

Behavioral and Pharmacological Therapies for Late-Life Insomnia

A Randomized Controlled Trial

Charles M. Morin, PhD

Cheryl Colecchi, PhD

Jackie Stone, PhD

Rakesh Sood, MD

Douglas Brink, PharmD

SLEEP DISTURBANCE IS A PREVALENT problem in late life. Between 12% and 25% of healthy seniors report chronic insomnia, and these estimates are even higher among older adults with coexisting medical or psychiatric illness.¹⁻⁴ In addition to normal age-related changes in the physiological aspects of sleep, the increased incidence of health problems and medication use, combined with lifestyle changes associated with retirement, place older adults at increased risk for disrupted sleep.⁵ Insomnia, the most common sleep disorder, may involve trouble falling asleep, frequent or prolonged nocturnal awakenings, or early morning awakenings with an inability to return to sleep.⁶ Older adults report primarily, although not exclusively, difficulty in maintaining sleep. Although not all sleep changes are pathologic in late life,^{7,8} severe sleep disturbances are associated with daytime fatigue and impaired functioning, reduced quality of life, and increased health care costs.^{9,10} When left untreated, chronic insomnia may increase vulnerability to major depression² and, among older adults with cognitive impairments, may hasten nurs-

See also p 1034 and Patient Page.

Context Insomnia is a prevalent health complaint in older adults. Behavioral and pharmacological treatments have their benefits and limitations, but no placebo-controlled study has compared their separate and combined effects for late-life insomnia.

Objective To evaluate the clinical efficacy of behavioral and pharmacological therapies, singly and combined, for late-life insomnia.

Design and Setting Randomized, placebo-controlled clinical trial, at a single academic medical center. Outpatient treatment lasted 8 weeks with follow-ups conducted at 3, 12, and 24 months.

Subjects Seventy-eight adults (50 women, 28 men; mean age, 65 years) with chronic and primary insomnia.

Interventions Cognitive-behavior therapy (stimulus control, sleep restriction, sleep hygiene, and cognitive therapy) (n = 18), pharmacotherapy (temazepam) (n = 20), or both (n = 20) compared with placebo (n = 20).

Main Outcome Measures Time awake after sleep onset and sleep efficiency as measured by sleep diaries and polysomnography; clinical ratings from subjects, significant others, and clinicians.

Results The 3 active treatments were more effective than placebo at posttreatment assessment; there was a trend for the combined approach to improve sleep more than either of its 2 single components (shorter time awake after sleep onset by sleep diary and polysomnography). For example, the percentage reductions of time awake after sleep onset was highest for the combined condition (63.5%), followed by cognitive-behavior therapy (55%), pharmacotherapy (46.5%), and placebo (16.9%). Subjects treated with behavior therapy sustained their clinical gains at follow-up, whereas those treated with drug therapy alone did not. Long-term outcome of the combined intervention was more variable. Behavioral treatment, singly or combined, was rated by subjects, significant others, and clinicians as more effective than drug therapy alone. Subjects were also more satisfied with the behavioral approach.

Conclusions Behavioral and pharmacological approaches are effective for the short-term management of insomnia in late life; sleep improvements are better sustained over time with behavioral treatment.

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ing home placement.¹¹ Despite its high prevalence and negative impact, less than 15% of individuals with chronic insomnia receive treatment.⁴

Author Affiliations: Université Laval, Sainte-Foy, Québec (Dr Morin); and Medical College of Virginia/Virginia Commonwealth University, Richmond (Drs Morin, Colecchi, Stone, Sood, and Brink).

When treatment is initiated, pharmacotherapy is the most common method for insomnia management.^{3,12,13} Although short-term use of hypnotic medi-

Corresponding Author and Reprints: Charles M. Morin, PhD, Université Laval, École de Psychologie, Pavillon FAS, Sainte-Foy, Québec, Canada G1K 7P4 (e-mail: cmorin@psy.ulaval.ca).

cations may be useful and indicated for acute insomnia,^{14,15} there is little information on long-term efficacy or on whether changes in sleep are sustained when the medication is discontinued. A meta-analysis¹⁶ of 22 placebo-controlled hypnotic trials (n = 1894) concluded that benzodiazepines and zolpidem produced improvements in sleep latency, number of awakenings, total sleep time, and sleep quality. However, median treatment duration was only 1 week and follow-up was often limited to evaluating withdrawal effects 1 or 2 nights after drug discontinuation. Thus, although the acute effects of benzodiazepine receptor agents are well documented, controlled evaluations of hypnotic effects beyond the acute treatment phase are warranted. Also, as older adults consume a disproportionate number of hypnotic medications, often for prolonged periods and despite being at greater risk for residual daytime effects,^{4,15,17,18} additional studies of the short- and long-term effects of hypnotics are particularly needed.

Several nonpharmacological interventions have been shown effective for the clinical management of insomnia. Behavioral treatment methods seek to change poor sleep habits, alter faulty beliefs and attitudes about sleep, and promote better sleep hygiene practices. Two meta-analyses^{19,20} of more than 50 treatment studies (>2000 patients) concluded that behavioral interventions produce improvements of sleep in about 70% to 80% of patients with primary insomnia. With an average treatment duration of 5 hours, implemented over a 4-week period, sleep latency and time awake after sleep onset were reduced to near normative values (ie, <30 minutes). One particular strength of behavioral therapies is that sleep improvements are maintained over time. The efficacy of nondrug interventions has also been documented for late-life insomnia. When older adults are screened for other primary sleep disorders (eg, sleep apnea and periodic limb movements), their treatment response is similar to that of younger adults.²¹⁻²⁵ Despite these promising results, behavioral interventions remain underused in primary care settings.²⁶

Only 3 studies have directly compared the efficacy of behavioral and phar-

macological treatments for insomnia.²⁷⁻²⁹ These preliminary studies have shown that drug treatment (triazolam in all 3) produced faster improvements, while behavioral treatment yielded more durable benefits. Behavioral and pharmacological treatments are not mutually exclusive, and their combined use may prove the most successful approach for persistent insomnia. The present study was designed to evaluate the separate and combined effects of behavioral and pharmacological treatments for insomnia in older adults. The main objective was to evaluate which treatment or combination produces the best short- and long-term outcomes on subjective and objective sleep parameters.

METHODS

Subjects

Prospective subjects were recruited through newspaper advertisements and letters to physicians. Inclusion criteria were (1) age 55 years or older; (2) sleep-onset or maintenance insomnia, defined as sleep-onset latency and/or wake after sleep onset longer than 30 minutes per night at least 3 nights per week; (3) insomnia duration of at least 6 months; and (4) a complaint of at least 1 negative effect during waking hours (eg, fatigue, impaired functioning, mood disturbances) attributed to insomnia. Exclusion criteria were (1) evidence that insomnia was directly related to a medical disorder or adverse effects of medication; (2) presence of sleep apnea (apnea-hypopnea index >15) or periodic limb movements during sleep (myoclonic index with arousal >15); (3) regular use of a hypnotic medication or other psychotropic medication with an inability or unwillingness to discontinue medication; (4) currently in psychotherapy; (5) presence of major depression or other severe psychopathologic conditions based on a brief self-report screening measure (ie, Brief Symptom Inventory³⁰) and the Structured Clinical Interview for *DSM-III-R*³¹; and (6) cognitive impairment as suggested by a score lower than 23 on the Mini-Mental State Examination.³² These selection criteria are consistent with those of the International Classification of Sleep Disorders³³ and the *Diagnostic and Statistical*

*Manual for Mental Disorders*³⁴ for primary and chronic insomnia.

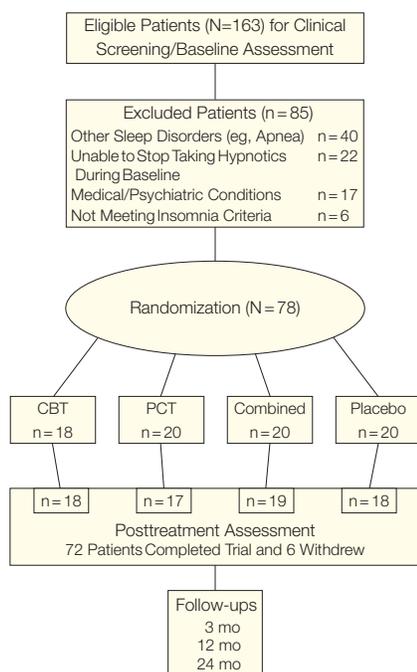
Prospective subjects underwent a multistep screening evaluation, which consisted of (1) telephone screening and (2) a sleep history interview, a psychological assessment, and a medical history taking with physical examination. These evaluations were conducted respectively by a board-certified sleep specialist, a clinical psychologist, and by a physician. Team meetings were regularly held to ascertain that subjects met the study criteria. Forty-eight persons of the 163 who underwent step 2 evaluation were excluded because of psychopathology (n = 9), another suspected sleep disorder (n = 9), lack of interest or inability to avoid taking sleeping medication prior to randomization (n = 21), medical problems (n = 6), or not meeting criteria for insomnia (n = 3). One hundred fifteen individuals underwent the final screening phase of polysomnography. After this final evaluation, another 37 subjects were excluded owing to sleep apnea (n = 23), periodic limb movements during sleep (n = 6), a combination of these 2 conditions (n = 2), no evidence of insomnia (n = 3), or for other medical or psychiatric reasons or lack of interest (n = 3).

The remaining 78 subjects were randomly assigned to either cognitive-behavior therapy (CBT, n = 18), pharmacotherapy (PCT, n = 20), combined CBT and PCT (n = 20), or a placebo condition (n = 20). Of the 78 participants, 50 (64.1%) were women and 28 (35.9%) men, with a mean age of 65 years (SD, 7 years). The average education level was 14.4 years (SD, 2.5 years). All subjects were community-dwelling residents; 70 (89.7%) were white, 7 (9%), black, and 1 (1.3%), Native American; 53 (67.9%) were married, 37 (47.4%), retired. Most (49; 62.8%) of the subjects reported mixed sleep-onset and maintenance insomnia; 22 (28.2%) reported sleep maintenance insomnia and 5 (6.4%) reported sleep-onset insomnia only. The average insomnia duration was 16.8 years (SD, 16.9 years) and 60 (76.9%) had previously used sleeping medication. **TABLE 1** presents illustrative data for demographic and clinical variables. **FIGURE 1** illustrates participant flow in the study protocol. All subjects pro-

Table 1. Demographic and Clinical Characteristics*

Characteristics	CBT (n = 18)	PCT (n = 17)	Combined (n = 19)	Placebo (n = 18)	Dropouts (n = 6)	Total (N = 78)
Age, mean (SD), y	64.4 (7.5)	64.1 (6.4)	65.2 (6.9)	64.9 (7.1)	68.8 (7.1)	65.0 (6.9)
Sex, M/F	5/13	8/9	6/13	6/12	3/3	28/50
Education, mean (SD), y	14.2 (2.3)	14.9 (2.3)	13.5 (2.4)	15.1 (2.9)	14.2 (2.4)	14.4 (2.5)
Occupation, No.						
Employed	8	10	4	5	1	28
Retired	5	4	12	12	4	37
Homemaker	5	3	3	1	1	13
Insomnia duration, mean (SD), y	16.2 (14.8)	15.4 (15.5)	20.0 (23.1)	17.8 (15.5)	14.0 (14.1)	16.8 (16.9)
Use of sleep aid, No.						
Never used	4	3	5	4	2	18
Past use	3	5	5	3	1	17
Current use	11	9	9	11	3	43
Brief Symptom Inventory						
Global severity index, mean (SD)	0.4 (0.3)	0.45 (0.3)	0.31 (0.2)	0.53 (0.4)	0.4 (0.3)	0.42 (0.3)

*CBT indicates cognitive-behavior therapy; PCT, pharmacotherapy.

Figure 1. Participant Flow in the Study

CBT indicates cognitive-behavior therapy; PCT, pharmacotherapy.

provided written informed consent prior to study entry. The study protocol was approved by the institutional review board of the Medical College of Virginia/Virginia Commonwealth University.

Measures

All participants completed the same outcome measures, including daily sleep dia-

ries, polysomnography, and clinical rating scales. Subjects receiving active or placebo medications completed the post-treatment (end-of-treatment) assessment while they were still taking medication (prior to tapering).

Sleep Diaries. Participants kept daily sleep diaries for at least 2 weeks prior to treatment, during the 8 weeks of treatment, and for 2 weeks at each of the follow-ups. Several parameters were monitored in the diaries (eg, bedtime, arising time, sleep-onset latency, wake after sleep onset, medication intake). The main outcome variables were wake after sleep onset (amount of time awake from the initial sleep onset to the last awakening), total wake time (summation of sleep-onset latency, wake after sleep onset, and early morning awakening), total sleep time, and sleep efficiency) ratio of total time spent asleep to the actual time spent in bed and multiplied by 100). The subjects were instructed to complete their diaries every morning at breakfast time and bring them to each session during treatment; they were mailed in for follow-ups. Although sleep diary data do not reflect absolute values obtained from results of electroencephalography, they provide a reliable index of insomnia³⁵ and are used as standard outcome assessment in insomnia research. In addition to allowing for prospective monitoring of sleep in the subject's home environment and over extensive periods of time, sleep diary data reflect an important di-

mension of chronic insomnia, ie, the subjective perception of sleep.

Polysomnography. Subjects underwent 3 consecutive nights of sleep laboratory evaluation both prior to (within 2 weeks) and at the end of treatment. Bedtime and arising time in the sleep laboratory were within half an hour of the subjects' typical sleep schedule. The polysomnographic montage included electroencephalographic, electromyographic, and electro-oculographic monitoring. Sleep stages, respiratory disturbances, and limb movements were scored according to standard criteria³⁶ by an experienced technician who was blind to subjects' condition. Respiration (air flow, tidal volume, and oxygen saturation) and anterior tibialis electromyographic readings were recorded during the first night to detect sleep apnea or periodic limb movements. Outcome measures (wake after sleep onset, total wake time, total sleep time, and sleep efficiency) were based on the average of pretreatment nights 2 and 3 and posttreatment nights 5 and 6. To allow for an adaptation to the laboratory, data from the first night of each assessment phase were not used in computing baseline and posttreatment means.

Clinical Outcome Ratings. The Sleep Impairment Index is a 7-item scale that yields a quantitative index of insomnia severity; it was used as a collateral measure of treatment outcome.⁶ Ratings on a 5-point scale were obtained on the per-

ceived severity of sleep onset, sleep maintenance, and early morning awakening problems; interference with daytime functioning; noticeability of impairment caused by the sleep problem; distress/concern caused by the sleep problem; and satisfaction with current sleep pattern. A composite score was obtained by summing the 7 ratings, and higher scores indicated more severe insomnia (total range, 5-35). Several items were added to this scale at posttreatment assessment and at follow-ups to assess overall degree of improvement, treatment compliance, and satisfaction with the treatment received. Parallel versions of the Sleep Impairment Index were completed by subjects, significant others (eg, spouses), and by a clinician (before and after treatment only). The Sleep Impairment Index has adequate psychometric properties and has been shown sensitive to changes in previous treatment studies of insomnia.^{24,25}

Design and Procedures

A 4 (condition) × 2 (assessment) placebo-controlled randomized design was used, with repeated measures on the second factor. After completing baseline assessments, subjects were randomly assigned to 1 of 4 conditions: CBT (n = 18), PCT (n = 20), combined (n = 20), and placebo (n = 20). Because CBT treatment was provided in a group format, participants were randomly assigned to conditions in clusters of 4 to 6 subjects at one time. All treatments were administered based on a manual⁶ over 8 weekly outpatient therapy sessions. The PCT and placebo conditions were administered in a standard double-blind fashion, and the combined condition was blinded only for the medication component. Due to the nonpharmacological nature of CBT, neither subjects nor therapists were blinded to it.

Treatment Conditions

Cognitive-Behavior Therapy. Subjects receiving CBT attended 8 weekly 90-minute therapy sessions conducted in small groups of 4 to 6 individuals. Treatment consisted of a structured, multifaceted intervention involving behavioral,

cognitive, and educational components that targeted different facets of late-life insomnia.⁶ The behavioral component incorporated sleep restriction therapy³⁷ and stimulus control procedures.³⁸ Sleep restriction consists of curtailing time in bed to the actual sleep time. For example, if an individual reported sleeping an average of 6 hours per night out of 8 spent in bed during baseline assessment, the initial "sleep window" prescribed for the first week of treatment was 6 hours. This sleep window was gradually altered according to the subject's sleep efficiency (ratio of total sleep time to time in bed) based on the sleep diary data from the previous week. Allowable time in bed was increased by 15 to 20 minutes when sleep efficiency exceeded 85%, decreased by the same amount when sleep efficiency was lower than 80%, and kept stable when sleep efficiency fell between 80% and 85%. Implementation of these rules was flexible, and adjustments were made on the basis of subjects' acceptance and willingness to comply. The allotted time in bed was never less than 5 hours per night, regardless of the subject's total sleep time from the week before. The stimulus control procedures were designed to regulate the sleep-wake schedules and to bring subjects to reassociate the bed/bedroom and bedtime stimuli with sleep rather than with the frustration and anxiety associated with lying in bed trying to sleep. These procedures are as follows: (1) go to bed only when sleepy; (2) use the bed and bedroom only for sleep and sex (ie, no reading, TV watching, or worrying in bed or the bedroom during the daytime or at night); (3) get out of bed and go to another room when unable to fall asleep within 15 to 20 minutes; (4) repeat this step as often as necessary, either when trying to fall asleep or to get back to sleep; and (5) arise at the same time every morning regardless of the amount of sleep obtained the previous night. Daytime napping was made optional during the initial sleep restriction phase, as long as it was limited to less than 1 hour and occurred before 3:00 PM.

The cognitive therapy component was designed to alter faulty beliefs and attitudes that often serve to exacerbate in-

somnia.⁶ Examples of faulty beliefs included (1) unrealistic expectations about sleep requirements (eg, the need to sleep 8 hours every night); (2) misattributions or amplifications of the consequences of insomnia (eg, all daytime impairments are due to poor sleep); and (3) erroneous beliefs about strategies to promote sleep (eg, spending excessive time in bed). In addition to formal cognitive therapy, there was an educational component about sleep and aging aimed at distinguishing normative from pathologic sleep changes occurring in late life, and at reviewing sleep hygiene principles about the effects of diet, exercise, caffeine, alcohol, and environmental factors.

Pharmacotherapy. Subjects assigned to the active medication condition were prescribed temazepam (Restoril) to be taken 1 hour before bedtime. The initial dosage was 7.5 mg per night, and the dosage was gradually increased, based on treatment response and adverse effects, up to 30 mg per night, maximum. Subjects were instructed to use sleep medication a minimum of 2 to 3 nights per week, but the medication was made available 7 nights if they chose to use it more frequently. Subjects met once a week with the study physician for a 20-minute consultation on medication management. During these sessions, the physician monitored medication intake over the previous week (pill count) and reviewed therapeutic response and adverse effects. Aside from providing support and encouragement to comply with treatment and discussing general information about sleep changes in late life, no behavioral recommendations were allowed in this treatment condition.

The rationale for selecting temazepam for this study was based on its documented efficacy for older adults³⁹ and minimal daytime residual effects. Temazepam has a moderate to slow absorption rate and an intermediate half-life elimination. It has no active metabolite, produces minimal accumulation with multiple doses, and is well tolerated by elderly persons.^{40,41} For these reasons, temazepam it was the best hypnotic medication for treating sleep-maintenance insomnia in older adults.

Combined CBT and PCT. Subjects in the combined CBT and PCT treatment condition received both the active medication (temazepam) and CBT. They attended 8 weekly individual therapy sessions with a psychiatrist to discuss medication management issues and 8 weekly group therapy sessions with a psychologist to review all cognitive behavioral procedures.

Placebo. Subjects in the placebo condition were treated according to a protocol identical to those receiving the active medication. The placebo medication was provided in identical gelatin capsules, and dosage was adjusted according to perceived therapeutic response and adverse effects. Subjects in this condition were offered an active treatment after completing the 3-month follow-up.

Therapists

The CBT sessions were led by a licensed clinical psychologist or a postdoctoral fellow in clinical psychology. Therapists had previously treated a minimum of 4 clinical subjects using this protocol before their enrollment in this study. A manual⁶ outlining each session was used. A third-year psychiatry resident provided medication treatment for those in conditions involving either an active or a placebo drug. A treatment manual was also used for the PCT sessions. This manual outlined the structure of each consultation, the issues that needed to be covered, and the type of information that was not allowed to be discussed (ie, behavioral recommendations). All therapy sessions were audiotaped and reviewed regularly with the project director to ensure adherence to protocol.

Follow-up

All treated participants were contacted by mail at 3, 12, and 24 months after completing treatment. At each follow-up, they were sent sleep diaries to keep for 2 weeks and were asked to complete the same rating scales and questionnaires administered at baseline and post-treatment assessment.

Data Analysis Plan

Multiple outcome measures were collected as part of this study, but the present report focuses on selected sleep vari-

ables (ie, wake after sleep onset, sleep efficiency, total wake time, and total sleep time) that are most relevant to the problem of sleep-maintenance insomnia in older adults. The main comparisons of interest were to determine whether active treatments were more effective than placebo, whether a combined behavioral and pharmacological approach was more effective than either of its single components alone, and whether there were differential improvement rates over time across treatment modalities.

RESULTS

Preliminary Analyses

Of the 78 subjects enrolled in the study, 71 completed the treatment protocol, 6 dropped out prior to reaching the midtreatment phase (CBT = 0; PCT = 3; combined = 1; placebo = 2), and 1 subject receiving placebo completed more than half of the intervention. Midtreatment data for this latter subject were used for statistical analysis. Of the 6 dropouts, 3 (PCT) discontinued treatment because of adverse effects, and 3 (1 combined and 2 placebo) refused to continue taking medication because of lack of efficacy. There were no significant differences in the demographic or clinical variables between subjects who completed and those who dropped out of the study. The statistical analyses were computed with and without dropouts, and both methods produced similar outcomes. The latter method was retained to ensure that subjects included in the analyses had received an adequate dosage of treatment (completed $\geq 50\%$ of the treatment protocol). Thus, the results are based on 72 subjects: 18 in CBT, 17 in PCT, 19 in the combined group, and 18 placebo. There was no significant baseline difference across conditions for demographic variables, insomnia severity and duration, prior use of sleep medications, number of physical illnesses, and medications used.

Sleep Data

For each dependent measure, a 4 (group) \times 2 (time; baseline to posttreatment assessment) repeated-measures analysis of variance (ANOVA) was conducted. Sig-

nificant group \times time interactions, indicating a differential treatment effect across groups, were followed by post hoc comparisons using the Tukey Honestly Significant Difference test. Means and SDs for selected outcome measures are presented in TABLE 2 (additional data on other sleep variables are available on request from the corresponding author).

Sleep Diary. Repeated-measures ANOVAs on sleep diary data, using wake after sleep onset, sleep efficiency, total wake time, and total sleep time as dependent measures, yielded significant time effects on all 4 dependent measures ($P < .001$ for all 4). Significant group \times time interaction effects were obtained for total wake time ($F_{3,68} = 5.55$; $P = .002$) and sleep efficiency ($F_{3,68} = 5.52$; $P = .002$). Post hoc comparisons revealed that subjects in all 3 active groups were significantly more improved than those in the placebo condition at posttreatment assessment ($P < .05$ for all 3). Likewise, posttreatment values for wake after sleep onset were significantly lower for all 3 active treatment conditions compared with the placebo condition ($P < .01$ for all 3). Although there was no significant difference among the active treatments, the data suggest a trend for the combined condition to yield greater improvement rates than either of its single components (FIGURE 2). For example, the percentage reduction of wake after sleep onset was highest (63.5%) for the combined condition, followed by CBT (55%), PCT (46.5%), and placebo (16.9%). Likewise, the improvement rate on sleep efficiency was also highest (20.9%) for the combined condition, followed by CBT (16.5%), PCT (10.3%), and placebo (4.4%).

Polysomnography. Repeated-measures ANOVAs on polysomnographic data, using the same dependent measures, yielded significant time effects for all 4 variables ($P < .001$ for all), and significant group \times time interaction effects for total wake time ($F_{3,66} = 2.76$; $P = .05$) and sleep efficiency ($F_{3,66} = 2.70$; $P = .05$). Post hoc comparisons showed that, for both variables, only the combined condition produced greater improvements than the placebo condition ($P < .05$ for both). A

near-significant group \times time interaction effect was obtained for wake after sleep onset ($F_{3,66} = 2.53$; $P = .07$). Post hoc comparisons indicated that the subjects in the 3 active treatment conditions spent less time awake after sleep onset than subjects in the placebo condition. Although no significant difference emerged among the 3 active treatment conditions, the combined condition tended to yield higher improvement rates on most measures. For example, the percentage reduction of wake time after sleep onset was highest (63.3%) for the combined condition, followed by CBT (48.5%), PCT (38.6%), and placebo (7.7%).

Clinical Outcome Ratings

On the Sleep Impairment Index scale, significant group \times time interactions were obtained on composite scores for subjects ($F_{3,63} = 9.86$; $P = .001$), significant

others ($F_{3,53} = 5.41$; $P = .003$), and clinicians' ratings ($F_{3,54} = 9.31$; $P = .001$). Post hoc comparisons indicated that subjects in the CBT and combined conditions rated themselves as significantly more improved (and less impaired) than subjects in either PCT ($P = .01$) or placebo conditions ($P = .002$) (FIGURE 3). There was no significant difference between the 2 medication-only conditions. Combined and CBT subjects were more satisfied, less distressed, and felt less interference with daytime functioning than subjects in the PCT or placebo conditions ($P < .05$ for all). Comparisons of significant others' ratings showed that subjects in all 3 active treatment conditions were perceived as more improved than control subjects ($P < .05$ for all), but no between-group differences were obtained among the active treatments. The same pattern of results was obtained for

clinicians' ratings, which showed all treated subjects as more improved than the control subjects ($P < .05$ for all), with no difference among the active treatments. Analyses of posttreatment global ratings of improvement yielded significant group effects for subjects ($F_{3,68} = 7.54$; $P = .001$), significant others ($F_{3,62} = 10.35$; $P = .001$), and clinicians ($F_{3,61} = 16.37$; $P = .001$). The subjects in the CBT condition rated their improvements higher than those in placebo, and those in the combined group rated their improvements greater than those in PCT or placebo ($P < .05$ for all).

Follow-up

Follow-up data were collected at 3, 12, and 24 months after treatment completion. The number of subjects available at each follow-up assessment is shown in Table 2. Follow-up analyses were con-

Table 2. Group Means and Number of Subjects in Each Treatment Condition*

Assessment Modes	CBT	PCT	Combined	Placebo
Wake After Sleep Onset				
Sleep diary				
Pretreatment	49.58 (51.7) 18	55.09 (37.8) 17	56.96 (48.3) 19	62.24 (37.7) 18
Posttreatment	22.29 (17.0) 18	29.48 (19.5) 17	20.78 (20.2) 19	51.73 (22.7) 18
3-mo Follow-up	28.37 (25.6) 16	34.82 (17.9) 13	34.95 (34.4) 17	61.14 (33.8) 13
12-mo Follow-up	21.22 (17.9) 16	44.97 (23.7) 12	33.57 (28.4) 16	63.21 (33.8) 7
24-mo Follow-up	33.06 (41.3) 13	50.50 (29.5) 12	39.67 (38.0) 14	59.93 (48.5) 10
Polysomnography				
Pretreatment	66.85 (53.0) 18	60.38 (40.2) 17	69.22 (46.9) 19	67.60 (44.0) 18
Posttreatment	34.44 (22.0) 18	37.09 (33.2) 16	25.43 (22.6) 19	62.38 (39.4) 17
Sleep Efficiency				
Sleep diary				
Pretreatment	68.26 (14.2)	72.37 (13.5)	64.00 (16.3)	69.11 (14.0)
Posttreatment	84.80 (7.2)	82.68 (6.4)	84.86 (11.6)	73.49 (11.4)
3-mo Follow-up	82.83 (9.9)	81.10 (8.0)	76.94 (19.5)	73.29 (11.6)
12-mo Follow-up	86.46 (7.5)	77.44 (9.0)	76.68 (19.1)	68.50 (15.6)
24-mo Follow-up	84.70 (12.2)	75.28 (8.2)	77.87 (19.1)	74.30 (13.7)
Polysomnography				
Pretreatment	77.58 (10.0)	77.03 (10.2)	76.26 (11.3)	78.65 (10.3)
Posttreatment	86.08 (6.1)	84.47 (8.7)	86.73 (6.8)	79.91 (8.7)
Total Sleep Time				
Sleep diary				
Pretreatment	321.50 (79.8)	340.21 (73.6)	289.77 (64.7)	331.04 (59.5)
Posttreatment	352.00 (52.4)	383.90 (56.8)	331.93 (65.4)	350.70 (64.7)
3-mo Follow-up	355.57 (54.34)	373.53 (73.6)	327.75 (87.4)	370.34 (75.3)
12-mo Follow-up	375.32 (54.07)	353.52 (61.8)	317.04 (98.0)	319.75 (80.0)
24-mo Follow-up	386.70 (63.41)	351.73 (60.1)	330.63 (85.6)	330.53 (116.0)
Polysomnography				
Pretreatment	353.90 (43.8)	342.90 (51.0)	346.90 (45.8)	371.00 (50.1)
Posttreatment	360.70 (34.4)	378.20 (46.3)	356.10 (38.0)	373.80 (49.5)

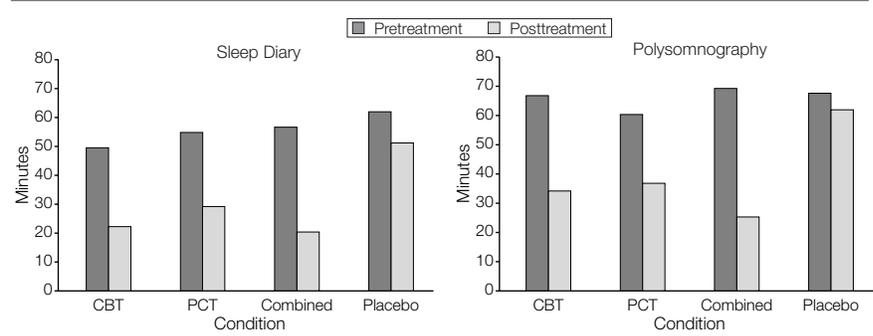
*CBT indicates cognitive-behavior therapy; PCT, pharmacotherapy. All data are mean (SD). Numbers following parentheses are number of subjects in the group.

ducted with the 3 active treatment groups only because placebo subjects were offered an active treatment after the 3-month follow-up, and too few subjects were left for meaningful comparisons of long-term outcomes. In both the PCT and combined conditions, there were 5, 6, and 7 subjects who resumed medication at the 3-, 12-, and 24-month follow-up, respectively. In the CBT condition, there were 3 to 4 subjects who used medication at each follow-up.

Since polysomnographic assessment was conducted only at baseline and post-treatment assessment, follow-up assessments for sleep variables were based on daily sleep diary only. For each dependent measure, within-group *t* tests were conducted to examine possible changes from posttreatment assessment to each of the follow-up assessments. For the CBT condition, there was no significant change on any of the dependent variables at any follow-up, suggesting that treatment gains achieved by posttreatment assessment were well maintained (FIGURE 4). For the PCT condition, significant worsening from the posttreatment period was noted at the 24-month follow-up for total wake time ($t_{11} = -1.92$; $P = .04$), sleep efficiency ($t_{11} = 2.22$; $P = .03$), and wake after sleep onset ($t_{11} = -2.04$; $P = .03$). For the combined condition, significant changes were obtained at all 3 follow-ups on measures of total wake time, sleep efficiency, and wake after sleep onset ($P < .05$ for all), indicating significant worsening of sleep pattern over time.

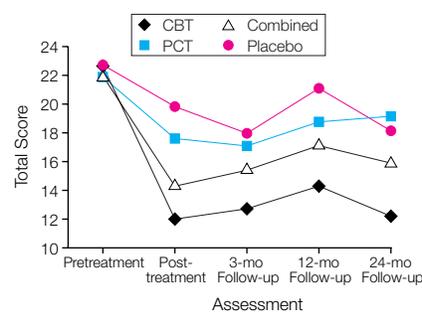
Paired *t* tests were also computed on clinical outcome ratings from subjects and significant others at posttreatment assessment and follow-up. For the CBT condition, subjects' ratings were higher (less favorable) at 12-month follow-up than at posttreatment assessment ($t_{16} = -1.81$; $P = .04$), whereas significant others' ratings were lower (more improved) at the 3- and 12-month follow-ups than at posttreatment assessment ($P < .05$ for all). Subjects' ratings in the combined condition were higher at the 12-month follow-up than their posttreatment ratings ($t_{14} = -2.32$; $P = .02$). There was no significant change in the PCT condition.

Figure 2. Changes in Wake After Sleep Onset From Pretreatment to Posttreatment as Measured by Sleep Diaries and Nocturnal Polysomnography



Sleep diary data are based on 2 weeks of self-monitoring at baseline (before treatment) and the last 2 weeks of treatment. Polysomnographic data are based on nights 2 and 3 before and nights 5 and 6 after treatment. CBT indicates cognitive-behavior therapy; PCT, pharmacotherapy.

Figure 3. Pretreatment, Posttreatment, and Follow-up Changes in Total Scores for the Sleep Impairment Index (Patient Version)

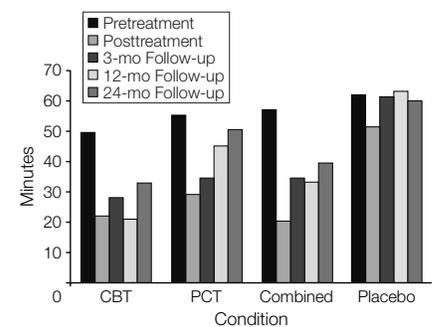


CBT indicates cognitive-behavior therapy; PCT, pharmacotherapy.

Clinical Significance of Sleep Improvements

To examine the clinical (as opposed to statistical) significance of outcome, we computed the proportion of individuals in each condition who reached a sleep efficiency greater than 85%. This criterion is the typical cutoff score used to distinguish clinically impaired from normal sleep. The proportions of subjects meeting this criterion at posttreatment assessment were 55.6% (10/18) for CBT, 47.1% (8/17) for PCT, 68.4% (13/19) for combined, and 22.2% (4/18) for placebo ($\chi^2_3 = 8.37$; $P = .04$). There were significantly more subjects in the CBT ($P = .04$) and combined ($P = .005$) conditions meeting this criterion than in the placebo con-

Figure 4. Changes in Wake After Sleep Onset as Measured by Sleep Diaries Over Assessment Periods



CBT indicates cognitive-behavior therapy; PCT, pharmacotherapy.

dition. When using polysomnographic data, there were 10 subjects in the CBT condition, 10 in PCT, 13 in combined, and 6 in placebo meeting this criterion at posttreatment assessment ($P = .22$). Another criterion, the proportion of subjects who scored lower than 15 on the Sleep Impairment Index (clinician's version) at posttreatment assessment, provides an estimate of the number of subjects no longer meeting insomnia diagnostic criteria. Those proportions were 78% (CBT), 56% (PCT), 75% (combined), and 14% (placebo), respectively ($\chi^2_3 = 15.77$; $P = .001$). There were significantly more subjects in each treatment condition meeting this criterion than in the placebo group ($P < .05$ for all).

Treatment Attendance and Compliance

The number of treatment sessions attended averaged 7.78 per subject (range, 6-8 sessions), for an overall attendance rate of 97.1%. Make-up sessions either in person or via telephone were conducted whenever possible. There were no significant differences between conditions for treatment attendance. One-way (group) ANOVAs on subjects' self-ratings of compliance with treatment revealed no significant group difference. Subjects in all 4 conditions rated their degree of compliance equally high (group means >4.2 on a 5-point scale). Likewise, there was no significant difference on compliance ratings provided by significant others. No difference was noted between conditions on ratings of therapist competence, empathy, and support. We examined compliance with medication use by comparing the proportion of medicated nights and average dosage used. No significant differences were found among the 3 groups in the percentage of medicated nights (placebo, 79%; PCT, 75%; combined, 70%) or in the average dosage used during the treatment period (placebo, 20 mg/night; PCT, 20 mg/night; combined, 16 mg/night). Urine drug screens were performed at baseline and posttreatment assessment. At baseline, 1 subject in the combined condition had traces of benzodiazepine; at posttreatment assessment, all PCT and combined subjects had positive screen findings, and all but 1 subject in the placebo condition had negative urinalysis findings for benzodiazepines.

COMMENT

The present findings indicate that behavioral and pharmacological therapies, alone or in combination, are effective in the short-term management of late-life insomnia. Their main effects are to improve sleep continuity and efficiency. Subjects who received an active treatment were able to maintain sleep more efficiently than placebo-control subjects. There was a nonsignificant trend for the combined condition to produce slightly higher improvement rates on sleep con-

tinuity measures than either treatment alone during the initial treatment phase. Follow-up results showed that behavior therapy yielded the most durable improvements in sleep patterns; PCT gradually lost its clinical benefits over time; and the combined approach yielded more variable long-term outcomes.

These results extend those from previous studies^{21-25,39,41} and provide additional evidence that chronic insomnia is a treatable condition even in late life. Once older adults are well screened for other sleep disorders such as sleep apnea and periodic limb movements, 2 conditions particularly prevalent in the elderly, their treatment response is similar to that of younger adults.^{16,19} For example, the percentages of reduction of time awake after sleep onset were 64% for the combined condition, 55% for the CBT, and 47% for PCT, with all 3 conditions reducing their posttreatment values on this variable below the 30-minute criterion typically used to define sleep-maintenance insomnia. These clinical benefits were also corroborated with objective electroencephalographic measures. As illustrated in Figure 2, the magnitude of clinical improvements obtained on polysomnographic measures is slightly smaller but clearly in the same direction as those obtained on subjective daily sleep diary measures.

The clinical significance of these results is illustrated by the proportion of patients who reached a normative sleep efficiency of 85% or who obtained a normative score of less than 15 on the Sleep Impairment Index measure. These findings were paralleled by collateral ratings from patients and significant others. Patients who received active treatments rated themselves and were judged by significant others as more improved than control patients on measures of insomnia severity, interference with daytime functioning, and level of distress.

Although all 3 active treatments were effective during the initial intervention, sleep improvements were not equally well maintained across conditions. Initial gains produced by behavioral and pharmacological interventions were well maintained at the short-term (3-

month) follow-up. However, long-term (12- and 24-month) follow-up data showed a different trajectory of change in that only behavioral treatment produced durable changes. Clinical benefits obtained by subjects treated with drug therapy alone were gradually lost. Only 1 of 12 subjects available at the 12-month follow-up had a sleep efficiency greater than 85%; by the 24-month follow-up, subjects treated with medication alone had returned to their baseline sleep difficulties. The combined approach produced more variable outcomes across subjects. Although about half of the subjects in the combined approach retained a sleep efficiency greater than 85% at each follow-up, 3 subjects (outliers) reported significant worsening of sleep difficulties, which contributed to the overall deterioration of the combined treatment condition.

These latter findings indicate that, while a combined approach may produce slightly larger benefits initially, these gains are not necessarily better maintained. One possible explanation is that subjects treated with hypnotic medications, singly or combined, may attribute their initial gains exclusively to the medication. These subjects may be more vulnerable to relapse when the drug is discontinued. While these findings are consistent with Hauri's study,²⁷ and may suggest that insomnia is best treated with a behavioral approach alone, additional research is needed to determine the optimal model for integrating behavioral and drug therapies. A sequential approach, in which medication would be initiated first and behavioral treatment introduced or continued when the drug is discontinued, may prove a more effective method than concurrent initiation and discontinuation of both treatment modalities together.

Some methodological limitations preclude more definite conclusions about effectiveness of treatment for late-life insomnia. While this was the largest controlled clinical trial comparing behavioral and pharmacological therapies for insomnia, the sample size was relatively small; this may have reduced power to detect more differences among the conditions. Another limitation con-

cerns the generalizability of the findings. The sample was composed mainly of subjects responding to newspaper advertisements; in addition, many subjects were excluded because of comorbid medical or psychiatric illness or because they were unable to stop taking their hypnotic medications prior to enrollment. Future studies should enroll more subjects from primary care settings and examine outcome as a function of medical and psychiatric conditions often associated with insomnia.^{2,42} Finally, long-term follow-up data must be interpreted cautiously because of the increasing attrition over time and because several subjects initiated or resumed medication between posttreatment and follow-up assessments.

Despite these limitations, this study has implications for clinical practice. Insomnia is a widespread complaint, particularly in late life, and is associated with functional impairments, diminished quality of life, and increased health care costs.^{2,4,9,10} In addition, insomnia is typically undertreated and nondrug interventions are underused by health care practitioners. Our results indicate that chronic insomnia can be effectively treated in late life with structured and sleep-focused interventions aimed at changing poor sleep habits and faulty beliefs and attitudes about sleep. Although such behavioral intervention is more time consuming than drug therapy, it is worth the investment because therapeutic gains are well maintained. The results also indicate that PCT alone, although

effective in the short term, may not be sufficient for long-term management of chronic insomnia. Because insomnia is often a recurrent problem⁴² and because many older persons use hypnotic medications for much longer than the standard recommended period,¹⁸ more studies are needed to evaluate long-term effects of hypnotics. Despite the intuitive appeal in combining drug and nondrug interventions, the present results call for additional research to further evaluate other models (eg, sequential approaches, maintenance trials) for optimal integration of biobehavioral approaches in the clinical management of insomnia.

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