ELSEVIER

Sleep Medicine 1 (2000) 279–288

SLEEP MEDICINE

www.elsevier.com/locate/sleep

Original article

Psychophysiological insomnia: combined effects of pharmacotherapy and relaxation-based treatments

Raymond C. Rosen[a,*], Daniel S. Lewin[b], Lawrence Goldberg[c], Robert L. Woolfolk[d]

[a]*Department of Psychiatry and the Sleep Disorders Center, UMDNJ-Robert Wood Johnson Medical School, 675 Hoes Lane, Piscataway, NJ 08854, USA*

[b]*Department of Psychiatry, Pittsburgh University School of Medicine, Pittsburgh, PA, USA*

[c]*The Carrier Foundation, Skillman, NJ, USA*

[d]*Department of Psychology, Rutgers University, Piscataway, NJ, USA*

Received 8 February 2000; received in revised form 16 May 2000; accepted 17 May 2000

Abstract

Objective: To compare treatment outcomes associated with combined pharmacologic and non-pharmacologic treatments for psychophysiological insomnia.

Background: Treatments for insomnia have included a variety of pharmacotherapy and cognitive–behavioral interventions, although few studies have investigated the combined efficacy of drug and non-drug therapy.

Methods: Forty-one patients with primary insomnia were randomly assigned to one of three treatment groups: (i) estazolam + muscle relaxation, (ii) estazolam + guided imagery, and (iii) estazolam + sleep education. After 4 weeks of active treatment, subjects were withdrawn from medication and followed for an additional 6 months.

Results: Significant improvements were observed in self-report measures of total sleep time, sleep efficiency, and wakefulness after sleep onset in the combined drug and relaxation groups, compared to a significant improvement in total sleep time only in the educational control group. At follow-up, all three groups showed significant improvements across the major sleep measures. Positive changes were also observed in quality of life measures, including mood state and self- ratings of daytime arousal.

Conclusions: These findings provide support for the value of combined pharmacotherapy and relaxation training in the treatment of psychophysiological insomnia.

Keywords: Insomnia; Behavior therapy; Benzodiazepines; Withdrawal insomnia

1. Introduction

Psychophysiological insomnia is a highly prevalent disorder, which affects approximately 10–15% of the adult population [1–4]. A wide variety of somatic and psychological symptoms have been associated with the disorder, including marked disruptions in social, occupational and cognitive functioning [5–8]. In particular, psychophysiological insomnia is associated with increased risk for major depression, chronic absenteeism, and work-related or automobile accidents [6,7]. Additionally, abuse of alcohol and prescription or nonprescription drugs have frequently been reported in patients with a history of chronic sleep difficulties [7,9]. Despite the potentially serious

* Corresponding author. Tel.: +1-732-235-4485; fax: +1-732-235-5818.

E-mail address: rosen@umdnj.edu (R.C. Rosen).

1389-9457/00/$ - see front matter
PII: S1389-9457(00)00042-3

consequences of the disorder, less than 15% of insomnia patients seek specialized treatment services [3].

Current treatment approaches for psychophysiological insomnia include pharmacotherapy, sleep hygiene training, and cognitive–behavioral interventions [10–14]. Pharmacotherapy may be beneficial in the management of transient or short-term sleep difficulties, but is contraindicated for the treatment of chronic insomnia. A variety of benzodiazepine and non-benzodiazepine sedative hypnotics are currently in use [15–17]. Nonpharmacological treatment interventions are also recommended, including sleep restriction, stimulus control, and relaxation-based approaches [12,18]. According to a meta-analysis of nonpharmacological treatment approaches [13], both single and multicomponent interventions for insomnia are significantly more effective than no-treatment controls. Relaxation-based approaches compare favorably with other interventions in decreasing sleep latency, reducing the time spent awake after sleep onset, and improving overall sleep quality. Furthermore, relaxation therapy has been found to be effective in improving sleep maintenance and reducing hypnotic use in medication-dependent elderly patients [14].

Several studies have compared somatic and cognitive relaxation therapies for insomnia [19–21]. Woolfolk and McNulty [21] found that cognitive relaxation and guided imagery were associated with increased treatment efficacy compared to progressive muscle relaxation. Maintenance of treatment gains was also significantly greater in the cognitive relaxation group. Other studies have shown marked improvements in subjective and polygraphic measures of sleep following progressive muscle relaxation training [22,23]. The mechanism of action of relaxation training in insomnia is controversial, although it has been suggested that the primary effect may be via mental distraction or blocking of the cognitive intrusions typically associated with psychophysiological insomnia [13].

Despite widespread use of both pharmacotherapy and behavioral interventions for the treatment of insomnia, relatively few studies have investigated the effectiveness of combined treatment approaches. Results have also been inconsistent to date. In one study [24], a combination of sleep hygiene training and triazolam was compared to sleep hygiene training alone. Although no difference was observed between treatment groups immediately post treatment, results favored the non-drug therapy group at 1-year follow up. Triazolam plus cognitive–behavioral therapy has similarly been compared to triazolam alone [25]. In this study, significant improvements in sleep latency and total sleep time were observed in both treatment groups, although a slight advantage was found for the combined drug and behavioral group at 5-month follow-up. Similar findings were reported by Morin et al. [26] in a study of combined cognitive–behavioral therapy and temazepam, compared to cognitive–behavioral or pharmacotherapy alone. Moreover, none of the studies thus far have evaluated the effects of treatment on mood state or daytime functioning.

To date, no studies have investigated the combined use of relaxation training and short-term hypnotic use, despite clinical considerations favoring this particular treatment combination [14]. In the present study, two methods of relaxation training combined with pharmacotherapy (estazolam) were compared to a combination of pharmacotherapy and sleep education. Given the previous reports of improved outcome with cognitive compared to somatic relaxation methods [13,21], we also attempted to evaluate the combination of two different types of relaxation training with pharmacotherapy. To control for therapist contact time and patient expectations, a pharmacotherapy plus sleep education (attention control) condition was included in the study.

2. Methods

2.1. Subjects

Forty-one adult patients with chronic, psychophysiological insomnia were recruited through community advertisements ($n = 32$) and referrals from a hospital-based sleep disorders center ($n = 9$). Respondents aged 21–65, who met ICSD criteria for psychophysiological insomnia, and who were free of major medical and psychiatric disorders were invited to participate. Subjects were excluded if they had used a sedative-hypnotic medication on a regular basis within 2 weeks of study commencement or had received other forms of treatment for insomnia. Sixteen subjects were excluded from participation due to medical illnesses ($n = 3$), psychiatric disorders

($n = 5$), recent use of sedative hypnotics ($n = 5$), and presence of other sleep disorders ($n = 3$).

Screening measures consisted of sleep and psychological questionnaires, overnight polysomnography when indicated ($n = 6$), wrist actigraphy, and in-person or telephone interviews. Patient exclusions were based on data obtained from screening questionnaires and telephone interviews, as well as polysomnographic data for concomitant sleep disorders. After complete description of the study to the subjects, written informed consent was obtained.

Of the 41 subjects who were randomized for study, 32 or 78% completed the full study protocol. Three subjects were discontinued due to adverse side effects of the study medication (daytime fatigue, headaches), and six subjects were withdrawn due to psychological difficulties or noncompliance with the study protocol. Four of these subjects withdrew voluntarily and two were dropped from the study due to lack of attendance or completion of study records. All withdrawals occurred prior to completion of the study. The study completers were 11 men and 21 women, whose mean ages were 46.1 ± 10.8 and 48.3 ± 10.9 years, respectively. The mean duration of insomnia prior to treatment was 10.98 ± 9.9 years. Patient characteristics at baseline are shown in Table 1.

2.2. Study design

The study was a 4-week, randomized, parallel group design with each subject assigned to one of three treatment conditions: (i) muscle relaxation + estazolam; (ii) guided imagery + estazolam; and (iii) sleep education + estazolam. Following 4 weeks of treatment, subjects were withdrawn from medication and provided with sleep hygiene instructions for an additional 4-week period. A final follow-up assessment was conducted at 6 months following treatment completion. As shown in Fig. 1, patient assessments were conducted at baseline, after 4 weeks of treatment, and at 1-and 6-months follow-up.

2.3. Assessment instruments

Self-report measures of sleep and psychological functioning were administered during baseline, treatment, and follow-up as follows: (i) daily sleep logs were completed for 2 weeks prior to randomization, during treatment and the initial follow-up phase. Variables recorded were sleep latency, total sleep time, wakefulness after sleep onset, number of nocturnal awakenings, sleep efficiency (total sleep time/time in bed × 100), and subjective sleep quality. (ii) The pre-sleep arousal scale (PSAS) [27], sleep efficacy scale (SES) [28], and sleep hygiene practice and knowledge scale (SHPK) [28] were administered at baseline. These measures were used to assess attitudes, beliefs, and behaviors associated with sleep at baseline. (iii) Mood state was assessed by means of the Beck depression inventory (BDI) [29] and Taylor manifest anxiety scale (TMAS) [30]. (iv) A mini-motion logger actigraph (Ambulatory Monitoring Inc., Yardsley, NY) was used to assess total sleep time and arousals in a sub-sample of patients

Table 1
Baseline characteristics by treatment group

Baseline characteristics	Treatment group		
	Muscle relaxation, mean (SD)	Guided imagery, mean (SD)	Education control, mean (SD)
Gender	Women = 6 (67%)	Women = 7 (58%)	Women = 8 (73%)
	Men = 3 (33%)	Men = 5 (41%)	Men = 3 (27%)
Age (years)	48.1 (12.5)	49.0 (11.7)	46.8 (8.3)
Insomnia duration (years)	9.7 (8.4)	15.3 (12.2)	6.4(4.5)
Caffeine (cups/day)	0.6 (0.7)	1.7 (1.5)	1.3 (1.3)
Alcohol (drinks/week)	0.8 (1.0)	1.5 (3.7)	0.5 (0.7)
Beck depression inventory (0–63)	7.9 (5.9)	7.5 (6.1)	7.5 (4.4)
Taylor manifest anxiety scale (0–50)	18.5 (8.5)	19.3 (9.5)	16.3 (9.6)
Sleep efficacy scale (0–45)	22.9 (6.7)	26.1 (4.8)	21.5 (4.9)
Sleep hygiene practice scale (0–133)	26.6 (15.1)	25.7 (9.5)	27.5 (13.7)
Pre-sleep arousal scale (16–80)	32.2 (9.0)	32.4 (9.6)	36.8 (12.2)

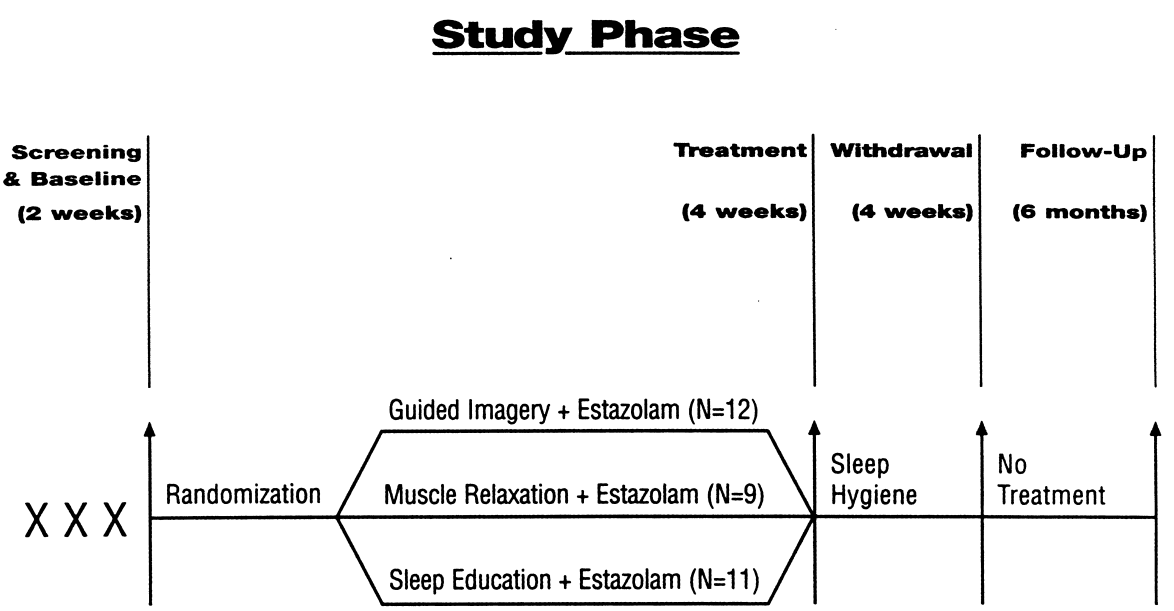

Fig. 1. Study design.

($n = 13$). Five of these subjects were in group 1, four in group 2 and four in group 3. These data are not included in the present report.

2.4. Treatment conditions

Subjects were required to participate in seven one-hour treatment sessions, which were conducted in a group format of 3–5 subjects per group. The time of treatment contact was matched across groups. Following an initial orientation session, subjects received training in one of two relaxation conditions, or were assigned to a sleep education (control) condition. Additionally, all subjects were instructed to take the study medication (estazolam 1 mg hs) approximately 30–60 min prior to bedtime. During the 4th week of treatment, estazolam dosage was halved (0.5 mg hs). No medication was provided after the 5th treatment week. Subjects were instructed to minimize changes in sleep practices or the bedroom environment during the active treatment phase.

All treatment sessions were conducted by the second author (DSL), a doctoral student in clinical psychology at the time of the study. Clinical supervision was provided by the principal investigator (RCR), a board-certified sleep specialist. Medication administration and psychiatric supervision was provided by the third author (LG), a board-certified psychiatrist and medical consultant to the sleep disorders center.

2.5. Non-drug interventions

Following the initial orientation, the active treatment groups received instruction in progressive muscle or imagery-based relaxation training. A detailed description of these procedures is provided elsewhere [21]. Subjects were instructed to engage in two daily 15 min practice sessions, and the time of practice was monitored by means of daily practice logs (guided imagery group mean daily practice time $= 17.3 \pm 7.8$ min; muscle relaxation group mean practice time $= 23.8 \pm 7.7$ min). All subjects in the two relaxation groups received tape-recorded treatment sessions to facilitate home practice.

2.5.1. Deep muscle relaxation

Subjects were informed that progressive muscle relaxation would reduce physical tension and arousal, and thereby facilitate sleep onset. Detailed instructions were provided for tension and relaxation of major muscle groups, and subjects were instructed to attend

closely to the resultant sensations of tension and relaxation. Each training session began with deep breathing exercises and instruction in somatic tension release. Following muscle relaxation training, subjects were instructed to maintain a state of somatic relaxation. No specific imagery or cognitive instructions were used in the presentation of this technique.

2.5.2. Guided imagery relaxation

Subjects were informed that imagery techniques would be used to reduce cognitive intrusions and negative thoughts associated with sleep onset difficulties. During group practice and in-home sessions, subjects were instructed to visualize with their eyes closed some designated objects (light bulb, blackboard, hour glass, kite, candle, bowl of fruit), and to focus all of their attention on these images. Each object was visualized in sequence for 2 min. Subjects were instructed to practice at home by visualizing each object sequentially for approximately 2 min, and then repeating the sequence for an overall duration of 15 min, two times daily. Aside from instructions to take several deep breaths at the start of each practice session, no somatic or muscle relaxation instructions were provided.

2.5.3. Sleep education (control)

Subjects were given no specific expectations, except that the study medication would be of benefit in restoring normal sleep during the period of treatment. Subjects were presented with didactic information about various aspects of sleep physiology and behavior, including biological determinants of sleep, sleep stages and mechanisms, sleep in non-human species, and the pharmacology of sleep. No particular instructions were provided for managing sleep onset or maintenance difficulties, nor were somatic or cognitive relaxation techniques addressed.

2.6. Treatment withdrawal and follow-up phase

During the initial post-treatment session, all subjects were provided with a popular self-help manual ('No more sleepless nights') and brief instructions in sleep hygiene skills. Subjects were invited to attend a termination session following the 4th week of follow-up, at which time treatment issues and sleep hygiene skills were reviewed. During the follow-up phase, subjects were required to maintain nightly sleep logs, and to refrain from the use of sedative-hypnotics. Although perfect compliance in record keeping was achieved, 11 subjects (relaxation = 1, imagery = 5, sleep education = 5) reported occasional use of over-the-counter or prescription hypnotics during the follow-up phase. Six months after conclusion of the treatment phase, all subjects were recontacted for completion of a follow-up questionnaire. This resulted in a 91% overall response rate.

2.7. Statistical analysis

Comparison between treatment groups with respect to baseline sleep log variables such as total sleep time (TST), sleep latency (SL), sleep efficiency (SE), wakefulness after sleep onset (WASO), and number of awakenings (NA) was conducted by means of analysis of variance (ANOVA) procedures. Univariate analyses were performed to assess normality of distribution of each of the major sleep variables. With the exception of baseline WASO, all sleep variables met normality requirements in each of the study phases. Between and within group differences were assessed by means of a two-way, repeated measures ANOVA procedure, following which analysis of covariance (ANCOVA) was performed on total sleep time, sleep efficiency, and sleep latency. Baseline values were employed as a covariate in this analysis to correct for pre-treatment differences. A series of one-tailed, paired *t*-tests were employed to analyze change from baseline to treatment on each of the major sleep variables. Pearson correlation coefficients were calculated to assess the relationship between sleep logs and actigraphy, and between treatment response and baseline predictor variables. Bonferroni adjustments ($P < 0.05$) were used to correct for multiple statistical comparisons. All analyses were performed using SAS version 6.03 (SAS Institute, Inc., Cary, NC) on an IBM desktop computer.

3. Results

3.1. Group comparability at baseline

As shown in Table 1, all treatment groups were well matched at baseline with regard to gender, age, self-rated sleep quality, and mood state. About 2/3 of each

group was female, and the mean age range was 45–50 years. Total sleep time in all three groups was approximately 5.5–6.0 h and sleep efficiencies ranged from 73 to 78%. No significant differences were observed between groups on any sleep variables derived from the nightly logs at baseline.

3.2. Treatment effects

A total of 32 subjects completed the treatment phase of the study. Of the nine non-completers, three were in group 1 (relaxation + pharmacotherapy), two were in group 2 (guided imagery + pharmacotherapy), and four were in group 3 (sleep education + pharmacotherapy). As shown in Table 2, all three groups showed a significant improvement in total sleep time over the course of treatment. The average increase in total sleep time was 65.0 min in the muscle relaxation + pharmacotherapy group, 40.0 min in the guided imagery + pharmacotherapy group, and 34.0 min in the sleep education + pharmacotherapy group. On the sleep efficiency and wakefulness after sleep onset (WASO) variables, only the two relaxation groups showed a significant improvement from baseline to treatment. A non-significant trend towards improvement in sleep latency and reported number of awakenings was observed in all three groups.

3.3. Group by treatment phase effects

A main effect for treatment phase was noted for all groups across all phases of the study on each of the major sleep log variables, total sleep time ($F(2,25) = 16.75$; $P < 0.0001$) sleep efficiency ($F(2,25) = 12.55$; $P < 0.001$) and sleep latency ($F(2,25) = 5.04$; $P < 0.01$). As shown in Figs. 2 and 3 below, total sleep time and sleep efficiency

Table 2
Changes in self-reported sleep across treatment groups[a]

Sleep variables	Treatment group		
	Muscle relaxation, mean (SD)	Guided imagery, mean (SD)	Education control, mean (SD)
Total sleep time (min)			
Baseline	340 (53)	353 (70)	340 (60)
Treatment	401 (33)	391 (55)	370 (53)
Change	+65 (33)*	+40 (32)*	+34 (34)*
Sleep efficiency (%)			
Baseline	74.1 (11)	78.6 (10)	73.9 (80)
Treatment	84.0 (6)	85.2 (6)	80.3 (10)
Change	+9.7 (8)*	+7.4 (6)*	+6.4 (8)
Sleep latency (min)			
Baseline	33 (17)	26 (20)	38 (38)
Treatment	24 (18)	18 (13)	20 (19)
Change	−8 (9)	−8 (9)	−17 (21)
Wakefulness after sleep onset (min)			
Baseline	40 (20)	60 (48)	62 (64)
Treatment	26 (21)	30 (32)	60 (43)
Change	−17 (12)*	−33 (25)*	−5 (38)
Number of awakenings			
Baseline	2.5 (1.6)	2.3 (1.4)	2.0 (0)
Treatment	1.7 (0.8)	2.0 (1.3)	1.8 (1.0)
Change	−0.9 (0.9)	−0.3 (0.8)	−0.2 (1.0)

[a] *One tailed paired *t*-test, bonferroni adjusted for number of comparisons, $P < 0.05$.

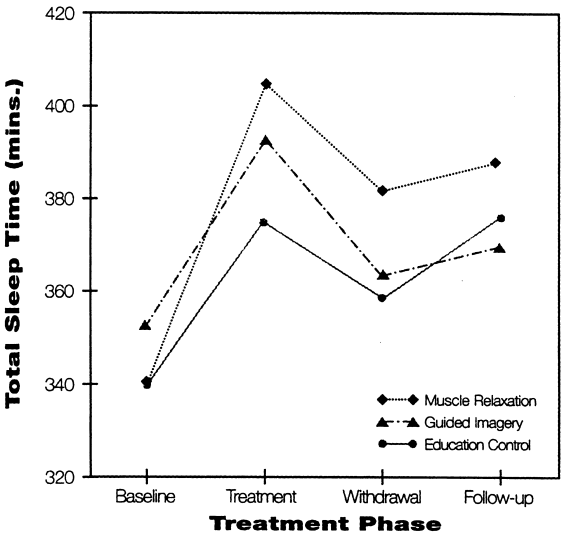

Fig. 2. Total sleep time (min).

increased in all three groups during the treatment phase, decreased during the medication withdrawal period, and increased during follow-up. For the sleep efficiency measure, a significant time by group interaction effect was observed across all study phases ($F(4,56) = 2.5$, $P < 0.05$). As shown in Fig. 3, sleep efficiency declined markedly during treatment withdrawal in both the muscle relaxation and guided

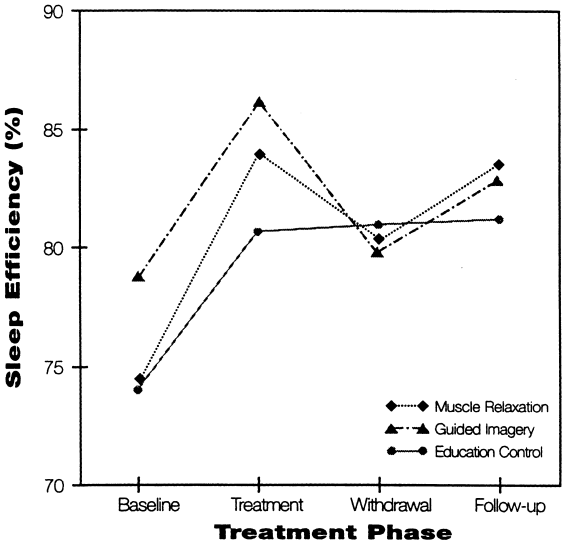

Fig. 3. Sleep efficiency (%).

imagery groups, and was unchanged during this phase in the attention control condition. At follow-up, patients in all three conditions reported higher sleep efficiencies and increased total sleep time compared to baseline (see Figs. 2 and 3). Similar effects were noted for the variables of sleep latency and number of awakenings.

3.4. Questionnaire measures

The effects of treatment on questionnaire measures of sleep quality, daytime sleepiness and mood state are shown in Table 3. Significant changes were observed on the measures of pre-sleep arousal (PSAS) ($F(2,25) = 2.91$; $P < 0.05$) and sleep efficacy (SES) ($F(2,25) = 10.91$; $P < 0.001$) from baseline to follow-up. Similarly, a significant improvement was noted in the BDI scores for all three groups ($F(2,25) = 9.81$; $P < 0.004$). No significant changes were observed on the TMAS or sleepiness rating scale.

Finally, correlational analyses were performed to assess the relationship between age, duration of insomnia, and use of alcohol and caffeine at baseline with changes over the course of treatment. Duration of insomnia was found to be significantly correlated with the decrease in sleep time from the treatment to withdrawal phase ($r = 0.65$, $P < 0.05$; df = 1). Similarly, a near-significant association was found between duration of insomnia and the decrease in sleep efficiency from treatment to withdrawal ($r = 0.44$, $P > 0.05$; df = 1). No other correlations examined approached statistical significance.

4. Discussion

The major goal of this study was to evaluate potential interactive effects of somatic and cognitive relaxation training during pharmacotherapy for chronic insomnia. Although all three treatment groups showed an improvement in total sleep time during the active treatment phase, the two relaxation groups showed additional benefits in sleep efficiency and wakefulness after sleep onset. These findings indicate that both imagery and muscle relaxation training increased the efficacy of short-term, hypnotic therapy for insomnia. Although no clear differences were found for the effects of muscle relaxation compared to guided imagery training plus pharmacotherapy, a slight

Table 3
Questionnaire measures: change from baseline to follow-up

Questionnaire measure	Treatment group			
	Muscle relaxation, mean (SD)	Guided imagery, mean (SD)	Control, mean (SD)	Significance level
Beck depression inventory				
Baseline	7.9 (6)	7.5 (6)	7.5 (4)	$F = 9.81$
Follow-up	5.6 (6)	4.9 (5)	2.5 (2)	$P < 0.004$
Taylor manifest anxiety scale				
Baseline	18.5 (9)	19.3 (10)	16.3 (10)	NS
Follow-up	14.1 (12)	16.8 (9)	13.8 (10)	
Sleepiness rating scale				
Baseline	3.0 (1)	3.8 (1)	3.5 (1)	NS
Follow-up	3.3 (1)	4.1 (1)	3.9 (1)	
Sleep efficacy scale				
Baseline	22.9 (7)	26.1 (5)	21.6 (5)	$F = 10.91$
Follow-up	30.6 (5)	30.7 (4)	29.0 (8)	$P < 0.001$
Pre-sleep arousal scale				
Baseline	32.2 (9)	32.4 (10)	36.8 (12)	$F = 2.91$
Follow-up	25.1 (4)	27.8 (11)	29.6 (10)	$P < 0.04$

trend in favor of the muscle relaxation group was observed. Positive changes in mood, pre-sleep arousal, and self-efficacy were observed in all three treatment conditions.

Surprisingly, both relaxation groups showed a sharp decline in sleep continuity during the drug withdrawal phase, compared to the attention control group. As shown in Fig. 3, this finding was most apparent in the effects on sleep efficiency. Patients in the attention control group maintained treatment gains during this phase of the study, whereas sleep efficiency declined by approximately ten percent in both relaxation groups. This finding contradicted our initial hypothesis that relaxation training during the active treatment phase would lead to better maintenance of treatment gains, and would minimize occurrence of 'rebound insomnia' during the drug withdrawal phase. In accounting for this unexpected finding, it is possible that patients in the two active treatment conditions misattributed improvements in sleep quality to the effects of relaxation training per se, and underestimated the positive effects of the medication. Alternatively, our findings may be explained as due to the effects of state-dependent learning, as relaxation skills acquired during hypnotic use may fail to generalize during the drug withdrawal phase. Other possible explanations of this effect include introduction of the self-help manual during the withdrawal phase, and increased use of medication by five of the subjects in the sleep education group.

The results of this study are also contrary to the earlier findings of Woolfolk and McNulty [21]. These authors reported that guided imagery training resulted in significantly improved sleep efficiency and total sleep time compared to deep muscle relaxation. The lack of differences observed between the two active treatments in the present study may be due to the additive effects of pharmacotherapy treatment, which could have obscured potential differences in the nonpharmacological treatments. Additive effects of combined drug and behavioral treatments for insomnia have been observed in at least two other studies [25,26]. The present findings suggest combining drug and non-drug therapy may obscure or eliminate differences between specific non-drug interventions for insomnia.

Some limitations of the present study are worth noting. In particular, the relatively small sample size and lack of long-term follow up limit the generalizability of the findings. Due to the small sample size, for example, it was not possible to compare the effectiveness of treatment in sub-groups of patients, (e.g. males vs. females). While all subjects met ICSD criteria for psychophysiological insomnia, the groups were heterogeneous with respect to age, duration of insomnia, and types of symptoms exhibited, (e.g. sleep onset vs. maintenance difficulties). Another limitation is the lack of polysomnographic data on the major sleep variables, although other studies have reported concordance between polysomnographic and sleep log outcome measures in clinical trials of insomnia [26]. Finally, the present study was designed to investigate combined effects of drug and non-drug treatments, and further studies are needed to compare the effects of combined treatment with drug or non-drug therapy alone.

Overall, results of the present study indicate that a combined drug and non-drug therapy approach is feasible and effective in improving sleep quality in patients with psychophysiological insomnia. The results not only indicate that the behavioral and drug treatments can be combined, but that there is a somewhat better response during treatment which may persist slightly during longer-term follow-up. Although withdrawal showed decreases in levels of improvement in all conditions, there was no indication for rebound insomnia or even return of symptoms to the baseline values, except for the WASO withdrawal results. Improved sleep was associated with a decrease in pre-sleep arousal, improved self-efficacy, and reduced scores on the BDI in all treatment conditions.

Acknowledgements

This research was supported by GRS Grant #HR-437-24 to R.C. Rosen and L. Goldberg. Medication supplies for the study were provided by Abbott Pharmaceuticals.

References

[1] Rosekind MR. The epidemiology and occurrence of insomnia. J Clin Psychiatry 1992;53:4–6.
[2] Hohagen F, Rink K, Kappler C, Schramm E, et al. Prevalence and treatment of insomnia in general practice: a longitudinal study. Eur Arch Psychiatry Clin Neurosci 1993;242:329–336.
[3] Mellinger GD, Balter MB, Uhlenhuth EH. Insomnia and its treatment: prevalence and correlates. Arch Gen Psychiatry 1985;42:225–232.
[4] Bixler EO, Kales A, Soldatos CR. Sleep disorders encountered in medical practice: a national survey of physicians. Behav Med 1979;6:1–6.
[5] Waters WF, Adams SG, Binks P, Vernado P. Attention, stress and negative emotion in sleep-onset and sleep-maintenance insomnia. Sleep 1993;16:128–136.
[6] Kales JD, Kales A, Bixler EO, Soldatos CR, et al. Biopsychobehavioral correlates of insomnia. Am J Psychiatry 1984;141:1371–1376.
[7] Ford DE, Kamerow DB. Epidemiological study of sleep disturbances and psychiatric disorders. J Am Med Assoc 1989;262:1479–1489.
[8] Pollak CP, Perlick D, Linsner JP, Wenston J, et al. Sleep problems in the community elderly as predictors of death and nursing home placement. J Comm Health 1990;15:123–135.
[9] Morin CM, Gramling SE. Sleep patterns and aging: comparison of older adults with and without insomnia complaints. Psychol Aging 1989;4:290–294.
[10] Gillin JC, Byerley WF. The diagnosis and management of insomnia. N Engl J Med 1990;322:239–248.
[11] Balter MB, Uhlenhuth EH. New epidemiological findings about insomnia and its treatment. J Clin Psychiatry 1992;53:34–39.
[12] Lacks P, Morin CM. Recent advances in the assessment and treatment of insomnia. J Consult Clin Psychol 1992;60:586–594.
[13] Morin CM, Culbert JP, Schwartz SM. Nonpharmacological interventions for insomnia: a meta-analysis of treatment efficacy. Am J Psychiatry 1994;151:1172–1180.
[14] Lichtstein KL, Johnson RS. Relaxation for insomnia and hypnotic medications use in older women. Psychol Aging 1993;8:103–111.
[15] Maczaj M. Pharmacological treatment of insomnia. Drugs 1993;45:44–55.
[16] Vogel G. Clinical uses and advantages of low doses of benzodiazepine hypnotics. J Clin Psychiatry 1992;53:19–22.
[17] National Institutes of Health. Drugs and insomnia: the use of medications to promote sleep. J Am Med Assoc 1984;251:2410–2414.
[18] Morin CM, Kwentus JA. Behavioral and pharmacological treatments for insomnia. Ann Behav Med 1988;10:91–100.
[19] Lacks P, Bertelson AD, Gans L, Kunkel J. The effectiveness of three behavioral treatments for different degrees of sleep-onset insomnia. Behav Ther 1983;14:593–605.
[20] Espie CA, Lindsay WR, Brooks DN, Hood EH, et al. A controlled comparative investigation of psychological treatments for chronic sleep-onset insomnia. Behav Res Ther 1989;27:79–88.
[21] Woolfolk RL, McNulty TF. Relaxation treatment for insomnia: a component analysis. J Consult Clin Psychol 1983;51:495–503.
[22] Borkovec TD, Grayson TD, O'Brien GT, Weerts TC. Relaxation treatment of pseudo insomnia and idiopathic insomnia: an

electroencephalographic evaluation. J Appl Behav Anal 1979;12:37–54.
[23] Borkovec TD, Weerts TC. Effects of progressive relaxation on sleep disturbances: an electroencephalographic evaluation. Psychosom Med 1976;38:173–180.
[24] Hauri PJ, Wisbey J. Can we mix behavioral therapy and hypnotics? Sleep Res 1993;22:207.
[25] Milby JB, Williams V, Hall JN, Khuder S, et al. Effectiveness of combined triazolam-behavioral therapy for primary insomnia. Am J Psychiatry 1993;150:1259–1260.
[26] Morin CM, Colecchi CA, Stone J, Sood RK, et al. Cognitive–behavior therapy and pharmacotherapy for insomnia: update of a placebo-controlled clinical trial. Sleep Res 1995;24:303.
[27] Nicassio PM, Mendlowitz DR, Fussell JJ, Petras L. The phenomenology of the presleep state: the development of the pre-sleep arousal scale. Behav Res Ther 1985;23:263–271.
[28] Lacks P, Rotert M. Knowledge and practice of sleep hygiene techniques in insomniacs and poor sleepers. Behav Res Ther 1986;24:365–368.
[29] Beck AT, Ward CH, Mendelson M, Mock J, et al. An inventory for measuring depression. Arch Gen Psychiatry 1961;4:561–571.
[30] Spielberger CD, Gorsuch RL, Lushene RE. STAI manual for the state-trait anxiety inventory. Palo Alto, California, CA: Consulting Psychologists Press, 1970.