significantly improved on many of the psychological variables (i.e. trait anxiety, depression, hysteria, hypochondriasis, and paranoia) and some of the physiological variables (i.e., SOL at home, REM %, and stage 1 %). On the other hand, the PR group showed improvements on only a few of the same monitored variables, that is trait anxiety, paranoia, the Four-Choice R.T. test and home SOL.

Of interest is the fact that even though most insomniacs obtained, demonstrated, as

indexed by the SOL device, lengthy sleep-onset latencies at home (mean pre=71.21 min., post=45.11 min.), the latencies recorded in the sleep laboratory were overall much shorter (mean pre=42.77 min., post=28.00 min. with the polygraph and mean pre=46.03 min., post=32.93 min. with the SOL device). This will be commented on later in the discussion.

High Anxiety (HI-A) versus Low Anxiety (LO-A)

The above results fail to yield conclusive differences between the two therapy groups. This however does not invalidate the concept underlying our main hypothesis. In fact, the basic notion was that trait anxiety was a mediating factor in sleep-onset insomnia. Thus, in the AMT group, trait anxiety would be more reduced, which in turn would shorten SOL, thus demonstrating a possible relationship between trait anxiety and the maintenance of sleep-onset insomnia. In fact, we observed that trait anxiety was equally reduced in both groups. It is therefore consistent with our notion that no significant interaction emerged. However, within each group, there were individual differences in trait anxiety reduction. To investigate the trait anxiety/insomnia relationship, we arbitrarily redivided our insomniac sample into two new groups: one composed of subjects showing a substantial decrease in trait anxiety, the low anxiety group (LO-A), and one of subjects demonstrating a minor decrease in trait anxiety, the high anxiety group (HI-A). Having done this, and if our premise is accurate, we would expect to observe greater improvements in insomnia in the LO-A group (the one with the greatest decrease in trait anxiety after therapy), as compared to the HI-A group.

This arbitrary redistribution of the sample into high and low anxiety subgroups was carried out as follows. As was mentioned earlier, trait anxiety was measured using Spielberger's STAI. When an individual completes this test, a raw score is calculated and then can be translated into a percentile score or a standard score. We used the latter since it gave us a wider

range of scores especially in the case of extreme raw scores thus providing us with more differentiation as anxiety levels increased.

On the basis of these standard scores, the subjects placed in the LO-A group were those whose standard score on the STAI (Trait) decreased 10 points or more while the subjects in the HI-A group were those not showing a decrease of more than 10 points. As shown in Table 4, this arbitrary redistribution yielded 10 individuals per group.

Tables 5 and 6 summarize the means and standard errors of the mean for the personality tests administered as well as for the different sleep parameters for these newly defined groups. As expected, two way repeated measures analyses of variance on the new anxiety groups revealed the same within-group treatment differences as those obtained with the original grouping: reductions in trait anxiety ($F=20.15$, $p< 0.001$), the Beck ($F=12.48$, $p< 0.003$) and the Hs ($F=9.8$, $p< 0.006$), D ($F=11.04$, $p< 0.004$), Hy ($F=4.65$, $p< 0.04$) and Pa ($F=11.30$, $p< 0.003$) scales of the MMPI. In addition, significant interactions also emerged on measures of trait anxiety ($F=7.55$, $p< 0.01$), on the Beck Depression Inventory ($F=6.03$, $p< 0.02$), as well as on the Hysteria ($F=6.47$, $p< 0.02$), Depression ($F=6.74$, $p< 0.01$), Hypochondria ($F=4.19$, $p< 0.05$), Psychopathic Deviance ($F=5.49$, $p< 0.03$), and the Psychasthenia ($F=5.11$, $p< 0.03$) subscales of the MMPI.

Analyses of simple main effects revealed that the LO-A group significantly improved in trait anxiety ($F=14.53$, $p< 0.004$), on the Beck Depression Inventory ($F=37.29$, $p< 0.001$), as well as on several MMPI personality scales (Hysteria : $F=16.18$, $p< 0.003$; Depression : $F=17.49$, $p< 0.002$, Hypochondriasis : $F=18.26$, $p< 0.002$; Paranoia : $F=6.59$, $p< 0.03$, and Psychasthenia : $F=7.63$, $p< 0.02$) (see Figure 5 for LO-A's MMPI group profile).

Similar two way repeated measures ANOVA calculated on the Four-Choice R. T. data revealed a within-group difference ($F=17.66$, $p< 0.001$) indicating a significant decrease from pre to post treatment. However, there was no interaction.

TABLE 4

**NUMBER OF SUBJECTS, SEX & AGE RANGE
(HI-A* VERSUS LO-A#)**

	HI-A (n=10)	LO-A (n=10)
AGE RANGE:	22 63 (mean=33.7)	19 61 (mean=38.1)
SEX:	M=2 F=8	M=4 F=6

#LO-A= group were standard score on the STAI decreased 10 points or more

*HI-A= group were standard score on the STAI did not decreased 10 points or more

TABLE 5

**MEANS & S.E.M.@ FOR THE PERSONALITY AND
PERFORMANCE TESTS (HI-A & LO-A)**

	Group	Before		After		T.E.#	INTER.#	S.M.E.#
		Mean	S.E.M.	Mean	S.E.M.			
STAI	HI	84.9	2.88	76.6	4.85	**	**	*
	LO	93.1	2.61	58.6	10.30			**
Beck	HI	8.33	1.44	7.11	1.69	**	*	*
	LO	12.9	2.85	6.10	2.42			**
MMPI:								
Hs	HI	58.7	2.46	57.5	4.27	**	*	ns
	LO	70.6	3.79	57.0	2.87			**
D	HI	66.3	4.65	64.9	4.05	**	**	ns
	LO	74.1	5.14	62.7	3.08			**
Hy	HI	64.8	2.18	64.6	4.30	*	*	ns
	LO	68.9	2.54	61.2	1.89			**
Pd	HI	60.0	5.09	65.0	3.24	ns	*	ns
	LO	64.7	3.50	61.6	2.96			ns
Mf	HI	56.9	4.52	52.3	5.56	ns	ns	*
	LO	47.9	4.90	47.9	5.42			ns
Pa	HI	61.4	1.87	56.1	3.64	**	ns	*
	LO	67.1	2.90	62.0	3.00			*
Pt	HI	62.1	3.55	63.7	4.25	ns	*	ns
	LO	62.2	3.91	56.7	3.15			*
Sc	HI	60.5	3.00	61.0	5.28	ns	ns	ns
	LO	62.6	4.35	58.9	2.88			ns
Ma	HI	54.3	5.27	57.8	5.41	ns	ns	ns
	LO	55.8	3.36	59.8	2.39			ns
Si	HI	50.5	2.88	50.8	2.42	ns	ns	ns
	LO	54.9	4.12	51.8	3.22			ns
4-Ch.	HI	979	36.11	1029	30.94	**	ns	**
	LO	936	27.48	990	23.36			*

** : p < 0.01; * : p < 0.05; ns : p > 0.05

: T.E.= Within group (treatment Main Effects); Inter.= Interaction; S.M.E.= Simple Main Effects

@= Standard Error of the Mean

TABLE 6

**MEANS & S.E.M.@ FOR THE DIFFERENT SLEEP
PARAMETERS (HI-A & LO-A)**

	Group	Before		After		T.E.#	Inter.#	S.M.E.#
		Mean	S.E.M.	Mean	S.E.M.			
SOL Dev.	HI	76.08	13.19	64.18	13.97	* *	ns	ns
	LO	66.33	9.00	26.04	5.06			* *
SOL Poly.	HI	61.51	21.54	36.83	11.74	ns	ns	ns
	LO	24.04	3.09	19.18	2.87			ns
% Sleep Stages								
1	HI	0.09	0.03	0.03	0.01	*	ns	ns
	LO	0.10	0.05	0.03	0.01			ns
2	HI	0.52	0.02	0.55	0.04	ns	ns	ns
	LO	0.56	0.03	0.55	0.03			ns
3	HI	0.09	0.01	0.10	0.02	ns	ns	ns
	LO	0.08	0.01	0.08	0.01			ns
4	HI	0.06	0.02	0.07	0.02	*	ns	ns
	LO	0.04	0.01	0.10	0.02			*
Delta	HI	0.16	0.02	0.18	0.03	*	ns	ns
	LO	0.12	0.02	0.17	0.02			* *
REM	HI	0.20	0.03	0.21	0.02	ns	ns	ns
	LO	0.21	0.01	0.22	0.01			ns
Sleep Effic.	HI	0.88	0.06	0.92	0.02	ns	ns	ns
	LO	0.94	0.01	0.93	0.02			ns
Sleep Satisf.	HI	2.50	0.22	2.25	0.21	* *	ns	ns
	LO	2.41	0.14	1.97	0.18			*

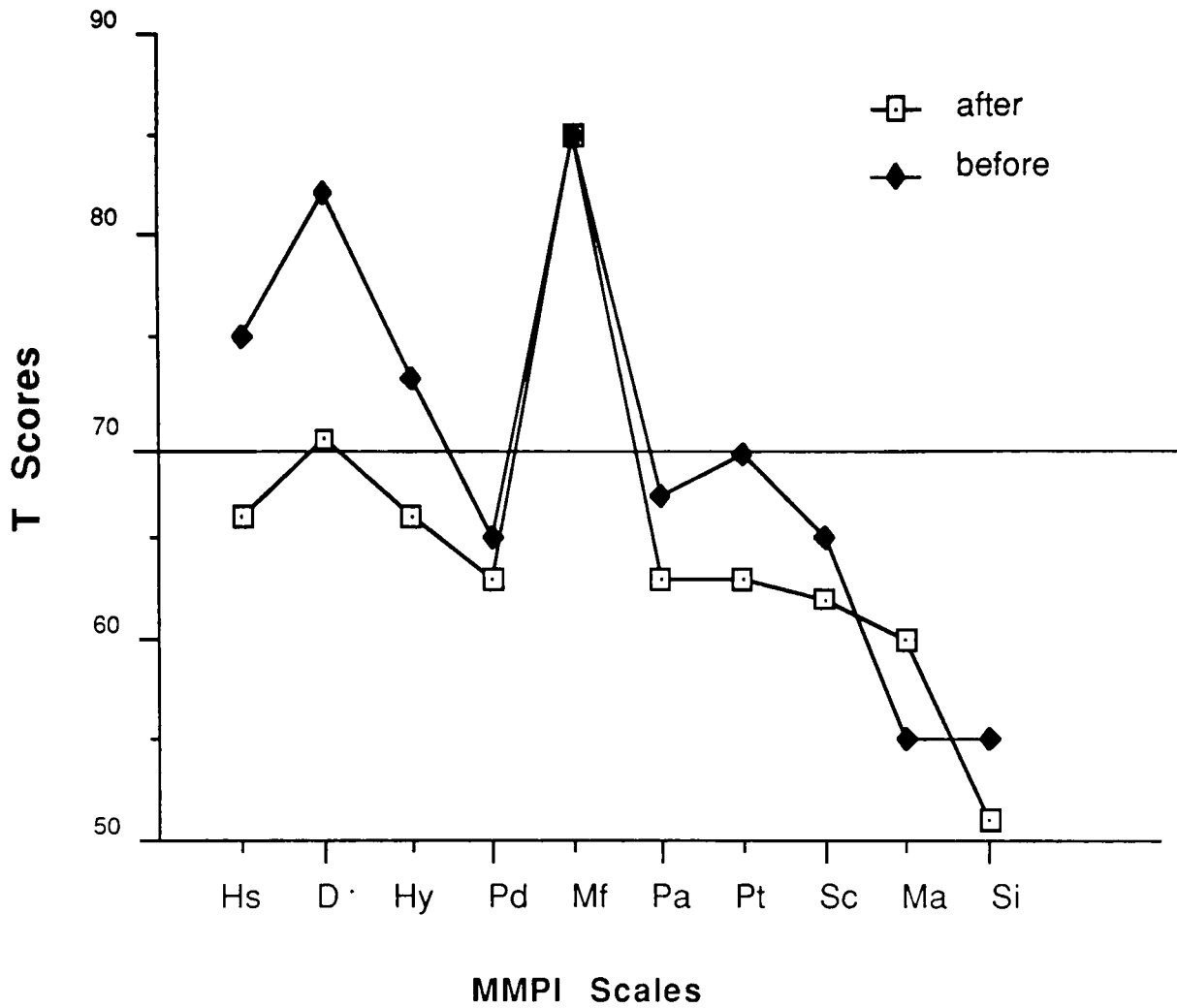
** : $p < 0.01$; * : $p < 0.05$; ns : $p > 0.05$

: T.E.= Within group (Treatment Main Effects); Inter.= Interaction; S.M.E.= Simple Main Effects

@= Standard Error of the Mean

FIGURE 5

**Before & After Treatment Scores on the MMPI
for the LO-A Group**



On the sleep latency measures, a within-group treatment difference ($F=13.18$, $p<0.002$) and a tendency toward a significant interaction ($F=3.90$, $p<0.06$) was present on the measures of sleep-onset latency at home. Simple main effects on the LO-A and the HI-A groups revealed an important decrease in home SOL for the LO-A group ($F=22.79$, $p<0.001$) but no significant decrease in the HI-A group.

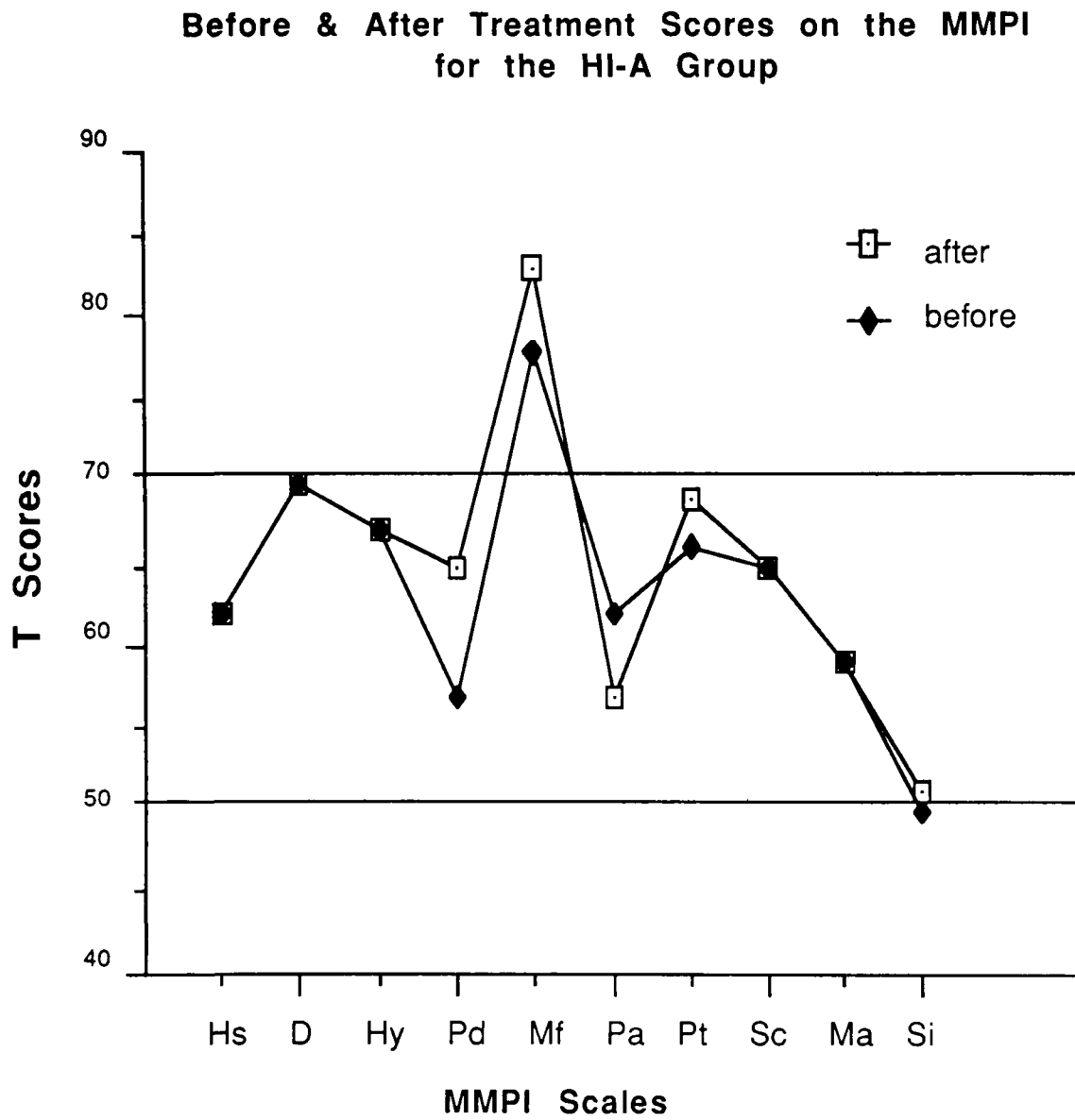
As for the different sleep parameters, within-group differences were also observed in stage 1 decreases ($F=5.65$, $p<0.02$), stage 4 increases ($F=4.40$, $p<0.05$), delta sleep increases ($F=5.25$, $p<0.03$), and on self-reported sleep satisfaction ($F=11.16$, $p<0.001$). However, no interactions on these measures were found indicating that both the HI-A and the LO-A group had similar stage 1 proportions and sleep satisfaction ratings.

Subsequent tests for simple main effects on the LO-A group showed that the subjects significantly increased their amount of stage 4 sleep after treatment ($F=6.38$, $p<0.03$), as well as in their overall delta sleep (stage 3 and 4 combined) ($F=9.98$, $p<0.01$) after treatment. In addition, the LO-A subjects reported sleeping significantly better after therapy ($F=6.66$, $p<0.03$), while no such significance in self-rated sleep satisfaction was detected in the HI-A group.

Of interest among other simple main effects was the total lack of improvement in both the HI-A and LO-A groups in sleep-onset latencies in the laboratory (HI-A, $F=1.20$, $p<0.30$; LO-A, $F=1.93$, $p<0.20$).

The simple main effects observed for the LO-A group are in contrast to those calculated for the HI-A group, which overall showed improvements on markedly fewer measures. The measures showing improvements were trait anxiety ($F=7.65$, $p<0.04$), the Four-Choice R.T. performance ($F=15.52$, $p<0.004$), and the Paranoia ($F=4.99$, $p<0.05$) and Masculinity/Femininity ($F=5.40$, $p<0.04$) scales of the MMPI (see Figure 6 for HI-A's MMPI group profile). Two additional measures, the Psychopathic Deviance scale of the MMPI, and

FIGURE 6



the sleep satisfaction self-rating almost reached significance ($F=3.84$, $p < 0.08$ and $F=4.57$, $p < 0.06$ respectively).

In sum, there are minute differences between the two therapies which are not significant enough to show up as interactions but that are nevertheless still noticeable with the calculation of simple main effects.

Correlates of Improvements in Sleep-Onset Latency

Our results point to a clear reduction in home SOL. We felt it would be interesting to examine which variables correlated with this improvement. Pearson correlation coefficients were calculated between SOL reductions at home and changes in psychological measures such as the STAI (Trait), the Beck Depression Inventory and the different MMPI scales (see Table 7). In other words, what were the best predictors or correlates of reduction in SOL? Significant or near significant positive correlations were found between home SOL improvements and the STAI-Trait (standard scores) ($r=0.42$, $p < 0.06$), the Beck Depression Inventory ($r=0.56$, $p < 0.01$), and some MMPI scales such as Hysteria ($r=.402$, $p < 0.07$), Hypochondriasis ($r=0.62$, $p < 0.01$), Psychastenia ($r=.67$, $p < 0.01$), and Schizophrenia ($r=0.55$, $p < 0.01$).

As shown in Table 8, no Pearson correlation coefficients between SOL improvements at home and changes in laboratory sleep parameters, sleep satisfaction and overall vigilance (as measured by the Four-Choice Reaction Time test) attained significance. Nevertheless, it was clear that individuals improving on their SOL's at home tended to increase their amount of Delta sleep (stages 3 and 4 combined) at the expense of stages 1 and 2. In addition, this improvement in sleep-onset latency also had a tendency to be combined with better performance on the Four-Choice, possibly indicating increased morning vigilance as sleep improved.

TABLE 7

**CORRELATION COEFFICIENTS BETWEEN SOL
IMPROVEMENTS AT HOME AND CHANGES
IN PSYCHOLOGICAL MEASURES**

	SOL
STAI (Standard Score)	0.417 *
BECK	0.559 **
MMPI:	
Hs	0.538 **
D	0.190 ns
Hy	0.606 **
Pd	0.216 ns
Mf	0.114 ns
Pa	0.158 ns
Pt	0.680 **
Sc	0.548 **
Ma	0.103 ns
Si	0.156 ns

*: $p < 0.06$; **: $p < 0.05$; ns: $p > 0.05$

TABLE 8

CORRELATION COEFFICIENTS BETWEEN SOL IMPROVEMENTS AT HOME AND CHANGES IN LABORATORY SLEEP PARAMETERS, SLEEP SATISFACTION AND VIGILANCE

	SOL
STAGE 1	0.016 ns
STAGE 2	0.148 ns
STAGE 3	- 0.146 ns
STAGE 4	- 0.066 ns
DELTA (3+4)	- 0.173 ns
SLEEP SATISF.	0.189 ns
FOUR CHOICE	- 0.201 ns

ns: $p > 0.05$

DISCUSSION

AMT versus PR

In interpreting the above mentioned results, special attention must be directed at the possibility of Type I errors. Given the many variables employed, in combination with a small sample of subjects, we have increased the probability of attaining significance by chance alone. This was counterbalanced by making very clear predictions as to the effects of therapy on their respective group.

We had predicted that both AMT and PR would reduce trait anxiety and our results supported this. However, our results failed to support the prediction that AMT would lead to a greater reduction in trait anxiety as compared to PR. Nevertheless, many significant simple main effects, namely on home SOL, REM %, sleep satisfaction, trait anxiety, depression and some scales of the MMPI (Hs, D, Hy, Pa), indicated that some distinctions could be made between the efficacy of AMT and that of PR.

Hypothesis 2 was only partially supported. Both therapies were successful in reducing sleep-onset latencies in the home environment. However, SOL measures in the laboratory were left unchanged, even after the end of treatment. This finding is in direct contradiction with Ladouceur and Gros-Louis (1984) and Lacks' (1987) statements evaluating the objective success rate of progressive relaxation at around 59%

Obviously, since the laboratory SOL's remained roughly the same, hypothesis 3, stating that AMT would lead to greater SOL reductions in the laboratory was not supported. Even though hypothesis 4 was not supported either, AMT was however shown to be as effective

as PR in reducing sleep-onset insomnia at home.

To a certain degree, hypothesis 5 was supported. AMT significantly affected depression (as measured by the Beck and the MMPI), in addition to reducing scores on the Hs, Hy and Pa scales of the MMPI. In fact, the changes observed in the AMT group closely resemble those observed in Montplaisir et al's (1984) relaxation group. On the other hand, PR in this study, was only successful in decreasing the Pa scale of the MMPI. Finally, contrary to hypothesis 6, AMT did not have greater positive effects on morning vigilance than PR. The overall effects were similar in both groups.

One obvious question was : "Was AMT, as a new therapeutic tool, used to its full potential in treating individuals suffering from sleep-onset insomnia ?" First, it is important to restate the observation that AMT was to a certain degree measurably superior to PR. AMT subjects, as a group, fell asleep faster, felt better and were overall more satisfied with their sleep. Nevertheless, as researchers, we cannot be satisfied with this limited success. We must probe the results in an effort to know if that change was in any way hindered by some procedural applications of the technique with insomniacs. In fact, this may have been the case. In this study, AMT was administered using a proven cassette format. Some subjects remarked that they found it difficult to listen to the final AMT cassette at bedtime. These individuals were required to heighten their anxiety several times during the exercise. Some complained of still feeling somewhat anxious even after the completion of the nightly session. They reported that this voluntary increase in amount of anxiety at bedtime hindered on their sleep-onset. Certain individuals thought it was counterproductive to raise anxiety at a time when it should be at its lowest.

Obviously, our results suggested otherwise. Still, dealing with this "worry about overworrying" could very possibly increase the therapy's effectiveness. One way of bypassing this obstacle would be to instruct subjects to listen to the AMT tape during the day while

practicing only the progressive relaxation component of the treatment at bedtime. With these instructions, the individual would profit fully from a technique which otherwise may not be tailored to the special needs of insomniacs.

It is also possible that, for severe insomniacs at least, AMT would only reach its fullest potential if administered in its original format, that is in weekly therapy sessions with a live therapist. This may be especially true with individuals whose insomnia is more chronic and thus often accompanied by psychopathologies such as severe depression or mild psychosis. Here, AMT would be best used as a component of a larger, more complex multimodal approach aimed at modifying various behaviors, one of them being the sleep-onset problem. But from the results obtained in this study, it is safe to assume that, with sleep-onset insomniacs showing only slight abnormalities following psychometric testing, AMT (in its cassette format) is a far more effective treatment than PR alone.

PR'S Efficacy

Indirectly, this study lends further support to the efficacy of PR with sleep-onset insomniacs. On the other hand, our results did not confirm some of the usual observations often reported in the mainstream literature on the effects of relaxation on insomnia.

As suggested by Lacks (1987), the past success of PR in dealing with sleep-onset insomnia was to a great extent based on subjective reports from the subjects. We have argued for the need for more objective measures, from which the idea to develop and use the SOL device, in conjunction with the polygraph, has originated. To our knowledge, this research is the first to demonstrate that PR (and AMT), objectively improve sleep-onset latencies at home. This home measure appears even more important now that there is an increasing number of insomniac studies reporting shorter sleep-onset latencies in the laboratory as compared to longer

self-reported latencies. This will be commented on at length later on in the discussion.

Even though the group of insomniacs who underwent PR therapy improved their SOL's at home, they did not significantly improve in their overall evaluation of their sleep satisfaction. In addition, their basic psychological makeup remained relatively unchanged, with the exception of trait anxiety and paranoia (as assessed by the MMPI). This is in sharp contrast with the results reported by Montplaisir et al., (1984). In their study, twenty insomniacs received relaxation training over a three months period. Results on the MMPI performed at the beginning and at the end of the study revealed statistically significant decreases for the depression, psychasthenia, schizophrenia, hysteria, hypochondriasis and social isolation subscales. In addition, anxiety (as measured by the 16 PF) decreased significantly in the relaxation group, but not in the control group.

Comparing the effects of relaxation on sleep and personality, a few explanations can be suggested to account for the differences between the Montplaisir study and ours. The important difference between the two studies lies in the length of the relaxation treatment program offered. While our treatment program lasted nine weeks, the relaxation program described in Montplaisir's protocol terminated after twelve weeks. However, it is unlikely that three additional weeks of therapy would amount to such a change in personality structure.

Still another difference that might help us explain the discrepancies in results is the variation in therapy format. In the Montplaisir study, a therapist taught relaxation to the subject then met him/her every two weeks for practice and progress feedback. This, in turn, focused attention upon the subject. In our protocol, the same procedures were followed but a lot of emphasis was put on practicing the relaxation cassette at home. The therapist's role was therefore restricted to re-explaining the treatment's procedures first presented during the opening session. This, in effect, made the therapist-subject relationship more "distant". It is possible that

the differential quality of contacts with the therapist might have precipitated the subsequent personality changes in the Montplaisir group.

In concluding their study, Montplaisir et al. (1984) stated: "These results show that relaxation therapy not only improves sleep and daytime vigilance but markedly modifies personality profiles derived from psychometric tests" (pp. 147). The suggestion made here that relaxation alone can lead to profound psychological changes in the basic personality makeup is to be accepted with reserve. Apparently, the model used by Montplaisir and his colleagues to account for their results is that, for certain insomniacs at least, the lack of proper sleep causes deep personality changes. Restoration of sleep through relaxation would, in effect, help the individual return to his/her "basic" personality structure. If this is in fact the underlying model employed by the authors, then our study sheds some doubts on its generalizability. In analyzing and interpreting our data, it becomes more and more apparent that on the contrary, prior to sleep improvements, certain aspects of personality must first be positively affected. To further investigate this assumption, we must at this point examine the analyses of the LO-A versus the HI-A redivision of groups.

LO-A versus HI-A

Redividing our sample into LO-A (the one recording the greatest decrease in trait anxiety) and HI-A groups (the one recording the lowest decrease in trait anxiety) permitted us to isolate to a certain extent the relationship between trait anxiety and the reduction in sleep-onset insomnia. The subsequent working hypotheses tested were similar to those used with the two therapy groups. Thus, it was expected that the LO-A group would exhibit SOL reductions in the home environment when compared to the HI-A group.

It was also expected that in the LO-A group, significant decreases in the negative psychological correlates of insomnia, such as depression (as measured by the Beck Depression Inventory) and certain MMPI subscales (i.e. Hs, D, Hy, and Pt), would be recorded.

Analyses did show that the LO-A group significantly improved on a number of psychological and physiological measures. In the LO-A group, these improvements indicated better psychological well being after the treatment, and greater SOL decreases at home. In contrast, SOL in the laboratory remained relatively unchanged after treatment in both anxiety groups. Finally, the HI-A and LO-A group gave significantly better sleep satisfaction ratings after the treatment as compared to before the treatment.

It was notable to observe that the HI-A group also demonstrated a significant decrease in trait anxiety without improving, as the LO-A group did, on numerous other psychological and physiological variables.

So apparently, the relationship between trait anxiety and sleep-onset insomnia, may have been somewhat incomplete. This in turn, incited us to return to the basic results to possibly identify any additional etiological factors, other than trait anxiety.

Depression, Anxiety, and Insomnia

As shown earlier, both the AMT and the PR groups significantly decreased in trait anxiety after their respective therapy. However, depression in the two groups was being influenced in very different ways. Planned contrasts showed that PR had no significant effects on depression as measured by both the MMPI and the Beck Depression Inventory. On the other hand, both measures of depression were profoundly reduced in subjects who were offered AMT. This indicates that, with a decrease in trait anxiety, a lowering of depression may be an additional component crucial to the successful treatment of sleep-onset insomnia.

An examination of the role of depression in the treatment outcome of the HI and LO anxiety groups yielded similar results. The HI-A group, even after significant decreases in trait anxiety, did not show any signs of improvement its level of depression. However, the sweeping positive effect observed throughout the LO-A measures after treatment also included a significant decrease in depression as measured by the Beck and the MMPI. So apparently, in order to observe a meaningful improvement in sleep-onset latency and personality structure, a significant decrease in depression alone or combined with trait anxiety must at the onset be noted. This would account for the strong correlation between depression and sleep-onset latency, a correlation that is even stronger than the one found between trait anxiety and sleep-onset latency.

Having brought this relationship to light, it is interesting to notice that a similar pattern has also been observed in another independent study,. Montplaisir et al. (1986), after offering relaxation to their insomniacs, reported that their sample improved most significantly on the depression scale of the MMPI and in anxiety (as measured by the MMPI and one of the second order factors of the 16 PF). This indirectly supports our own findings.¹

To sum up this section, it became evident that the manipulation of trait anxiety alone could not account for the overall positive changes in personality and sleep correlates that occurred in our insomniac sample. Closer investigation of the data suggest an the important role of depression in these results.

Thus a joint decrease in trait anxiety and depression appears closely associated with improvement in sleep-onset latency, sleep satisfaction and personality. Some would argue that similar results could be achieved by improvements in depression alone. Depression is known to be often reported in conjunction with insomnia (Jones et al., 1987). From our results, it can be postulated that: 1) depression and trait anxiety work jointly to maintain sleep-onset insomnia; or,

¹ Nevertheless, this still does not shed any light as to why Montplaisir et al.'s (1984) relaxation group reported greater personality changes than our PR group.

2) depression alone maintains this type of insomnia, making trait anxiety a by-product of the whole depression-insomnia "system". It was not the ultimate focus of this study to resolve such a controversy. Nevertheless, it is our intention to propose at this point a working model, based on our results, which may help in the future investigation of the role of depression in insomnia.

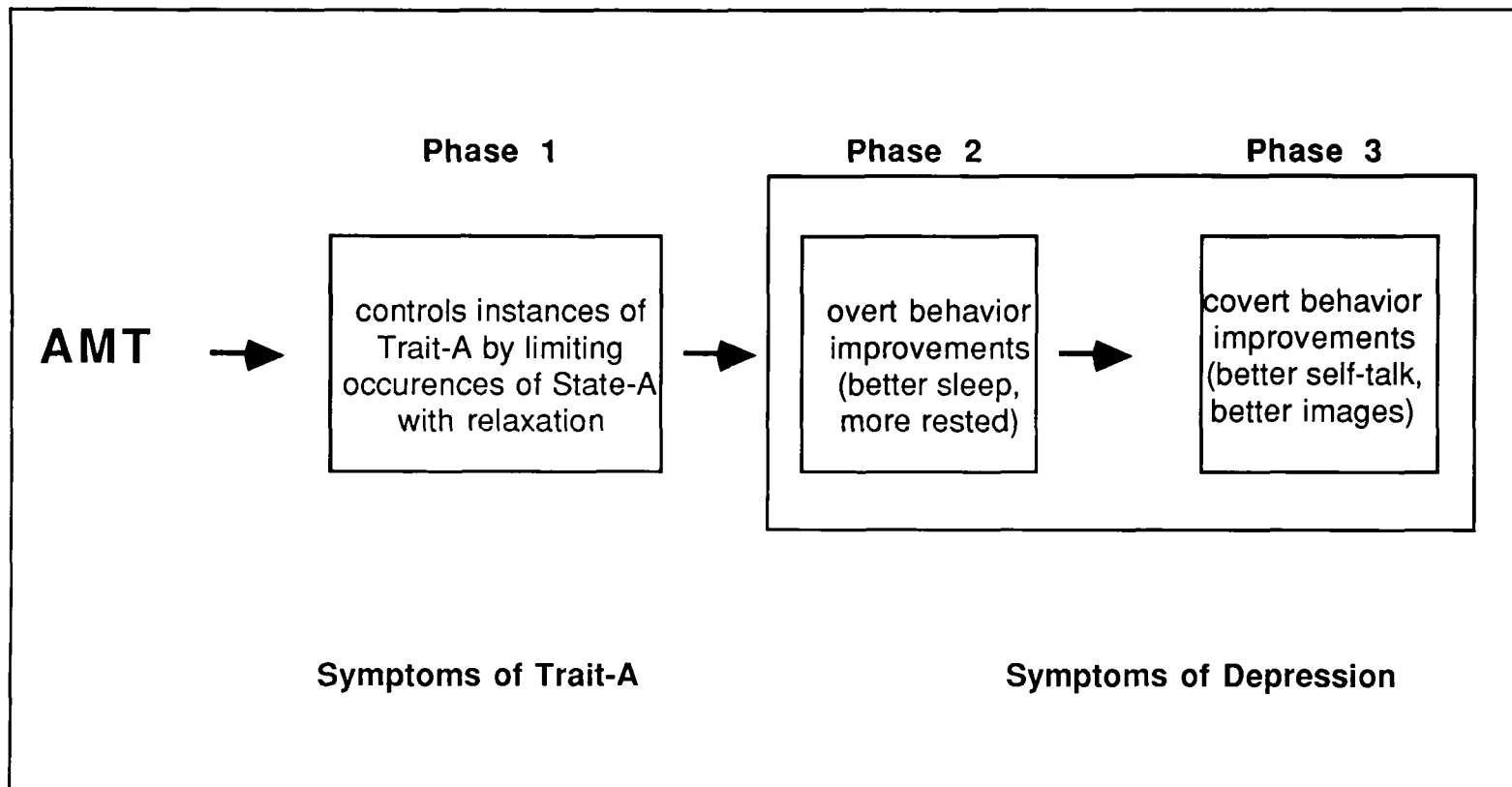
The Chicken or the Egg : A Behavioral Model

If we accept the importance of depression in possibly maintaining sleep-onset insomnia, a fundamental question, the answer to which may help elucidate the role depression plays remains: "Do improvements in personality (decrease in depression alone or in conjunction with a decrease in anxiety) lead to improvements in sleep or is it the other way around?" This question is simply a newer version of the old chicken and egg controversy (i.e. which came first?). From our results, it is possible to propose a behavioral model which can help one conceptualize the progression of therapy and better understand how improvements in behavior take place. But most of all, this model is intended as a tool to guide research in the hope of producing an even better model.

As Figure 7 illustrates, in this scheme the effects of AMT would be threefold. The first effect (occurring during Phase 1) would influence the symptoms often used to define trait anxiety. By decreasing the occurrences of state anxiety with the aid of relaxation, one would in effect perceive and report less trait anxiety. The increased frequency of state anxiety appears to be a contributing factor in the maintenance of high levels of trait anxiety. This overall effect would constitute Phase 1. As a consequence of pairing relaxation with sleep-onset, and the visualization of pleasant and success scenes, Phases 2 and 3 would ultimately follow. In Phase 2, improvements in overt behaviors (i.e., faster sleep-onset latencies, less nocturnal

FIGURE 7

A Behavioral Model of the Effects of AMT
on Trait-A and Depression



awakenings, waking up more rested) are observed. Finally, if therapy continues to be successful, Phase 3 will follow. In this final phase, covert behaviors (i.e., positive self-talk, positive visualization) will also begin to improve. The amelioration occurring during the second and third phases are all betterment of behaviors which in sum are used to describe symptoms of depression.

This is in line with Meichenbaum's (1971) earlier conceptualization of cognitions (or covert behaviors) where these are regarded as rapidly occurring "automatic thoughts" that can lead to maladaptive behavior and negative emotions which have to be "deautomized" and interrupted in therapy (Eifert, 1984). Thus, Meichenbaum (1971) maintained that cognitions are inner or covert symbolic stimuli which are subject to the same psychological learning principles as other behaviors. Thus, in our model, Phase 3 would be the actual modification of these covert behaviors in such a way as to remove the subject's overall maladaptive behaviors and accompanying negative emotions. So in essence, trait anxiety is first reduced in Phase 1 by the control of certain behaviors through relaxation. This is then followed, if allowed by the treatment, by modifications in other overt behaviors (Phase 2) and covert behaviors (Phase 3), which together results in a lowering of reported depression.

Using this model, we discover that PR limits its effects to the first two phases. Behavioral measures of trait anxiety, according to the model, decrease in Phase 1. This reduction in trait anxiety is in fact observed in the PR group. Then, in Phase 2, certain sleep parameters are somewhat affected, especially SOL at home and, to a lesser extent, self-reported sleep satisfaction. These improvements are not substantial enough to ultimately affect behaviors associated with self-reported depression. This is in contrast to AMT, which, in addition to affecting Phases 1 and 2, has an effect that crosses over to Phase 3, thus explaining the decrease in depression.

This scenario is in agreement with many of the reviewed studies assessing relaxation as

a viable therapy for insomniacs. Sleep often improves, but for many, sleep satisfaction and personality profile often remain unchanged. An earlier stated exception to this rule is the Montplaisir et al. (1986) study. With the aid of this model, the only plausible interpretation of the Montplaisir results is that, inherent to their therapy was a component which helped modify covert behaviors enough for depression to have been ultimately affected.

On the Measures Used

The discussion would not be complete without a rapid review of the different instruments used in the study. Although certain of these were very useful, others fell somewhat short of our overall expectations.

1) The psychological tests

A fair amount of information was gathered using the STAI, the Beck Depression Inventory and the MMPI. These tests were easily administered and in return, gave us insightful information on the subjects' progress in therapy. While the MMPI took approximately an hour to complete, filling out the Beck and the STAI took subjects no longer than five minutes per test. Rapid, yet accurate testing is often an important factor in developing a successful research protocol or an adequate therapy plan. Testing procedures which are too lengthy will only frustrate the subject/client, in the end affecting the test's outcome.

Given the proposed importance of anxiety and depression in the treatment of insomnia, it is recommended that the MMPI, the Beck and the STAI be used as a psychometric assessment and monitoring battery in therapy and/or research. For example, prior to treatment/research, all three tests would be administered. Then, the Beck and the STAI would be readministered periodically during therapy, or the experiment, in order to monitor treatment progress or any

changes subsequent to the experimental manipulation. It is important to note that the STAI in its entirety (the State and Trait forms) should be administered. In addition to measuring variations in trait anxiety, state anxiety would also be monitored, allowing a better understanding of the effects that fluctuations in state anxiety would have on sleep-onset. Finally, at the end of therapy/research, the MMPI would be used again to assess the extent of the therapeutic/experimental change. If we accept the model developed earlier, it is expected that, in conjunction with anxiety and depression decrease, significant improvements in sleep and sleep satisfaction would be observed.

2) Polysomnographic measures

The use of the polygraph enabled us to confirm that, in fact, insomniacs do in general sleep better in a foreign environment. One interesting and important finding was the nearly complete absence of polysomnographically measurable sleep disturbances in the laboratory. This in itself strongly supports Jacob et al.'s (1988) and Haynes et al.'s (1982) conclusion that, as predicted by the stimulus control paradigm, insomniacs in the sleep laboratory take less time to fall asleep than the usual latencies reported at home. The same authors attributed this to the fact that stimuli associated with wakefulness at home are no longer present in the sleep laboratory.

Furthermore, it was observed that in the AMT group, REM percentage significantly increased after treatment. This indirectly lends support to the theory first presented by Greenberg et al. (1968 A; 1968 B), which states that increased REM may play an important role in the restoration of psychological function. Polysomnographic measures also permitted us to observe significant delta wave increases in the LO-A group. This lends some support to the frequently reported observation which ties chronic insomniacs with reduced slow wave sleep, particularly stage 4 slow wave sleep (Globus et al., 1974; Coursey, Buchsbaum, & Frankel, 1975; Frankel et al., 1976; Gaillard, 1976, 1978; Hauri, 1983; Reynolds et al., 1984).

Another observation which is often reported is the apparent relationship between depression and slow wave sleep. Individuals who are depressed are observed to have much less delta sleep than non-depressed individuals (Taub et al., 1978; Borbely et al., 1984). Our results further support this notion given that subjects reporting being less depressed after therapy also modestly increased in their sleep. Hopefully, these polysomnographic changes in the laboratory (increased REM and delta sleep), which suggest a closer to normal sleep pattern and architecture, will also hold true in the home environment. There appears to be no reason to suspect the contrary but only home polysomnographic monitoring can give us a definite answer.

Faced with mounting data ill-advising long-term polygraphic monitoring of insomniacs in the laboratory, we now must begin considering in this specific case other alternatives, such as home monitoring. But, combined with an in-depth diagnostic interview, the polygraph will always remain a vital instrument in the proper diagnosis of various sleep disorders, including certain forms of insomnia. We are reminded of its usefulness when realizing that of 123 patients recently diagnosed (without the aid of a polygraph) as suffering from various sleep disorders, 49% of the sample (74 cases) were later found (with the polygraph) to have been misdiagnosed or that other important information had been missed (Jacobs et al., 1988).

3) The SOL monitor

Development of the SOL monitor followed the need to avoid the numerous drawbacks of subjective reporting, in the laboratory as well as in the home environment (Coursey and Frankel, 1977; Coursey et al., 1980; Freedman and Sattler, 1982). Insomniacs too often overestimate their sleep-onset latencies and the general severity of their condition (Borkovek, 1982). The SOL monitor became an appropriate apparatus to measure sleep-onset latency in the most unobstructive way possible. The information it relayed was very important in this study.

For example, the sleep-onset latencies at home and in the laboratory (as measured by the SOL device) were shorter than what was subjectively experienced and reported by the subjects. Thus, the use of the SOL device steered us away from the inaccuracy of subjective reporting.

It was clearly demonstrated that, in considering sleep-onset latency, insomniacs behave much differently at home as compared to how they behaved in the laboratory. The home sleep-onset latencies very much resembled what was subjectively experienced and reported by the subjects. This again lends some support to the stimulus control paradigm which emphasizes environmental cues surrounding the sleeping environment.

In spite of its numerous advantages, laboratory monitoring still remains an expensive and time consuming procedure in assessing any individual's normal (or abnormal) sleep behaviors. Other monitoring techniques, such as the Oxford Medilog 9000 System (a home sleep monitoring device; Sewitch and Kupfer, 1985), is a distinct advantage over standard laboratory monitoring, given that it allows the individual to be assessed in his/her natural environment. On the other hand, the need for overnight electrode placement necessitates the services of a trained technician in addition to accessibility to specialized equipment. Thus, even though a clear improvement over standard laboratory recording, the use of the Oxford Medilog 9000 System is still not without its own disadvantages. In sum, when considering the measurement of sleep-onset latency on a daily basis, the SOL monitor has some distinct advantages. It is inexpensive, portable, easy to use, non-intrusive, and quite accurate.

What does the SOL device really measure? Polysomnographic recording illustrated the fact that many insomniacs, in trying to fall asleep, frequently alternated between wakefulness and stage 1 before finally reaching stage 2. Yet, this wake-stage 1 shifting apparently did not affect the SOL monitor. On the other hand, a high correlation was obtained between onset of stage 2, as measured by the polygraph, and the SOL device's final readout. Interestingly, the SOL monitor systematically overestimated sleep-onset latencies by an average of about eight minutes.

This is a fairly respectable approximation considering the lengthy latencies recorded in our insomniac sample. Still, a portion of the overvaluation of SOL with the clock device may be due to the five minutes of non-button pressing required in order to record SOL, whereas stage 2 latencies with the polygraph, are identified, as suggested by Rechtschaffen and Kales (1968), at the first epoch of stage 2, even though there might be a return to stage 1 or to wake within the following five minutes. Thus, it is believed that by readjusting the SOL monitor to a lower than five minute criterion, we might reduce this discrepancy by a few minutes.

Some recent evidence suggests however that for most individuals, the SOL device will always overestimate the polysomnographically defined onset of stage 2 by at least a few minutes. Ogilvie and Wilkinson (1988), in an attempt to clarify the sleep/wake definition, presented faint tones at intervals averaging 16 seconds throughout a night's sleep. The subjects, on hearing the tones, were instructed to press a palm-mounted button to switch them off. In addition to a significant decrease in behavioral responses during stage 3, 4 and REM, the authors added that "Even in what Johnson (1973) has regarded as the undoubted sign of sleep, stage 2, behavioral responses are still present, particularly during the first 5 min. of stage 2 period" (Ogilvie and Wilkinson, 1988, pp.152). In brief, it would appear that certain individuals are still able to respond to their environment even when entering the first few minutes of stage 2. Thus the behavioral response of pressing a hand-held button, which is required of insomniacs in order to operate the SOL monitor, can also extend several minutes after the onset of stage 2. This explanation could account for part of the difference between stage 2 latency and the SOL device's final reading.

Now that we are aware of this overestimation factor, the SOL device has the potential to become a very useful research and clinical tool in evaluating sleep-onset latency problems at home and in the laboratory. In addition, its continuous use in therapy can also give us practical, yet objective, feedback on the client's progress in the home environment. This last statement is

in agreement with Ogilvie and Wilkinson (1988) when they state that "Adaptation of such techniques (detection of SO (sleep onset) using behavioral measures) may have useful clinical applications" (pp. 153). An additional improvement to the SOL device would be to modify it so that any button pressure during the night would be digitally stored for future computer analysis. This, in return, would give us valuable information not only regarding sleep-onset but also frequent nocturnal arousals and early morning awakenings. Such a device would in fact strongly resemble an apparatus developed by Holborn et al. (1987). The only difference lies in their device's reliance on all-night continuous computer generated tones which act as stimuli the subject has to shut off whenever he/she wakes up. We believe that these tones, in addition to measuring the sleep problems, could inadvertently accentuate sleep disruption. This could be particularly true in the case of insomniacs who may be more sensitive to external stimulation during sleep.

4) The Four-Choice Reaction Time test

Analysis of the data collected with the Four-Choice Reaction Time test indicated strong significant improvements following treatment in all groups formed (i.e., AMT, PR, LO-A, and HI-A). Several avenues can be explored in attempting to interpret these results.

The most obvious option would be to claim that the treatment was effective in restoring some function (possibly vigilance) which in turn was measured by an increased output in performance on the Four-Choice. On the other hand, one could attribute the increase rate of response to a mere practice effect. In fact, nine ten minute trials of the test were performed within a time span of three months. So it is conceivable that repetition alone could account for the observed progress.

Glenville and Wilkinson (1979), in a study repeatedly using the Four-Choice as a

measure of performance, stressed that three adaptation trials of 10 min each were sufficient to control for increased performance due to learning. More recently, Hébert (1988), in an attempt to clarify the role of learning in the Four-Choice R.T. improvements, formed a control group comprising nine normal individuals. The subjects were asked to perform the reaction time test following the same design to which our insomniacs were subjected, with the exclusion of the treatment, sleep recordings in the laboratory and the other psychological tests. Thus each subject was allowed to perform the test twice before the actual testing sessions started. This is in contrast to the three sessions prescribed by Glenville and Wilkinson (1979). Comparison with an experimental group of ten insomniacs indicated that prior to therapy, the control group's number of total responses was significantly higher than that of the insomniacs. After treatment, both groups significantly improved in number of total responses, but the insomniacs continued to remain significantly lower in their rate of responses when compared to the control group.

These results point to a strong learning effect in both groups but do not exclude a possible contribution stemming from the effects of therapy. It is quite conceivable that the improvements in the Four-Choice R.T. test were attributable to both practice and therapy. In the Hébert (1988) study, the control group formed was not really matched with our insomniacs. The group, composed mostly of students, in addition to being few in number, was not matched by age or sex to the older insomniac sample and had not spent the night in the laboratory prior to trials.

To evaluate the proportion of learning following repetitive testing on the Four-Choice R.T. test, a representative control group would have to be formed comprising 20 insomniacs, paired for sex, age and, if possible, severity of sleep disorder, to the sample in this study. This in turn would aid us in answering some of the questions concerning the Four-Choice's effectiveness in measuring performance improvements in insomniacs. Without such a study, the present results can only be interpreted with great caution.

On Improving this Study

This study was not exempt from flaws and many improvements could have helped enrich its quality. The first obvious one pertains to the number of subjects. The ratio of individuals contacted in the general population to actual subjects successfully completing the study was approximately 11 to 1. Were the criteria for inclusion too severe? The criteria for selection were stringent but not more so than any other study in the area. However, what did severely limit our sample was our "no drug use" criterion. Subjects using drugs and unable or unwilling to discontinue their use were automatically excluded from the study. Because of this, many individuals never made it to the personal interview.

The two following suggestions might have possibly corrected this recruitment problem. First, as strongly suggested by Espie et al. (1989) and Espie, Lindsay and Brooks (1988), procedures and protocols might have been devised in such a way as to allow inclusion of drug using insomniacs in the study without, however, compromising methodological control. Nowadays, most insomniacs ending up in hospitals and/or sleep clinics are also regular users of hypnotics. Samples which exclude these subjects are not really representative of the general population. For our final results to be valid, these specific insomniacs should also be studied. However, very specific controls would have to be introduced to control for dosage, length of use, and so on.

Second, the media could have been exploited more adequately. Television interviews, often accompanied with graphic explanations of the experimental procedures in the laboratory, were all at once too revealing, thus scaring away potential subjects. On the other hand, most radio interviewers were constantly attempting to "dramatize" the issues thus again fostering hesitation in the listeners. However, newspaper ads were quite effective in properly

communicating the desired message to the population. They were often to the point and carefully written. In addition, given that newspaper ads could easily be clipped out and kept for later use, this type of advertisement carried the information for extended periods of time. Unfortunately, this approach was used only late in the recruitment phase and the service was in the long run quite expensive.

Looking at the various tests selected for this study, one can only wonder about having chosen the Four-Choice R.T. test. It has enjoyed only limited use in previous studies. In addition, there appear to be some severe restrictions as to the interpretation of its results. As we have pointed out earlier, learning was most probably an intervening factor which subsequently influenced the subjects' final results. This in effect renders us helpless in attempting any worthwhile interpretation. Thus, measurement of vigilance might have been better served by some test other than the Four-Choice R.T. test.

As for the other tests, some of them could have been employed differently, thus maximizing their usefulness. We are referring here to the Beck Depression Inventory and the STAI. These tests were very useful in measuring pre and post treatment depression and anxiety. Nevertheless, their usefulness could have been exploited even more. As mentioned earlier, weekly or bi-weekly administrations of the Beck and the STAI-Trait would have allowed us to monitor therapeutic progress in several stages, instead of the pre-post measures collected in this study. In return, this would have permitted some level of comparison against the daily sleep-onset latencies collected at home, possibly enabling us to pinpoint which one of these two measures was affected first by the therapeutic intervention. In addition to these suggested improvements, daily monitoring of state anxiety at bedtime should have been undertaken. This procedure would have allowed a better understanding of daily variations in SOL and possibly any relation to situational anxiety.

Still another improvement proposed earlier in this thesis pertains to the specific use of

the AMT procedure on cassette with insomniacs. Given that the willful elevation of anxiety prior to sleep-onset might be detrimental to mental relaxation in some insomniacs, AMT would best serve its purpose if practiced during the day. Furthermore, to maximize treatment efficacy, the relaxation component of the technique could be rehearsed, in bed, at the time the client is ready to fall asleep.

Finally, there is one measure whose absence greatly diminishes the scope of this research. The measure we are referring to is body temperature. A recent literature review article by Sewitch (1987) on slow wave sleep deficiency and insomnia argues that it is necessary to observe a regulated, rapid drop in rectal, core-body temperature following sleep-onset in order to achieve sustained slow wave sleep (NREM stage 4). The author, based upon this premise, presents a theory suggesting "that the slow wave sleep deficiency so commonly associated with chronic, primary insomnia (Gaillard, 1976, 1978) is the result of a failure in the thermoregulatory system to show a regulated, rapid decrease in body temperature with sleep onset which persists for the first 1-2 hrs into the sleep period" (Sewitch, 1987, pp. 200).

It would have been of great interest to monitor body temperature in order to verify if effectively, improvements in sleep-onset latency and increased slow wave sleep would have coincided with decreases in body temperature after sleep-onset. The only drawback to measuring body temperature, is that the favored method, rectal temperature monitoring, would have probably been an additional deterrent for insomniacs to participate in the study.

CONCLUSION

This study attempted to demonstrate the superiority of Anxiety Management Training (AMT) in treating sleep-onset insomnia over one of the most successful forms of therapy presently used, progressive relaxation (PR). The postulated superiority of AMT was based on the hypothesis that trait anxiety was at the source of the insomnia, the reduction of which would lead to a reduction of the sleep-onset problem.

Our results showed that AMT was to a certain degree superior to PR in alleviating many of the psychological and physiological correlates of sleep-onset insomnia. However, the reasons for this amelioration are still somewhat unclear. In addition to trait anxiety being one of the principle etiological factors responsible for the maintenance of sleep-onset insomnia, our results demonstrated that a reduction in the overall reported level of depression was closely associated with a significant improvements in SOL, sleep satisfaction and other psychological measures.

From this, a behavioral model was proposed. Its usefulness was to guide future research in understanding the interactional role played by both depression and anxiety in the maintenance of sleep-onset insomnia.

In addition, this research also permitted a thorough assessment of the performance of the SOL device with a larger sample. It was found to be a very practical instrument in measuring SOL at home, even though it tends to slightly overestimate SOL to stage 2 when compared to the polygraph.

Finally, suggestions were given as to how specific psychometric instruments could be used in order to maximize their usefulness in therapy and in research with insomniacs.

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APPENDIX A : CONSENT FORM

Consent Form for the AMT Group

Marcel Viens, Ph.D. candidate, under the supervision of Dr. Joseph De Koninck, Director of the School of Psychology at the University of Ottawa is conducting a study to evaluate a therapy technique aimed at treating sleep onset insomnia. This proven technique is aimed at reducing general tension, which in turn should reduce sleep onset time. The aim of this study is to compare this technique to another one which has also proven successful.

- The Sleep Research Laboratory is located at 424 Montpetit on the campus of the University of Ottawa.
- Electrodes will be installed before each night spent in the laboratory. These electrodes are applied externally to the skin.
- Each subject will have its own private room and his/her comfort will be respected as much as possible (i.e. pillow, room temperature, private bathroom provided).
- A person will be on call in the laboratory if anything is needed during the night. He/she may be contacted via a special intercom system installed in the subject's room.
- It is our opinion, based on prior sleep research, that there is no health danger whatsoever for the participant to this research. Nevertheless, if your past health history suggests that any type of complications could occur because of your participation in this research, the experimenter should be consulted prior to the beginning of the experiment.
- The participant understands that in all seven (7) nights, divided into 3 blocks (1 night, 3 nights, 3 nights), will be spent in the laboratory during the experiment.
- I understand that I will be filmed during the night in order to study my sleep positions.
- I understand that I will be asked to complete some questionnaires about my overall sleep behavior before and following the end of treatment.
- I also understand that even though therapy will take place in an office supplied by the Centre for Psychological Services situated on the University of Ottawa Campus, the Centre or any of its staff are in no way liable for any of the effects or outcomes of the procedures used in this research.

I understand that there may be no direct benefit to me from participating in this study. I may withdraw from this study at any time even after signing this form. Any information that will be collected about me during this study at any time even after signing this form. Any information that will be collected about me during this study will be kept confidential, and any published data will be only in group form.

Participant's Name

Participant's Signature

I have explained the nature of this study to the participant and I believe that he or she has understood it.

Name of Investigator

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Participant's Name

Participant's Signature

I have explained the nature of this study to the participant and I believe that he or she has understood it.

Name of Investigator

APPENDIX B: QUESTIONNAIRES USED

GENERAL INFORMATION QUESTIONNAIRE

Date : _____

Name : _____

Address : _____

Telephone Numbers : (days) _____ (evenings) _____

Age : _____ Date of Birth : _____

Sex : Male () Female ()

Occupation : _____

Marital Status (circle one) : Single Engaged Married

Separated Divorced Widowed

In the past years, to better deal with my sleep problem, I have consulted

(circle the appropriate answers) : My family physician

A psychiatrist

A psychologist

Other (specify) _____

Name: _____ D.O.B. _____ Date: _____

SLEEP BEHAVIOR QUESTIONNAIRE

1. When was the last time you had a good night's sleep?

2. Are you sleeping sufficiently?

Often _____ Sometimes _____ Never _____

3. On average, how many hours per night do you sleep? _____

4. Do you sleep deeply?

Often _____ Sometimes _____ Never _____

5. At bed time, do you usually feel tired?

Often _____ Sometimes _____ Never _____

6. Do you go to bed at the same time every evening?

Often _____ Sometimes _____ Never _____

7. At what time do you usually go to bed each evening?

Before 22 hr ___ Between 22hr and 24hr ___

Between 24hr and 2hr ___ After 2hr ___

8. In general, how much time does it take you to fall asleep after you lie down and turn off the light?

Less than 15 min ___ Between 15 and 30 min ___

Between 30 and 45 min ___ Between 45 and 60 min ___

More than an hour (explain) _____

9. Do you wake up during the night?

Often_____ Sometimes_____ Never_____

If yes, on average, how many times?_____

10. At that time, do you have difficulties falling back to sleep?

Often_____ Sometimes_____ Never_____

11. When you wake up during the night, on average how much time does it take you to fall back to sleep?

Less than 15 min___ Between 15 and 30 min___

Between 30 and 45 min___ Between 45 and 60 min___

More than an hour(explain)_____

12. Do you wake up at the same time each morning?

Often_____ Sometimes_____ Never_____

13. Do you wake up too early in the morning without being able to fall back to sleep after?

Often_____ Sometimes_____ Never_____

14. On average, at what time do you wake up in the morning?

Before 6hr___ Between 6hr and 8hr___

Between 8hr and 10 hr___ After 10hr___

15. On average, how do you feel when you wake up in the morning?

Well awoken and well rested_____

Awaken but not well rested_____

Tired and without any energy_____

Still sleepy and wishing you could go back to sleep_____

Another way(explain)_____

16. Are you satisfied of your performance at work or other daily activities?

Often_____ Sometimes_____ Never_____

17. How frequently do you suffer from insomnia?

Every night___ Almost every night___

Sometimes___ Rarely___

18. Are you taking any sleep medication?

yes___ no___

If yes, with ones?_____

19. Did someone ever point out that you were moving suddenly during your sleep?

20. Do you have any respiratory problems during the night?

21. Have you ever suddenly fallen asleep during the day for a very short period of time?

Often_____ Sometimes_____ Never_____

If yes, at what moment of the day?_____

22. Do you have any medical problems? Which ones?

23. Are you taking any other types of medication than the ones mentioned in question 18? If yes, which ones?

24. Do you regularly drink alcohol?_____

If yes, how much per day?_____

25. Do you drink any coffee, tea, or cola?_____

If yes, how many cups or glasses per day?_____

26. Do you take naps during the day?_____

If yes, how many per day?_____

On average, how long does each one of them last?_____

At what moment(s) during the day?_____

27. Do you exercise regularly?_____

If yes, what kind of exercise is it?_____

How long does it last?_____

At what time of the day do you do them?_____

28. What do you usually do during the evening?

29. Do you live in a noisy environment?_____

If yes, what kind of noise is it?_____

How do you deal with this problem?_____

30. Do you think others sleep better than you do?

Yes, everyone_____

Most of them do_____

Some do_____

Few do_____

31. How many hours of sleep per night do you think other people enjoy?

32. During the day, do you worry about how well you will sleep the next night?

33. What do you do when you cannot sleep?

Bath _____

Relaxation technique _____

Eat _____

Have a drink _____

Have a cigarette _____

Exercise _____

Listen to music _____

Read _____

Other _____

34. What do you do when you cannot fall asleep?

35. What comes to mind when you are lying down in bed, unable to fall asleep?

36. Do you have at this moment any personal, marital, family, sentimental or professional types of problems?(explain)

37 In your own opinion, what causes your sleep disorder?

38. What results are you expecting from the treatment that will be offered to you?

Signature: _____

DAILY SLEEP SURVEY

Day (Date)	SOL Reading	Estimated time to sleep-onset	# of Awakenings	Sleep-Satisfaction				Comments
				Much 1	Some 2	Little 3	No 4	
1.				1	2	3	4	
2.				1	2	3	4	
3.				1	2	3	4	
4.				1	2	3	4	
5.				1	2	3	4	
6.				1	2	3	4	
7.				1	2	3	4	
8.				1	2	3	4	
9.				1	2	3	4	
10.				1	2	3	4	
11.				1	2	3	4	
12.				1	2	3	4	
13.				1	2	3	4	
14.				1	2	3	4	
15.				1	2	3	4	
16.				1	2	3	4	
17.				1	2	3	4	
18.				1	2	3	4	
19.				1	2	3	4	
20.				1	2	3	4	
21.				1	2	3	4	

SELF-EVALUATION QUESTIONNAIRE

STAI Form Y-2

Name _____ Date _____

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you *generally* feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

ALMOST NEVER
SOMETIMES
OFTEN
ALMOST ALWAYS

- | | | | | |
|--|---|---|---|---|
| 21. I feel pleasant | ① | ② | ③ | ④ |
| 22. I feel nervous and restless | ① | ② | ③ | ④ |
| 23. I feel satisfied with myself | ① | ② | ③ | ④ |
| 24. I wish I could be as happy as others seem to be | ① | ② | ③ | ④ |
| 25. I feel like a failure | ① | ② | ③ | ④ |
| 26. I feel rested | ① | ② | ③ | ④ |
| 27. I am "calm, cool, and collected" | ① | ② | ③ | ④ |
| 28. I feel that difficulties are piling up so that I cannot overcome them | ① | ② | ③ | ④ |
| 29. I worry too much over something that really doesn't matter | ① | ② | ③ | ④ |
| 30. I am happy | ① | ② | ③ | ④ |
| 31. I have disturbing thoughts | ① | ② | ③ | ④ |
| 32. I lack self-confidence | ① | ② | ③ | ④ |
| 33. I feel secure | ① | ② | ③ | ④ |
| 34. I make decisions easily | ① | ② | ③ | ④ |
| 35. I feel inadequate | ① | ② | ③ | ④ |
| 36. I am content | ① | ② | ③ | ④ |
| 37. Some unimportant thought runs through my mind and bothers me | ① | ② | ③ | ④ |
| 38. I take disappointments so keenly that I can't put them out of my
mind | ① | ② | ③ | ④ |
| 39. I am a steady person | ① | ② | ③ | ④ |
| 40. I get in a state of tension or turmoil as I think over my recent concerns
and interests | ① | ② | ③ | ④ |

Name _____ Date _____

DIRECTIONS: A number of statements which people have used to describe themselves are given below. Read each statement and then blacken in the appropriate circle to the right of the statement to indicate how you *generally* feel. There are no right or wrong answers. Do not spend too much time on any one statement but give the answer which seems to describe how you generally feel.

ALMOST NEVER
SOMETIMES
OFTEN
ALMOST ALWAYS

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- 22. I feel nervous and restless ① ② ③ ④
- 23. I feel satisfied with myself ① ② ③ ④
- 24. I wish I could be as happy as others seem to be ① ② ③ ④
- 25. I feel like a failure ① ② ③ ④
- 26. I feel rested ① ② ③ ④
- 27. I am "calm, cool, and collected" ① ② ③ ④
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- 40. I get in a state of tension or turmoil as I think over my recent concerns and interests ① ② ③ ④

MMPI™

MINNESOTA MULTIPHASIC™ PERSONALITY INVENTORY

S.R. Hathaway and J.C. McKinley

PROFILE

NAME _____

ADDRESS _____

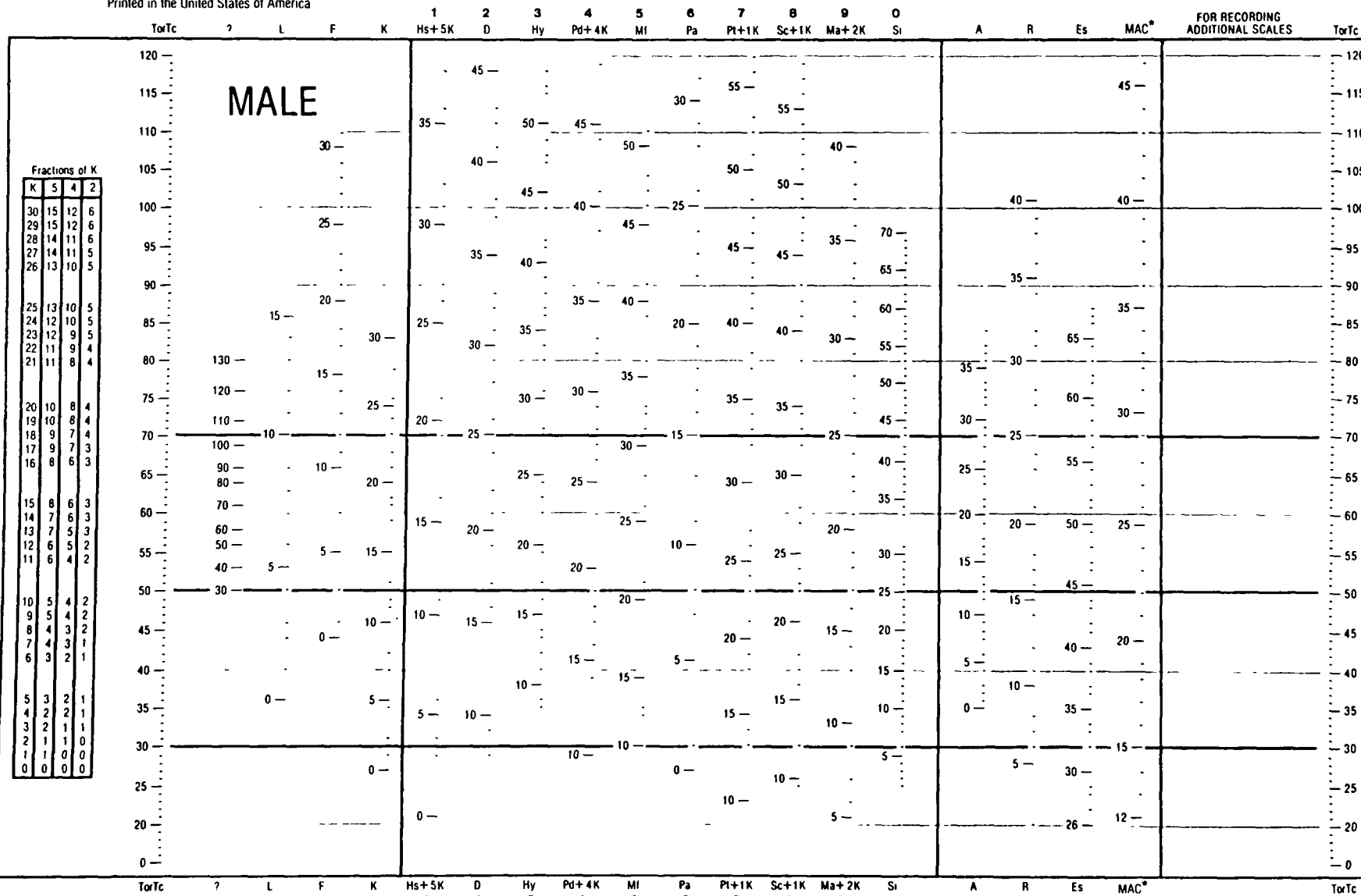
OCCUPATION _____ DATE TESTED / /

EDUCATION _____ AGE _____

MARITAL STATUS _____ REFERRED BY _____

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MMPI Code

Scorer's Initials _____

Raw Score _____

K to be added _____

A refined switch-activated time monitor for the measurement of sleep-onset latency

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(Received 12 August 1987)

Summary—An improved sleep-onset latency (SOL) monitor was developed to overcome the limitations of Franklin's clock device. It was validated against electrophysiological measures of SOL with 4 female students (3 normals and 1 insomniac) who slept in the laboratory for several consecutive nights. Data analysis revealed that the clock-monitored SOL was significantly correlated with latency to Stage 2, that there was no significant difference in mean measures of SOL obtained with the clock monitor and Stage 2 SOL. It is concluded that this simple clock device can constitute a useful tool to obtain reliable measures of SOL in the home environment and is specially well-suited to monitor progress in therapy with sleep-onset insomniacs.

INTRODUCTION

The objective monitoring of sleep-onset latency (SOL) is specially helpful for the assessment of the progress of various treatments of sleep-onset insomnia. However, the most reliable means of recording SOL, polysomnographic recordings in the laboratory, is expensive, cumbersome and not practical in ordinary clinical settings. For this reason, self-reports have been for a long time the only viable alternative. In an attempt to resolve the problem of the unreliability of self-reports for the measurement of SOL in the home environment, Franklin (1981) proposed the use of a switch-activated clock. Essentially a clock, activated at bedtime, is linked to a switch that a *S* holds in one hand and presses with the thumb. When the switch is released, supposedly at sleep onset, it deactivates the clock, thus recording SOL. In a study with 17 insomniacs, Franklin's (1981) SOL estimates obtained with the clock were significantly shorter than self-reports of SOL but were significantly longer than estimates provided by a partner. No comparisons were made with polysomnographic measures of SOL and clock-measured SOL. While going a long way in providing a simple and objective measure of SOL in the home environment, Franklin's device presents the following limitations. Firstly, the procedure requires that a partner set the clock to a predetermined time to avoid any data contamination arising from the *S*'s own awareness of the clock's preset time. Secondly, the clock stops with any deactivation (thumb release) of the switch and thus is subject to premature SOL recordings in cases of accidental or brief releases.

We are presenting here an improved clock monitor system which is based on the same principle but at the same time overcomes these limitations. In addition, it has been evaluated against polysomnographic measures of SOL.

METHOD

Subjects

Our sample comprised 4 female *S*s ranging in age from 19 to 28 yr. Three of them had no sleep disorders, the fourth suffered from sleep-onset insomnia. *S*s did not present any psychological or physiological pathology as assessed by the MMPI, a questionnaire on waking and sleeping habits, and an interview.

Design and procedure

The sleep-onset insomniac *S* slept for two series of 3 consecutive nights in the laboratory. One series was before and the other after a 9-week progressive relaxation treatment programme. The 3 normal *S*s slept for three series of 4 consecutive nights in the laboratory. In all cases, standard polysomnographic measures were recorded (Rechtschaffen and Kales, 1968) and SOL clock monitoring as described below was obtained.

SOL clock monitoring of SOL

The SOL clock consists of a time base counter and display module. As in the Franklin apparatus, the *S* is required to press with the thumb the button of a hand-held switch. The initial contact causes the display to go blank so that the time cannot be viewed by the *S*. Releases of the button within a preset interval (for example we used 5 min) are ignored by the device. Should a release exceed this period, the display is reactivated and shows the SOL time in a coded format. SOL is therefore measured from the moment the button is pressed to the time when the button has been released for a period exceeding 5 min (this preset time may be adjusted to other time intervals). In the morning, the *S* simply writes down the coded number on a special form. Only the experimenter can decode the number displayed and translate it to the proper SOL figure.

Polysomnographic measures of SOL

Standard monitoring of EEG, EOG and EMG were recorded. There is a controversy in the literature regarding the polysomnographic definition of sleep onset. The most widely accepted definition is that of Rechtschaffen and Kales (1968) and is simply the onset of Stage 1. Johnson (1973) has proposed however that the criteria should be the appearance of sleep spindles and K complex activity, indicating the onset of Stage 2. In view of the ongoing controversy (Ogilvie and Wilkinson, 1984), we decided to use the two measures for comparison to our SOL clock generated measures.

*To whom all correspondence should be addressed.

Table 1 Mean scores on the three measures of SOL and the corresponding Pearson correlation coefficients

S	Clock (var 1)	Stage 1 (var 2)	Stage 2 (var 3)	$R = 1-2$	$R = 1-3$	$R = 2-3$
1	36.50	8.32	29.00	0.09	0.83*	0.08
2	20.48	2.44	16.22	0.10	0.89*	0.38
3	18.05	11.00	25.83	0.53	0.73*	0.63
4 (insomniac)	37.80	29.25	35.75	0.95**	0.91*	0.85*

* $P < 0.05$, ** $P < 0.01$

The reliability of the polysomnographic scoring was ascertained by comparing the evaluation of SOLs from two independent judges who scored 6 nights of tracing. Their epoch-by-epoch (30 sec each) agreement of sleep stage scoring was above 80%.

Photographic recording

In order to monitor the body motility of Ss with respect to the switch manipulation, a video recording was obtained throughout the night. It required less than 7 W illumination in the room and was judged unobstructive to the Ss.

RESULTS

Out of the 42 nights of recording, the data for 4 nights were invalid since photographic recordings revealed in these cases that the Ss slept in a position where they applied pressure with their body on the button and thus held it down evidently beyond sleep onset. As explained further below, this drawback of the design of the switch has now been corrected.

Table 1 presents the mean measures of SOL for each S. In order to determine if there were differences between the three measures (polysomnographic SOLs and clock monitor SOL), a repeated measures ANOVA was performed for each S and *post hoc* contrasts were computed when the ANOVA revealed a significant effect. There were significant differences between clock-monitored SOL means and latency to Stage 1 for 2 of the 3 normal Ss ($P < 0.05$ and $P < 0.05$, respectively), whereas there was no significant difference between clock-monitored SOL means and latency to Stage 2 for all 3 Ss. Table 1 also presents the Pearson correlation coefficients between the measures. None of the correlations between latency to Stage 1 and clock-monitored SOL were significant ($r = 0.09$, $r = 0.10$, $r = 0.53$). However, correlations between Stage 2 latency and clock-monitored SOL were significant for all 3 Ss ($r = 0.83$, $P < 0.02$, $r = 0.89$, $P < 0.02$, $r = 0.73$, $P < 0.05$).

Similar results were obtained with the female insomniac's data. A significant difference was found between the means of the SOL clock monitor and the latency to Stage 1 and no significant difference was found between clock-monitored SOL means and latency to Stage 2. However, significant correlations were found between all three variables (Stage 1 and clock monitor, $r = 0.95$, Stage 1 and Stage 2, $r = 0.85$, clock monitor and Stage 2, $r = 0.91$). The distribution of sleep latencies across nights for this S is illustrated in Fig. 1. The dramatic decrease in SOL can be clearly seen from baseline (1-3) to post-relaxation-training nights (4-6). The SOL clock was equally as responsive as the polygraphic measures in measuring variations in SOL prior to and after the therapy programme.

DISCUSSION

These findings suggest that the improved SOL clock device provides a reliable measure of sleep-onset, as defined polysomnographically by stage 2 onset. It appears to be particularly applicable to insomniacs, since Borkovec (1982) reported that when awakened from Stage 1, insomniacs often report that they were not asleep, but when awakened from Stage 2, they do report that they were sleeping.

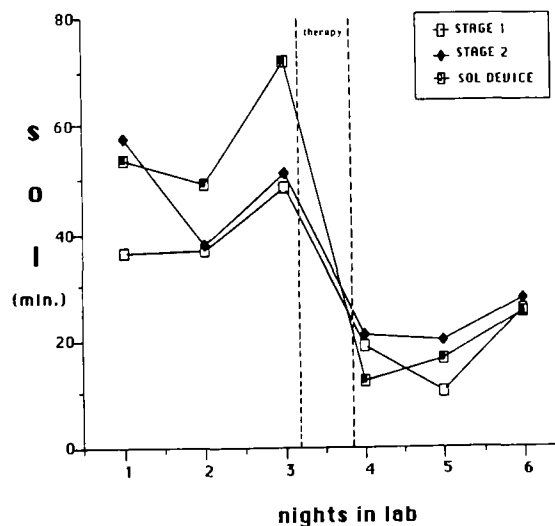


Fig. 1 Distribution of sleep-onset latencies as measured by polysomnographic stage 1, stage 2 and clock monitor. The first three nights are before therapy while the last three nights are post-therapy.

As for the problem encountered with Ss positioning themselves over the switch, we have now devised a protective extension ensuring isolation of the button of the switch, thus preventing activation and deactivation by means other than with the thumb

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