Cognitive Behavioral Therapy for Treatment of Chronic Primary Insomnia

A Randomized Controlled Trial

Jack D. Edinger, PhD
William K. Wohlgemuth, PhD
Rodney A. Radtke, MD
Gail R. Marsh, PhD
Ruth E. Quillian, PhD

Context Use of nonpharmacological behavioral therapy has been suggested for treatment of chronic primary insomnia, but well-blinded, placebo-controlled trials demonstrating effective behavioral therapy for sleep-maintenance insomnia are lacking.

Objective To test the efficacy of a hybrid cognitive behavioral therapy (CBT) compared with both a first-generation behavioral treatment and a placebo therapy for treating primary sleep-maintenance insomnia.

Design and Setting Randomized, double-blind, placebo-controlled clinical trial conducted at a single academic medical center, with recruitment from January 1995 to July 1997.

Patients Seventy-five adults (n=35 women; mean age, 55.3 years) with chronic primary sleep-maintenance insomnia (mean duration of symptoms, 13.6 years).

Interventions Patients were randomly assigned to receive CBT (sleep education, stimulus control, and time-in-bed restrictions; n=25), progressive muscle relaxation training (RT; n=25), or a quasi-desensitization (placebo) treatment (n=25). Outpatient treatment lasted 6 weeks, with follow-up conducted at 6 months.

Main Outcome Measures Objective (polysomnography) and subjective (sleep log) measures of total sleep time, middle and terminal wake time after sleep onset (WASO), and sleep efficiency; questionnaire measures of global insomnia symptoms, sleep-related self-efficacy, and mood.

Results Cognitive behavioral therapy produced larger improvements across the majority of outcome measures than did RT or placebo treatment. For example, sleep logs showed that CBT-treated patients achieved an average 54% reduction in their WASO whereas RT-treated and placebo-treated patients, respectively, achieved only 16% and 12% reductions in this measure. Recipients of CBT also showed a greater normalization of sleep and subjective symptoms than did the other groups with an average sleep time of more than 6 hours, middle WASO of 26.6 minutes, and sleep efficiency of 85.1%. In contrast, RT-treated patients continued to report a middle WASO of 43.3 minutes and sleep efficiency of 78.8%.

Conclusions Our results suggest that CBT represents a viable intervention for primary sleep-maintenance insomnia. This treatment leads to clinically significant sleep improvements within 6 weeks and these improvements appear to endure through 6 months of follow-up.

JAMA. 2001;285:1856-1864 www.jama.com

PERSISTENT PRIMARY INSOMNIA (PPI), a sleep disorder that predicts clinical depression and enhanced health care use, affects up to 5% of the general population and about 20% of those insomnia patients seen clinically.[1-15] Currently, sedative hypnotics or antidepressant drugs remain the most common treatments offered PPI patients.[3,12,16] However, numerous adverse effects encumber traditional hypnotics (eg, benzodiazepines), and evidence supporting the long-term efficacy/safety of antidepressants among nondepressed insomnia patients is currently lacking.[16-20] Moreover, these agents provide only symptomatic relief since they fail to address underlying mechanisms that sustain PPI. Consequently, patients commonly show a full return of their insomnia symptoms on termination of these treatments.[12,13,16-20]

Alternative, behavioral interventions, which target presumed perpetuating mechanisms of patients with PPI, have shown much more durable improvements following treatment. First-generation behavioral therapies, designed to correct sleep-disruptive habits (eg, stimulus control) or reduce bedtime arousal (eg, relaxation training [RT]), have proven very effective for treating sleep onset problems, but their results among the larger PPI subgroup reporting sleep maintenance complaints have been mixed.[21-28] However,

Author Affiliations: VA Medical Center (Dr Edinger) and Duke University Medical Centers (Drs Edinger, Wohlgemuth, Radtke, Marsh, and Quillian), Durham, NC. **Corresponding Author and Reprints:** Jack D. Edinger, PhD, Psychology Service (116B), VA Medical Center, 508 Fulton St, Durham, NC 27705 (e-mail: jack.edinger@duke.edu).

cognitive behavioral therapy (CBT), which combines cognitive therapy with strategies to improve sleep habits and limit time in bed, appears a promising, more universally effective treatment. Early results suggest CBT effectively addresses sleep-onset and maintenance problems, and it produces better long-term outcomes than pharmacotherapy (temazepam) and medication placebo.[29-32] Absent from the literature are reports of controlled trials comparing CBT with behavioral placebo and with first-generation behavioral interventions for treating sleep-maintenance complaints. Herein we report our double-blind, randomized trial conducted to compare CBT with a behavioral placebo therapy (PT) and with RT, for treating primary sleep-maintenance difficulties. We predicted (1) CBT would produce greater short-term and long-term improvements in sleep and related subjective symptoms than would PT or RT; and (2) RT would not outperform PT.

METHODS

Design

A double-blind, placebo-controlled, randomized group design was used. Participants were randomly assigned to treatments (CBT, RT, or PT) and therapists (1 female, 1 male). Enrollees were blinded to hypotheses and the nature of the PT, but they were told they had a 1 in 3 chance of PT assignment. Therapists were blind to hypotheses and were uninformed that 1 of the treatments they administered was a placebo. The protocol was approved by the Duke University Medical Center institutional review board. The first author met individually with volunteers prior to enrollment to obtain written informed consent, to inform them about therapist blinding, and to instruct them not to inform their therapists about the placebo condition. No payment was offered for study participation nor were there charges for the study's evaluations/treatments.

Participants

Recruitment occurred between January 1995 and July 1997 through newspaper advertisements and occasionally, via face-to-face solicitation (eg, sleep clinic patients). Volunteers between the ages of 40 and 80 years were considered for inclusion if they (1) met criteria[33] for PPI; (2) showed a 1-week average wake time after sleep onset (WASO) of 60 minutes or longer (sleep logs); (3) reported insomnia onset after age 10 years; and (4) had insomnia for 6 months or longer. Because poor sleep hygiene is commonly viewed[34-36] as contributory to PPI, we also required enrollees to report 1 or more sleep-disruptive practices (eg, napping, erratic sleep scheduling). Excluded were those who (1) were pregnant; (2) had terminal illnesses or sleep-disruptive medical conditions (eg, angina pectoris); (3) met criteria[37] for an Axis I psychiatric disorder; (4) were habitual substance abusers; (5) would not abstain from sleep aids (hypnotics, antidepressants); (6) required psychotropic medication for a psychiatric condition; (7) had periodic limb movements during sleep associated with more than 10 arousals/h (screening polysomnogram); (8) had symptoms of sleep apnea; (9) met clinical and polysomnogram criteria[38] for sleep state misperception.

Volunteers underwent telephone (newspaper respondents) or brief face-to-face (clinic patients) screening, and those passing this stage completed structured interviews, sleep log monitoring (1 week), a medical examination, thyroid testing, and screening polysomnogram. The interviews were conducted by a clinical psychologist using the Structured Interview for Sleep Disorders[33] and the Structured Interview for *Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition* Psychiatric Disorders, Patient version[37] since these were the most current instruments of their nature available when the study commenced. Medical examinations were conducted by a physician who is board-certified in neurology and sleep medicine. Seventy-five volunteers qualified, underwent pretreatment assessment, and subsequently were randomly assigned to the study's 6 treatment and therapist cells using computer-generated randomized blocks within sex and age strata (ie, <55 years vs ≥55 years). Prestudy power calculations deemed this sample size sufficient to accommodate a 15% drop-out rate yet detect the 49-minute posttreatment WASO (sleep logs) difference between CBT and RT found in our pilot work.[30] FIGURE 1 shows the participant flow whereas TABLE 1 presents descriptive data for the whole sample and each treatment group. Statistical tests (analysis of variance, χ^2) showed no significant demographic differences among these subgroups.

Measures

All participants completed the same screening and outcome measures prior to, during, and after treatment as described below.

Polysomnography. A screening polysomnogram and 2 subsequent polysomnograms for outcome assessment were requested of each enrollee. The first of the latter 2 polysomnograms was conducted 1 to 2 weeks before treatment whereas the second was conducted during the 2 weeks following the end of treatment. All polysomnogram studies were conducted in participants' homes using 8-channel Oxford Medilog 9000 (Oxford Medical Inc, Clearwater, Fla) analogue cassette recorders. The screening polysomnogram montage included electorencephalographic, electromyographic, electrooculographic, nasal/oral air flow, and anterior tibialis monitoring. Airflow and tibialis were excluded during the subsequent polysomnograms before and after treatment. Sleep stages and leg movements were scored using standard criteria[39] and our validated Medilog scoring approach.[40] For polysomnograms before and after treatment, scorers were blind to polysomnogram dates and participants' treatment assignments. Polysomnogram outcome measures included total sleep time, middle WASO (MWASO) defined as the cumulative time awake between sleep onset and the final morning awakening, ter-

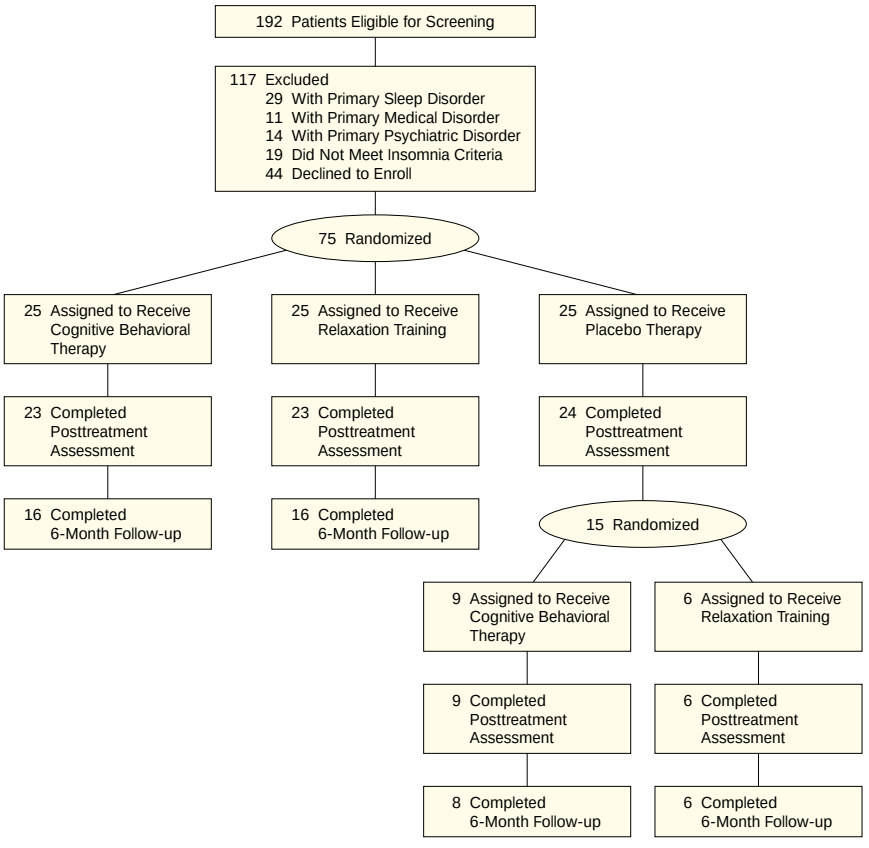
minal WASO (TWASO) defined as the time between final awakening and rising time, and sleep efficiency (sleep efficiency percentage = [total sleep time/total time in bed] × 100%). In these calculations, sleep onset was conservatively defined as the time between lights out and the first 10 minutes of sleep containing no more than 2 minutes of wake time, stage 1, or movement time.[41]

Sleep Logs. Participants completed sleep logs during a 2-week pretreatment baseline, the treatment phase itself, a 2-week posttreatment assessment, and a 2-week follow-up 6 months later. On arising, participants completed sleep log items about the previous night's bedtime, rising time, sleep onset latency, and both MWASO and TWASO. Additionally, sleep logs elicited respondents' ratings of the quality (1 = extremely poor; 5 = excellent) of each night's sleep. Outcome measures derived from logs included the estimates of total sleep time, MWASO and TWASO, sleep efficiency, and sleep quality.

Outcome Questionnaires. Participants completed a 13-item Insomnia Symptom Questionnaire (ISQ),[35] a 9-item Self Efficacy Scale (SES),[42] and the Beck Depression Inventory (BDI)[43] at baseline, midtreatment (ie, end of third treatment week), posttreatment, and the 6-month follow-up time points. We used the ISQ to assess improvements in subjective insomnia symptoms, the SES to detect changes in perceived control over sleep, and the BDI to assess changes in subtle PPI-related mood disturbances.[44-46] Each item on the ISQ is accompanied by a 100-mm horizontal line labeled "not at all" at its left extreme and "frequently" at its right extreme. The SES items include similar 100-mm analog scales labeled "not at all [confident]" at their left extremes and "very [confident]" at their right extremes. For both instruments, respondents drew a vertical line through the point on each item's analog scale to indicate their responses. The distance from the left end of the line to the response line reflects the item's score and the mean score across questionnaire items represents the respondent's overall score for that instrument. Inter-item correlations derived from the baseline administrations showed both the ISQ (Cronbach α = .73) and SES (Cronbach α = .71) had acceptable internal consistency. Research has also shown that both measures reflect subjective treatment-related improvements.[35,42] Since the revised version of the BDI was not available when this project began, we used the original BDI, which has well-established psychometric properties.[47-49]

Therapy Evaluation Questionnaire. Treatment credibility was assessed via responses (Likert ratings) to the 7-item Therapy Evaluation Questionnaire (TEQ).[50] The TEQ's first 5 questions assess perceived logic of and confidence in a treatment, willingness to repeat the treatment, and likelihood the treatment will help others. The final 2 items assess therapist warmth and competence. Participants completed the initial 5 TEQ items after their first treatment session and all 7 items after their last session. Inter-item correlations based on posttreatment responses suggest the TEQ has high internal consistency (Cronbach α = .79).

Figure 1. Participant Flow Diagram



Therapists and Treatments

One male (aged 30 years) and 1 female (aged 29 years) beginning-level clinical psychologists, naive to behavioral insomnia therapy, served as the project's therapists. Before treating study participants, they were required to review the project's treatment manual and audio recordings demonstrating treatments, and then show competence with each treatment via role-play sessions with the first author. Throughout the project, therapists pro-

vided their assigned participants 6 weekly, 30- to 60-minute individual sessions of their respective treatments. The first author jointly supervised the therapists every 8 to 12 weeks throughout the study to ensure their adherence to treatment protocol and to counter any emerging suspicions about the hypotheses or placebo condition. Once all enrollees completed treatment, therapists were debriefed about study hypotheses and placebo treatment.

The CBT recipients were first presented a standardized audio-cassette cognitive therapy module designed to correct misconceptions about sleep requirements and the effects of aging, circadian rhythms, and sleep loss on sleep/wake functioning. They were then given Bootzin[37] stimulus control instructions to (1) establish a standard wake-up time; (2) get out of bed during extended awakenings; (3) avoid sleep-incompatible behaviors in the bed/bedroom; and (4) eliminate daytime napping. Additionally, they each received an initial time in bed prescription equal to their average sleep times (from baseline logs) plus 30 minutes (ie, normal sleep latency and brief awakenings). With the established rising time, this prescription designated the earliest retiring time allowed each night. Sessions 2 through 6 entailed reviewing instructions and adjusting time in bed. The time in bed was increased by 15 minutes each week the patient showed a mean SE of 85% or higher, but reported continued daytime sleepiness. The time in bed was decreased by 15 minutes each week the patient showed a mean sleep efficiency of less than 80%. Otherwise, time in bed was held constant.

The RT assignees received progressive muscle RT,[51] introduced as a method for overcoming the "conditioned arousal" that perpetuates nocturnal wakefulness. Over 6 sessions, RT recipients first learned to alternately tense and relax 16 major skeletal muscle groups, and then to use progressively more efficient tensing-relaxing and passive relaxation exercises. After their second session, they were encouraged to use their relaxation skills to help them return to sleep on awakening at night. Each week, the RT recipients received an instructional audio-cassette tape locked in a tape player equipped with a mechanism used for covert monitoring of their intersession practice. They were instructed to practice the exercise once each day between sessions only with the assistance of this tape and tape player but they remained uninformed about the monitoring of their practice. However, they were discouraged from using the taped instructions in bed because "operating the recorder at night could prolong awakenings."

The patients assigned to PT received a quasi-desensitization treatment,[52] presented as a means of eliminating the "conditioned arousal," which prolongs nocturnal awakenings. Therapists helped each PT recipient develop a chronological 12-item hierarchy of common activities he/she did on awakening at night (eg, opening eyes, clock watching). Therapists also helped them develop 6 imaginal scenes of themselves engaged in neutral activities (eg, reading the newspaper). Each session, PT recipients were taught to pair neutral scenes with items on the 12-item hierarchy so, by the end of the sixth session, all hierarchy items had been practiced with therapist assistance. Each session, the exercise was tape recorded and the patient was given

Table 1. Demographic Characteristics and Therapist Assignment for the 3 Treatment Groups

Characteristic	Cognitive Behavioral Therapy (n = 25)	Relaxation Training (n = 25)	Placebo Therapy (n = 25)
Insomnia, mean (SD), y	13.0 (12.2)	13.2 (12.3)	14.8 (11.5)
Age, mean (SD), y	55.8 (12.1)	54.5 (10.2)	55.7 (9.5)
Sex			
Women	11	11	13
Men	14	14	12
Education, mean (SD), y	16.4 (3.6)	16.3 (3.3)	16.7 (2.7)
Marital status			
Married	18	19	17
Not married	7	6	8
Current hypnotic use			
None	21	16	21
<1 time/wk	0	2	3
1-4 times/wk	4	6	1
>4 times/wk	0	1	0
Therapist assignment			
Total for male therapist	12	13	13
Women	5	5	7
Men	7	8	6
Total <55 y	6	7	7
Women	3	3	3
Men	3	4	4
Total ≥55 y	6	6	6
Women	2	2	4
Men	4	4	2
Total for female therapist	13	12	12
Women	6	6	6
Men	7	6	6
Total <55 y	7	7	7
Women	3	3	3
Men	4	4	4
Total ≥55 y	6	5	5
Women	3	3	3
Men	3	2	2

this tape locked in a player like the device provided to the RT recipients. The patients assigned to PT were told to practice their exercises at home once each day, no less than 2 hours before bedtime, but to avoid using the tape or exercise during sleep periods.

After their posttreatment assessments, CBT and RT recipients were asked to return for their final outcome assessment 6 months later. Given their time already invested in the study, the PT patients were not asked to complete the additional 6-month follow-up before receiving active treatment. Instead, they were debriefed and immediately offered active treatment with their previously assigned therapist. Those who accepted were randomized to 6-week courses of CBT (3 women, 6 men) or RT (4 women, 2 men) but their subsequent data were not considered in any of the statistical comparisons conducted. To maintain their blinding, therapists were told that PT recipients were offered a second, more tested treatment, because their initial treatment was a new therapy that had not yet received sufficient testing to justify its isolated use. They were also told that the PT was a promising treatment deserving of the scrutiny provided by this project.

RESULTS

Treatment Attendance and Follow-up

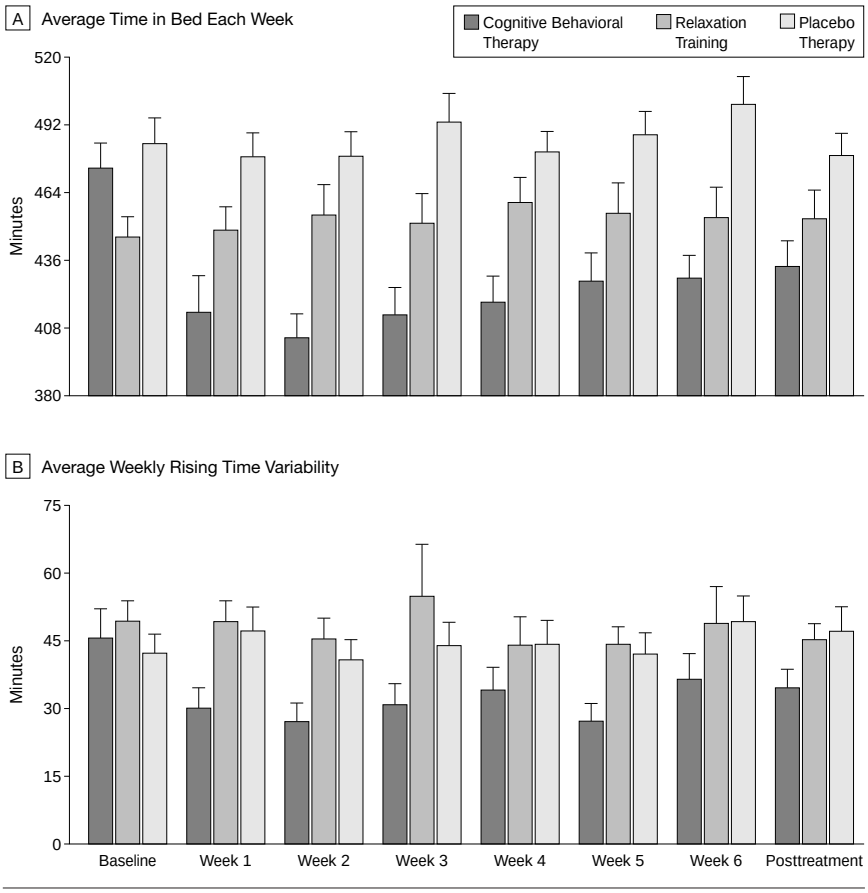
Seventy enrollees completed all scheduled sessions of their initial treatment. One PT assignee completed only 2 sessions and 1 RT recipient completed only 3 sessions; neither completed the midtreatment assessment. Three others (2 women receiving CBT and 1 man receiving RT) completed at least 4 sessions and midtreatment measures before withdrawing. Of those initially assigned to CBT or RT, 32 (16 CBT and 16 RT) completed questionnaires and 29 (14 CBT and 15 RT) completed sleep logs at follow-up. Comparisons showed married participants were more likely to return for follow-up than were unmarried individuals ($P=.04$); otherwise those who accepted and declined follow-up were demographically similar (Table 1). Marital status was not significantly related to treatment outcomes and no significant demographic differences were found between the CBT and RT subgroups who accepted or declined follow-up.

Treatment Credibility and Compliance

The TEQs showed no significant between-group differences (all $P \geq .47$) in regard to how credible and effective the patients initially believed their assigned treatments to be. At the conclusion of treatment, PT recipients reported significantly less willingness to recommend their treatment to others ($F_{2,65}=5.90$; $P=.004$) and significantly less confidence that their treatment would be effective for others ($F_{2,64}=5.63$; $P=.006$) than did CBT and RT recipients. Nonetheless, posttreatment ratings of therapist warmth and competence did not differ across groups ($P=.09$ for both).

The CBT compliance was assessed using sleep log measures of participants' average nightly times in bed and their within-subject SDs of daily rising times during baseline, each in-treatment week, and during the posttreatment assessment. The CBT recipients were expected to show more marked baseline-to-treatment phase decreases in their average time in bed and their rising time variability than the RT and PT recipients. FIGURE 2 shows group averages of these measures during baseline, all treatment weeks, and the posttreatment assessment. Analyses of covariance (ANCOVA) adjusted for baseline levels showed the CBT group spent sig-

Figure 2. Cognitive Behavioral Therapy Compliance Assessment



Data were derived from sleep logs. Error bars represent SE values.

nificantly less time in bed each treatment week and after treatment than did the other 2 groups ($P \leq .001$). Kruskal-Wallis tests with Bonferroni adjustment showed the CBT group had significantly less ($P \leq .02$) rising time variability than the RT or PT groups during treatment weeks 1, 2, and 5, although Figure 2 shows trends in this direction during and after treatment. Thus, these indices reflected reasonable CBT treatment compliance.

We assessed RT and PT compliance via the covert practice-monitoring data. FIGURE 3 shows RT participants had the expected pattern of longer practice sessions initially and briefer practices as treatment progressed. The PT recipients showed the expected 10 to 15 minute daily practice sessions throughout treatment. These data suggested acceptable levels of treatment compliance for these 2 groups. Moreover, the mean (SD) weekly practice times for RT of 83.9 (55.1) minutes and 74.5 (37.4) minutes for PT did not differ significantly ($P = .49$)

Treatment Purity

All therapy sessions were tape recorded and a randomly selected subset (12 CBT, 10 RT, and 7 PT) were selected for scrutiny. Using a checklist designed for this project, a blinded judge reviewed these tapes and identified treatment-specific instructions presented therein. This reviewer observed a mean (SD) of 3.5 (2.3) appropriate instructions during the CBT sessions, 3.8 (1.2) during RT sessions, and 2.7 (2.1) during PT sessions; these means were not statistically different ($F_{2,27} = 0.68$, $P = .52$). Furthermore, all sessions were rated 100% pure; none of the sessions contained elements from more than 1 treatment.

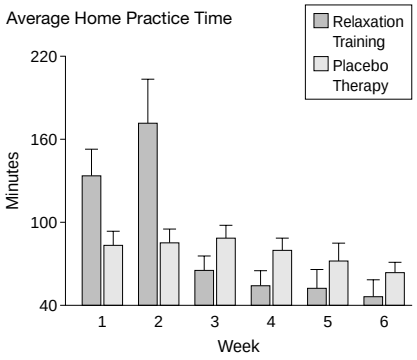
Treatment Comparisons

Analyses of variance showed no significant preintervention differences among the treatment groups on any of the outcome measures. However, visual inspection of these means (TABLE 2) suggested the RT group had slightly more disturbed sleep (logs and polysomnogram) and pathological scores on the outcome questionnaires at baseline than did CBT assignees. Thus, ANCOVA, which adjusted for pretreatment levels of outcome measures, was used for all planned treatment comparisons.

We first compared the 3 treatment groups across all posttreatment outcome measures. An initial set of these analyses showed no significant therapist effects so a 1-factor ANCOVA was used for these comparisons. The ANCOVA was conducted first excluding dropouts, and then with all participants using an intention-to-treat approach. The last in-treatment sleep logs and midtreatment questionnaires served as projected end points for those who withdrew prior to the posttreatment assessment. However, using relevant pretreatment predictors and data from those who actually completed posttreatment assessment, we used recommended regression methods[53] to estimate posttreatment sleep log (1 PT), questionnaire (1 RT and 1 PT), and polysomnography (2 CBT, 1 RT, and 4 PT) data for those lacking/declining these posttreatment measures. Resulting regression models were significant (all $P \leq .02$) and showed an acceptable mean (SD) prediction by R^2 analysis of posttreatment sleep log of 0.58 (0.19), questionnaire of 0.54 (0.16), and polysomnography of 0.61 (0.19).

ANCOVA with and without dropouts showed similar results so only the more conservative intention-to-treat analyses are presented. TABLE 3, which summarizes these comparisons, shows

Figure 3. Relaxation Training and Placebo Therapy Compliance Assessment



Data were obtained from the covert monitoring device used to monitor participants' home practice of assigned relaxation training and placebo therapy compliance exercises. Error bars represent SE values.

Table 2. Baseline Comparisons of Treatment Groups Across All Outcome Measures*

Measure	Adjusted Mean (SE) Cognitive Behavioral Therapy	Relaxation Training	Placebo Therapy	$F_{2,72}$	*P* Value
Total sleep time, min					
Sleep logs	348.1 (61.7)	315.1 (56.6)	347.1 (68.0)	2.28	.11
Polysomnography	361.6 (81.8)	342.2 (59.5)	352.5 (77.1)	0.44	.65
Middle wake time after sleep onset, min					
Sleep logs	55.0 (25.3)	52.8 (32.3)	60.6 (32.7)	0.44	.64
Polysomnography	44.1 (38.6)	45.1 (44.6)	63.0 (50.6)	1.40	.25
Terminal wake time after sleep onset, min					
Sleep logs	43.0 (33.6)	49.5 (45.6)	50.6 (36.5)	0.28	.76
Polysomnography	9.6 (12.4)	16.5 (26.1)	16.2 (28.6)	0.69	.51
Sleep efficiency, %					
Sleep logs	73.7 (11.4)	70.1 (12.9)	71.6 (11.6)	0.39	.68
Polysomnography	80.3 (10.5)	78.3 (10.4)	74.8 (13.4)	1.47	.24
Sleep quality (logs)	2.87 (0.52)	2.83 (0.41)	2.83 (0.52)	0.06	.94
Questionnaires					
Insomnia Symptom Questionnaire	54.4 (12.4)	58.5 (11.2)	51.7 (14.0)	1.85	.16
Self Efficacy Scale	43.5 (14.0)	43.0 (16.4)	51.0 (14.0)	2.28	.11
Beck Depression Inventory	4.9 (2.7)	6.6 (4.5)	4.9 (3.8)	1.79	.17

*Sleep quality ratings vary between 1 (poor) and 5 (excellent); Insomnia Symptom Questionnaire and Self Efficacy Scale scores may range from 0 to 100. High scores on the Insomnia Symptom Questionnaire and low scores on the Self Efficacy Scale are pathological. Beck Depression Inventory scores range from 0 (no depression) to 63 (severe depression).

the CBT group had higher mean polysomnogram and sleep log measures of sleep efficiency, and lower mean sleep log values of MWASO than did the RT and PT groups. They also had significantly higher posttreatment sleep quality ratings than did RT recipients. Most other measures showed significantly more favorable outcomes for CBT recipients than for PT assignees, whereas the RT and PT groups generally did not differ significantly. Only PT comparisons of BDI scores showed RT had more favorable results than CBT.

To compare patients' short-term and longer-term effects in the CBT and RT groups, we used a 2 (CBT vs RT) × 2 (accepted vs declined follow-up) × 2 (time: posttreatment vs follow-up). ANCOVA model in the analyses of data provided by these patients at both posttreatment and follow-up time points. All ANCOVAs were adjusted for baseline values and used actual or estimated posttreatment data as estimates of missing follow-up measures. Both treatments produced statistically similar improvements in sleep time ($P=.90$), BDI ($P=.50$), ISQ ($P=.12$), and SES scores ($P=.83$), and nonsignificant main effects for time ($P\geq.17$) in all analyses suggested posttreatment outcomes persisted during follow-up. FIGURE 4 shows that CBT produced significantly larger improvements in sleep-log WASO and efficiency across both time points than RT. Although both groups were averaging slightly more than 6 hours of sleep per night by the follow-up, the CBT group also showed an average MWASO of 26.6 minutes and an average sleep efficiency of 85.1% at this time point. In contrast, RT-treated patients showed an average MWASO of 43.3 minutes and an average sleep efficiency of 78.8% during the follow-up assessment. The single significant interaction ($F_{1,45}=5.93$; $P=.02$) showed CBT assignees who declined follow-up had higher posttreatment quality ratings than did those who returned, whereas the RT group showed an opposite trend. The sole remaining finding showed those patients who declined follow-up (mean [SE], 4.9 [0.8]) had higher ($F_{1,45}=7.17$; $P=.01$) BDI scores than did those returning for follow-up (mean [SE], 3.1 [0.6]).

We assessed the clinical significance of our results by computing the proportion of each group achieving at least a 50% reduction in pretreatment WASO (MWASO and TWASO) by the end of treatment. Sleep logs showed 64% (16/25) met this criterion for CBT, 12% (3/25) for RT, and 8% (2/25) for PT ($\chi^2=24.2$; $P=.001$). Cognitive behavorial therapy was significantly superior to RT ($P=.001$) and PT ($P=.001$). Using polysomnogram data, 40% (10/25) of the CBT group, 28% (7/25) of the RT group, and 12% (3/25) of the PT group met this criterion ($\chi^2=5.0$; $P=.08$). Additionally, we computed the proportions in each group having posttreatment ISQ scores of 41 or less, a cut point score with 92% sensitivity and 64% specificity for normal sleepers. Eliminating those below this cut-off at study entry, we found 59.1% (13/22) for CBT, 29.2% (7/24) for RT, and 4.8% (1/21) for PT went below this normative ISQ score on study completion ($\chi^2=14.8$; $P=.001$). The CBT group differed ($P=.001$) from PT by this criterion whereas the RT group did not ($P>.05$).

Table 3. Adjusted Results for Posttreatment Comparisons*

Measure	Adjusted Baseline Mean*	Adjusted Mean (SE)†			$F_{2,71}$	*P* Value	Post-hoc Tests‡
		Cognitive Behavioral Therapy (CBT)	Relaxation Training (RT)	Placebo Therapy (PT)			
Total sleep time, min							
Sleep logs	336.8	360.0 (8.4)	362.0 (8.6)	361.0 (8.4)	0.01	.99	NA
Polysomnography	352.1	372.4 (10.6)	337.9 (10.6)	334.0 (10.6)	3.96	.02	CBT > PT
Middle wake time after sleep onset, min							
Sleep logs	56.2	28.1 (4.2)	44.4 (4.2)	47.1 (4.2)	6.06	.004	CBT < RT and PT
Polysomnography	50.8	30.1 (8.9)	50.6 (8.9)	66.4 (9.0)	4.12	.02	CBT < PT
Terminal wake time after sleep onset, min							
Sleep logs	47.7	21.1 (6.4)	36.2 (6.3)	47.0 (6.3)	4.19	.02	CBT < PT
Polysomnography	14.1	4.2 (2.0)	10.2 (2.0)	12.4 (2.0)	4.39	.02	CBT < PT
Sleep efficiency, %							
Sleep logs	72.0	84.3 (1.7)	78.1 (1.6)	76.2 (1.6)	6.64	.002	CBT > RT and PT
Polysomnography	77.8	85.5 (1.9)	78.1 (1.9)	75.7 (2.0)	6.80	.002	CBT > RT and PT
Sleep quality (logs)	2.8	3.4 (0.1)	2.9 (0.1)	3.1 (0.1)	4.00	.02	CBT > RT
Questionnaires							
Insomnia Symptom Questionnaire	54.9	41.9 (2.5)	47.6 (2.6)	52.9 (2.6)	4.73	.01	CBT < PT
Self Efficacy Scale	45.8	62.8 (2.8)	60.6 (2.8)	52.9 (2.6)	3.35	.04	CBT > PT
Beck Depression Inventory	5.5	4.0 (0.5)	2.9 (0.5)	4.8 (0.5)	3.66	.03	RT < PT

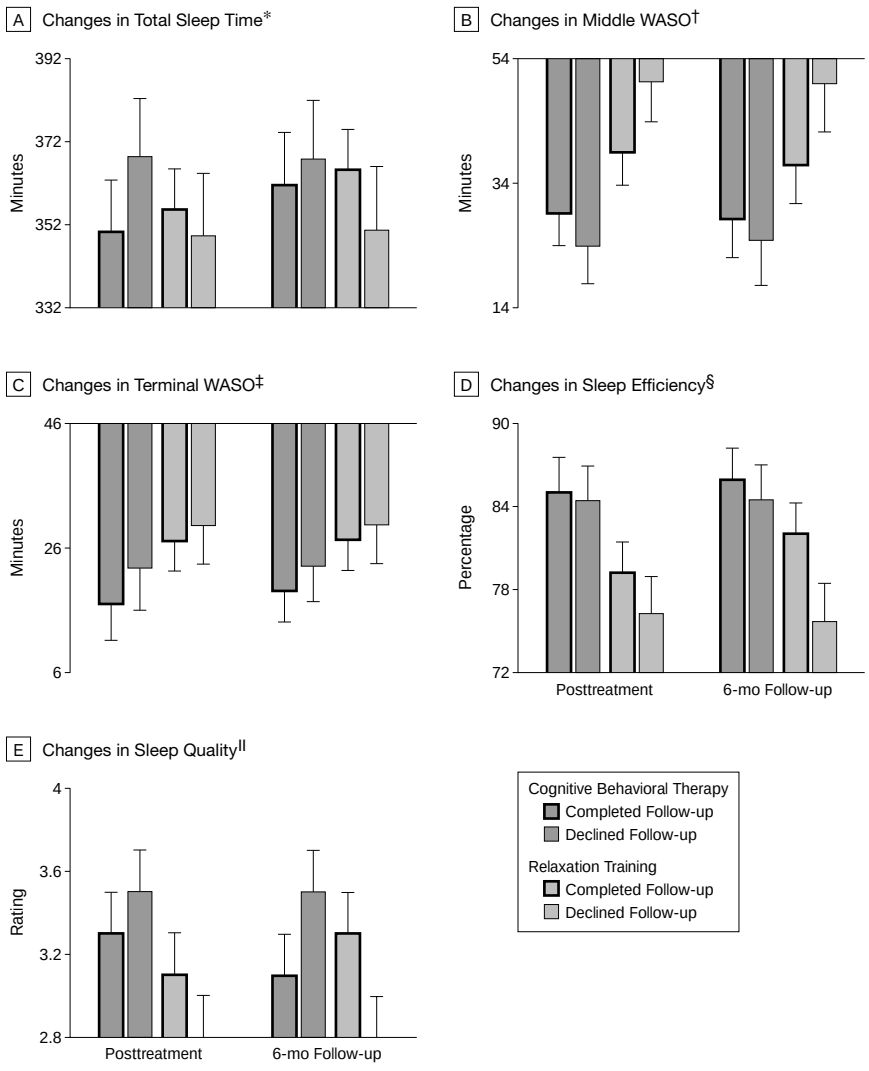
*NA indicates no post-hoc tests were performed.
†Analysis of covariance was used.
‡Significant differences found in Bonferroni-corrected paired comparisons.

COMMENT

Cognitive behavioral therapy produced the largest effects on measures of sleep fragmentation. On average, CBT recipients reported a 54% reduction in WASO by the end of treatment whereas the patients receiving RT and PT, respectively, reported only 16% and 12% reductions in this key measure. Although polysomnography suggested CBT produced somewhat more modest WASO improvements, those patients receiving RT and PT, on average, showed virtually no changes in their polysomnographic WASO measures. Likewise both sleep logs and polysomnography showed that CBT produced substantially greater short-term sleep efficiency improvements than did the other treatments. Furthermore, sleep log estimates of these 2 parameters as well as quality ratings favored CBT over RT across the 6-month follow-up period. In contrast, the effects of CBT on subjective sleep time (sleep logs) were more modest and not significantly different from the effects of other treatments. Polysomnography reflected even smaller sleep time improvements for CBT recipients, but the other 2 groups failed to show objective sleep time increases. Hence, only the CBT group showed both subjective and objective sleep time increases through treatment. In a broader context, the group receiving CBT, had increased sleep time, which suggests a moderate effect size that is similar to the more efficacious behavioral treatments but smaller than that typically reported for short-term pharmacotherapy.[26,27,54] Nonetheless, the modest effect of CBT on sleep time appears enduring through follow-up whereas pharmacotherapy's long-term effect on sleep time has yet to be documented.[54]

The clinical significance of symptom changes is reflected by our improvement criteria as well as by the average performances of participants in each treatment group. Almost two thirds of the CBT group reduced their initial sleep log WASO by 50% or more but only 12% of RT recipients and 8% of PT recipients achieved similar results. Polysomnographic data, though less impressive, showed a similar trend. On ending their participation, the average CBT recipient reported (sleep logs) a MWASO of less than 30 minutes, a level regarded as normal.[25,35] Neither of the other treatment groups reached normative levels for this measure. Furthermore, assuming a pathological pretreatment mean sleep time of about 5.5 hours, the average CBT-treated participant could expect to achieve a mean subjective sleep time of slightly over 6 hours (Figure 4) which, given what is known about human sleep requirements,[55] appears minimally normative/sufficient. Finally, CBT appeared superior to the other treatments in normalizing ISQ scores which reflect perceived sleep/wake functioning.

Figure 4. Cognitive Behavioral Therapy vs Relaxation Training After Treatment and at Follow-up: Sleep Log Data



Analysis of covariance was used for adjusted means (SEs). The x-axis in each part represents the adjusted baseline mean. Asterisk indicates a treatment effect of $F_{1,45}=.02$; $P=.90$. Dagger indicates a treatment effect of $F_{1,45}=8.77$; $P=.005$. WASO indicates wake time after sleep onset. Double dagger indicates a treatment effect of $F_{1,45}=4.24$; $P=.05$. Section symbol indicates a treatment effect of $F_{1,45}=9.10$; $P=.004$; the subgroup treatment effect (interaction of treatment multiplied by subgroups, such as completed vs declined follow-up) was $F_{1,45}=5.93$; $P=.02$. Data plotted are from the 29 (14 cognitive behavorial therapy and 15 relaxation training) who completed and the 21 (11 cognitive behavorial therapy and 10 relaxation training) who did not complete sleep logs at follow-up.

Admittedly this trial would have benefited by a larger study sample and more extensive use of objective outcome measures. Also, although our enrollees reported no hypnotic use during the trial, random urine screens to rule out occult benzodiazepine use may have been useful. Finally, our highly selected sample may limit generalization of our findings. Nonetheless, our results deserve serious consideration since a majority of chronic insomnia patients present sleep maintenance complaints, yet disproportionate numbers of pharmacologic and nonpharmacologic trials have targeted sleep-onset insomnia. Furthermore, insomnia remains undertreated and behavioral interventions are underused.[21,22] Given our results, CBT may have a deserving and important niche in the clinical management of PPI patients with sleep maintenance difficulties

Author Contributions: *Study concept and design:* Edinger, Marsh.
Acquisition of data: Wohlgemuth, Radtke, Marsh, Quillian.
Analysis and interpretation of data: Edinger, Wohlgemuth.
Drafting of the manuscript: Edinger.
Critical revision of the manuscript for important intellectual content: Edinger, Wohlgemuth, Radtke, Marsh, Quillian.
Statistical expertise: Edinger, Wohlgemuth.
Obtained funding: Edinger.
Administrative, technical, or material support: Edinger, Wohlgemuth, Radtke, Marsh, Quillian.
Study supervision: Edinger, Radtke, Quillian.
Funding/Support: This research was funded by grant R01-MH48187 from the National Institute of Mental Health.

REFERENCES

1. Mellinger GD, Balter MB, Uhlenhuth EH. Insomnia and its treatment: prevalence and correlates. *Arch Gen Psychiatry.* 1985;42:225-232.
2. Ford DE, Kamerow DB. Epidemiologic study of sleep disturbances and psychiatric disorders: an opportunity for prevention? *JAMA.* 1989;262:1479-1484.
3. Gallup Organization. *Sleep in America.* Princeton, NJ: Gallup Organization; 1991.
4. Gallup Organization. *Sleep in America.* Princeton, NJ: Gallup Organization; 1995.
5. Weyerer S, Dilling H. Prevalence and treatment of insomnia in the community: result from upper Bavarian field study. *Sleep.* 1991;14:392-398.
6. Ohayon MM. Prevalence of *DSM-IV* diagnostic criteria of insomnia: distinguishing insomnia related to mental disorders from sleep disorders. *J Psychiatr Res.* 1997;31:333-346.
7. Simon GE, VonKorff M. Prevalence, burden, and treatment of insomnia in primary care. *Am J Psychiatry.* 1997;154:1417-1423.
8. Chang PP, Ford DE, Mead LA, Cooper-Patrick L, Klag MJ. Insomnia in young men and subsequent depression: the Johns Hopkins Precursors Study. *Am J Epidemiol.* 1997;146:105-114.
9. Livingston G, Blizard B, Mann A. Does sleep disturbance predict depression in elderly people? a study in inner London. *Br J Gen Pract.* 1993;43:445-448.
10. Vollrath M, Wicki W, Angst J. The Zurich study, VIII: insomnia: association with depression, anxiety, somatic syndromes, and course of insomnia. *Eur Arch Psychiatry Clin Neurosci.* 1989;239:113-124.
11. Foley DJ, Monjan A, Simonsick EM, Wallace RB, Blazer DG. Incidence and remission of insomnia among elderly adults: an epidemiologic study of 6,800 persons over three years. *Sleep.* 1999;22(suppl 2):S366-S372.
12. Institute of Medicine. *Sleeping Pills, Insomnia, and Medical Practice.* Washington, DC: National Academy of Sciences; 1979.
13. National Commission on Sleep Disorders Research. *Wake up America: A National Sleep Alert: Volume 2.* Washington, DC: National Commission on Sleep Disorders Research; 1994.
14. Stoller MK. Economic effects of insomnia. *Clin Ther.* 1994;16:873-897.
15. Weissman MM, Greenwald S, Nino-Murcia G, Dement WC. The morbidity of insomnia uncomplicated by psychiatric disorders. *Gen Hosp Psychiatry.* 1997; 19:245-250.
16. Walsh JK, Schweitzer PK. Ten-year trends in the pharmacologic treatment of insomnia. *Sleep.* 1999; 22:371-375.
17. Greenblatt DJ. Pharmacology of benzodiazepine hypnotics. *J Clin Psychiatry.* 1992;53(suppl):7-13.
18. Kales A, Bixler EO, Tan TL, Scharf MB, Kales JD. Chronic hypnotic use: ineffectiveness, drug withdrawal insomnia and hypnotic drug dependence. *JAMA.* 1974;227:513.
19. Johnson LC, Chernik DA. Sedative hypnotics and human performance. *Psychopharmacology (Berl).* 1982;76:101-113.
20. Ray WA, Griffin MR, Schaffner W, Baugh DK, Melton J. Psychotropic drug use and the role of hip fracture. *N Engl J Med.* 1987;316:363-369.
21. Chesson AL Jr, Anderson WM, Littner M, et al. Practice parameters for the nonpharmacologic treatment of chronic insomnia. *Sleep.* 1999;22:1128-1133.
22. Morin CM, Hauri PJ, Espie CA, Spielman AJ, Buysse DJ, Bootzin RR. Nonpharmacologic treatment of chronic insomnia. *Sleep.* 1999;22:1134-1156.
23. Morin CM, Culbert JP, Schwartz SM. Nonpharmacological interventions for insomnia: a meta-analysis of treatment efficacy. *Am J Psychiatry.* 1994; 151:1172-1180.
24. Murtagh DR, Greenwood KM. Identifying effective psychological treatments for insomnia: a meta-analysis. *J Consulting Clin Psychol.* 1995;63:79-89.
25. Lacks P, Bertelson AD, Sugerman J, Kunkel J. The treatment of sleep-maintenance insomnia with stimulus control techniques. *Behaviour Res Ther.* 1983;21: 291-295.
26. Morin CM, Azrin NH. Stimulus control and imagery training in treating sleep-maintenance insomnia. *J Consult Clin Psychol.* 1987;55:260-262.
27. Davies R, Lacks P, Storandt M, Bertelson AD. Counter-control treatment of sleep-maintenance insomnia in relation to age. *Psychol Aging.* 1986;1:233-238.
28. Thorensen CE, Coates TJ, Kirmil-Gray K, Rosekind MR. Behavioral self-management in treating sleep-maintenance insomnia. *J Behav Med.* 1981;4:41-52.
29. Hoelscher TJ, Edinger JD. Treatment of sleep-maintenance insomnia in older adults: sleep period reduction, sleep education and modified stimulus control. *Psychol Aging.* 1988;3:258-263.
30. Edinger JD, Hoelscher TJ, Marsh GR, Ionescu-Pioggia M, Lipper S. A cognitive-behavioral therapy for sleep-maintenance insomnia in older adults. *Psychol Aging.* 1992;7:282-289.
31. Morin CM, Kowatch RA, Barry T, Walton E. Cognitive-behavior therapy for late-life insomnia. *J Consult Clin Psychol.* 1993;61:137-147.
32. Morin CM, Colecchi C, Stone J, Sood R, Brink D. Behavioral and pharmacological therapies for late-life insomnia. *JAMA.* 1999;281:991-999.
33. Schramm E, Hohagen F, Grasshoff MA, et al. Test-retest reliability and validity of a structured interview for sleep disorders according to *DSM-III-R*. *Am J Psychiatry.* 1993;150:867-872.
34. Bootzin RR. Effects of self-control procedures for insomnia. In: Stuart RB, ed. *Behavioral Self-Management.* New York, NY: Brunner/Mazel Inc; 1977: 176-195.
35. Spielman AJ, Saskin P, Thorpy MJ. Treatment of chronic insomnia by restriction of time in bed. *Sleep.* 1987;10:45-55.
36. Hauri P. Primary insomnia. In: Kryger MH, Roth T, Dement WC, eds. *Principles and Practice of Sleep Medicine.* Philadelphia, Pa: WB Saunders Co; 1994: 494-499.
37. *Structured Clinical Interview for DSM-III-R (SCID).* Washington, DC: American Psychiatric Press; 1990.
38. Diagnostic Classification Steering Committee. *ICSD-International Classification of Sleep Disorders: Diagnostic and Coding Manual.* Rochester, Minn: American Sleep Disorders Association; 1990.
39. Rechtshaffen A, Kales A. *A Manual of Standardized Terminology, Techniques, and Scoring Systems of Sleep Stages of Human Subjects.* Los Angeles: UCLA Brain Information Service/Brain Research Institute; 1968.
40. Hoelscher TJ, McCall WV, Powell J, Marsh GR, Erwin CW. Two methods for scoring sleep with the Oxford Medilog 9000: comparison to conventional paper scoring. *Sleep.* 1989;12:133-139.
41. Carskadon MA, Rechtshaffen A. Monitoring and staging human sleep. In: Kryger MH, Roth T, Dement WC, eds. *Principles and Practice of Sleep Medicine.* Philadelphia, Pa: WB Saunders Co; 1994:943-960.
42. Lacks P. *Behavioral Treatment for Persistent Insomnia.* New York, NY: Pergamon Press; 1987.
43. Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. *Arch Gen Psychiatry.* 1961;4:561-571.
44. Coursey RD. Personality measures and evoked responses in chronic insomniacs. *J Abnorm Psychol.* 1975; 84:239-249.
45. Kales A, Caldwell AB, Preston TA, Healey S, Kales JD. Personality patterns in insomnia. *Arch Gen Psychiatry.* 1976;33:1128-1134.
46. Edinger JD, Stout AL, Hoelscher TJ. Cluster analysis of insomniacs' MMPI profiles: relation of subtypes to sleep history and treatment outcome. *Psychosom Med.* 1988;50:77-87.
47. Callahan LF, Kaplan MR, Pincus R. Response patterns of rheumatoid arthritis patients on four widely used depression questionnaires. *Arthritis Rheum.* 1988; 31(suppl 4):S76.
48. Rehm LP. Assessment of depression. In: Hersen M, Bellack AS, eds. *Behavioral Assessment: A Practical Handbook.* New York, NY: Pergamon; 1976:233-259.
49. Beck AT, Steer RA, Garbin MG. Psychometric properties of the Beck Depression Inventory: twenty-five years of evaluation. *Clin Psychol Rev.* 1988;8:77.
50. Borkovec T, Nau SD. Credibility of analogue therapy rationales. *J Behav Ther Exp Psychiatry.* 1972; 3:247-260.
51. Bernstein D, Borkovec TD. *Progressive Relaxation Training.* Champaign, Ill: Research Press; 1973.
52. Steinmark SW, Borkovec TD. Active and placebo treatment effects on moderate insomnia under counter demand and positive demand instructions. *J Abnorm Psychol.* 1974;83:157-163.
53. Goldberg SC. Persistent flaws in the design and analysis of psychopharmacology research. In: Meltzer HY, ed. *Psychopharmacology: The Third Generation of Progress.* New York, NY: Raven Press; 1987: 1005-1012.
54. Nowell PD, Mazumdar S, Buysse DJ, Dew MA, Reynolds CF III, Kupfer DJ. Benzodiazepines and zolpidem for chronic insomnia: a meta-analysis of treatment efficacy. *JAMA.* 1997;278:2170-2177.
55. Bonnet MH. Sleep deprivation. In: Kryger MH, Roth T, Dement WC, eds. *Principles and Practice of Sleep Medicine.* Philadelphia, Pa: WB Saunders Co; 1994:50-67.

