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Behavioral Treatment of Insomnia: Treatment Outcome and the Relevance of Medical and Psychiatric Morbidity

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Recently, we undertook a case series study and found that behavior therapy for insomnia was effective as plied in the clinic setting and that the findings were similar to those in the "clinical trial" literature. In the present study, we evaluate a second set of case series data to assess (1) the replicability of our original findings, (2) if our treatment outcomes are statistically comparable to those in the literature, and (3) if medical and psychiatric morbidity influence treatment outcome. It was found that patients who completed four or more sessions of cognitive behavioral therapy for insomnia (CBT) were, on average, 33% improved. This average corresponded to a 56% reduction in wake time after sleep onset, a 34% reduction in sleep latency, a 29% increase in total sleep time, and a 13% decrease in number of awakenings per night. These findings are not significantly different from those reported in literature for both CBT

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and pharmacotherapy interventions. Medical and psychiatric comorbidity did not influence treatment outcome

KEY WORDS: sleep: insomnia: treatment outcome: behavioral treatment.

INTRODUCTION

In 1997 we established a full time behavioral sleep medicine clinic. After the first year of operation we undertook a clinical case series study (1997—1998) to evaluate this program (Perlis *et al.*, 2000). The central issues were (1) do patients who complete treatment exhibit significant pre-post change and (2) does the magnitude of improvement appear comparable to that which is reported in the clinical trials literature (e.g., Morin *et al*., 1994; Murtagh and Greenwood, 1995). The latter issue contained within it a more subtle question: is Cognitive Behavioral Therapy (CBT) as effective as it is efficacious. That is, does behavioral therapy for insomnia, as it is plied in clinical settings, yield results similar to those obtained in "clinical trial" research? In routine clinical practice there are a variety of factors that may influence treatment outcome that are not issues and/or are controlled in treatment efficacy studies. Some factors may limit patient ability to participate in, or benefit from, treatment (e.g., medical and/or psychiatric comorbidity). Some factors may enhance outcome (e.g., self-referral, payment for service, level of therapist specialty training and/or clinical experience). In the 1997—1998 case series study, it was found that patients were, on average, 43% improved. This average corresponded to a 65% reduction in sleep latency, 46% decrease in number of awakenings per night, a 48% reduction in wake time after sleep onset, and a 13% increase in total sleep time. These results, although not formally compared with normative values from the literature appeared comparable. Given that we were able to provide effective therapy and our results were similar to those in the literature, it seemed reasonable to conclude that there is a balance between the factors that detract from, and those that contribute to, positive treatment outcome in the clinic setting.

In the present analysis, we undertook a second clinical case series study (1998—1999) to determine the replicability of our original findings. In addition, we sought to formally (1) compare our clinical outcome data to normative data from the literature, and (2) assess whether medical and/or psychiatric comorbidity influence treatment outcome. In order to accomplish the first goal, we compared our 1998—1999 data to average sleep continuity values from a comparative meta-analysis data set we recently compiled (Smith *et al*., 2000). To accomplish the second goal, patients in our sample were identified as having or not having medical or psychiatric morbidity and these groups were evaluated for different treatment outcomes.

METHODS

Data Source

The data for the present study were compiled as part of a comprehensive chart review. Human subject rights were protected; approval for this chart review study was obtained from the research subject review board (University of Rochester's internal review board). The need for informed consent was waived provided that all data were coded to ensure patient anonymity.

Subjects and Setting

From October 1998 to November 1999, 89 consecutive patients were evaluated. Of the patients seen during this time, 2.2% were in continuing treatment, 32.6% completed treatment, 34.8% postponed treatment or were referred, and 30.3% elected to discontinue therapy. More than 95% of the patient sample described themselves as European American. The average age was 46 (±16) and 65% were female.

The clinic was housed within an active 10-bed sleep disorders center and was staffed by two psychologists and one physician. The majority of the patients in the sample were treated by one clinician with a PhD in Clinical Psychology, 15 years experience as a clinician, and a specialized expertise in Behavior Therapy and Behavioral Sleep Medicine. Prior to assuming clinical responsibilities, this therapist was provided with a 2-week training period during which she was familiarized with the clinical protocol and provided the opportunity to discuss and/or amend the treatment regimen. This clinician was also peer supervised on a weekly basis (clinic case conference).

General Protocol

Patients were physician or self-referred for insomnia treatment. All patients were triaged by telephone to the clinic and underwent an extensive intake interview. During their intake we determined whether or not the patient (1) had primary insomnia or insomnia secondary to stable medical and/or psychiatric conditions, (2) was using hypnotic medications, and (3) was inclined to attempt a behavioral treatment regimen.

If it was suspected that the patient had an unstable medical or psychiatric condition, the patient's primary care physician or psychiatrist was contacted, and/or the patient was referred for primary care. In the present context,

"stable medical and/or psychiatric condition" refers to the instances where the primary disorders were already being treated and/or where patients had elected not to receive treatment for the primary disorder. In the case of psychiatric disorders, none of the patients were evaluated to be at risk for harm to self, others, or in need of hospitalization.

If patients were using hypnotic medication, and elected to seek behavioral treatment, patients were referred back to the prescribing doctor to titrate off medication. In this instance, therapy was delayed until patients were medication free for 1—2 weeks. Patients who elected to discontinue medication and begin behavior therapy were monitored during the withdrawal period. Monitoring involved the use of daily sleep diaries. Active therapy was not delivered during the withdrawal period.

After the intake interview and/or medication withdrawal, an initial baseline measurement period (1—2 weeks) was obtained. Following this interval, patients were seen for an additional 3—9 sessions. Over the course of the first four treatment sessions, standardized interventions were undertaken including Sleep Restriction, Stimulus Control, Sleep Hygiene, and Cognitive Therapy. Brief descriptions of the procedures for these therapies follow (for more detailed information, please refer to Buysse and Perlis (1996) and/or Bootzin and Perlis (1992). Sessions 5—9 were used for extended monitoring, prolonged sleep restriction therapy, or for adjunctive therapies. Adjunct therapy included additional cognitive therapy, relaxation training and/or light therapy as needed for patients who were having difficulty staying awake until, or rising at, the prescribed hour. Sleep was monitored prospectively using sleep diaries for the duration of treatment.

Standard Therapy

Session 1 (Evaluation and 1—2 Week Baseline)

The intake evaluation session was typically 90—120 min. During the session, the clinician reviewed the patient's medical, psychiatric, and sleep disorders history. This interview was, in part, structured around questionnaire materials that were completed by the patient prior to the intake session. The questionnaires include an extensive sleep questionnaire, a medical history and symptoms checklist, a Beck Depression Inventory (BDI), a Beck Anxiety Inventory (BAI), and demographic questionnaire. By the end of this session, the clinician determined if a referral was required (for unstable primary medical or psychiatric problems), whether or not the patient needed to be withdrawn from medication, and/or whether or not the patient was willing to engage in a behavioral regimen. If the patient elected to continue

in treatment, he/she was instructed to keep a sleep diary for a period of 1-2 weeks and was instructed on how to complete this measure.

Session 2 (Sleep Restriction and Stimulus Control Therapy)

During this session, baseline sleep diary data were reviewed. This information was used to set the parameters for sleep restriction therapy and served as a means to guide the patient toward the treatment to be prescribed. Our standard approach was didactic. The patient and the clinician evaluated the data together. After reviewing the data and identifying certain basic assumptions, most patients easily deduced what might represent a good "counterstrategy." The primary assumption most patients identify was what we call "the positive correlation fallacy": the more time spent in bed, the more sleep one will get. Once the patient had identified one or more of the components of therapy, the clinician explained in detail the rationale and procedures for Sleep Restriction and Stimulus Control Therapy.

In brief, sleep restriction consists of (1) curtailing the amount of time spent in bed (TIB) so that this time frame matches the amount of time that the patient actually spends in bed asleep (TST) and (2) a process of upward titration so that TIB is extended in 15-min increments. The rule governing upward titration is that the patient must, on average, sleep efficiently for a week before the sleep opportunity is extended, that is 90% of the time spent in bed must be spent asleep. We did not restrict lower than 4 h TIB. For detailed Information regarding Sleep Restriction, see Spielman *et al.* (1987a,b).

Stimulus control consists of providing a set of instructions that curtail behaviors incompatible with sleep and insures that the patient does not spend appreciable amounts of time in bed awake. The core instructions are (1) Lie down to sleep, only when sleepy, (2) Use the bed only for sleep and sex, (3) If unable to fall asleep, get up and go into another room. Stay as long as needed, but when sleepy return to the bedroom to sleep, and (4) when in bed and awake for longer than 10 min, repeat Step 3. Unlike Bootzin's original formulation (Bootzin, 1972), we did not specify a time increment after which the patient should get out of bed and leave the bedroom. Instead we recommend that the moment the patient clearly perceives that he/she was awake and/or feels annoyed should be the cue to get up.

Session 3 (Sleep Hygiene and Sleep Restriction Therapy Adjustments)

At the beginning of this session, as with all sessions, sleep diary data were reviewed and charted. The upward titration process was begun and

sleep hygiene instructions were reviewed by having the patient read aloud the various imperatives and the corresponding rationales. After the patient and the clinician had identified whether the issue was relevant, the clinician reviewed in more detail the basic concepts and related clinical research. The amount of, and manner in which, information was presented varied according to patient interest.

In brief, sleep hygiene instruction refers to the identification of sleep-enhancing behaviors. Typically, sleep hygiene issues are reviewed with the patient by providing a list of guidelines that include both common sense directions and instructions that address self-defeating strategies that patients often adopt. Common sense instructions include "cut down on caffeinated products" and "avoid excessive liquids in the evening." Instructions regarding self-defeating strategies include "Avoid alcohol, especially in the evening" and "Don't smoke during the night when unable to sleep." (For more detailed information regarding sleep hygiene, see Zarcone, 1989.)

Session 4 (Sleep Restriction Therapy Adjustments)

Upward titration continued.

Session 5 (Sleep Restriction Therapy adjustments and Cognitive Restructuring)

Upward titration continued. We also undertook a Barlow-style approach to "decatastrophization" (e.g., Barlow, 1992). That is, we addressed the perception of dire consequences from sleep loss, using a form of cognitive restructuring. This involved reviewing the "worst possible outcome" scenarios and exploring the mismatch between the certainty that there would be negative outcomes and the frequency with which such events actually occurred. For more information on cognitive restructuring as it applies to insomnia, see Buysse and Perlis (1996) or Morin and colleagues' work on cognitive therapy for insomnia (e.g., Morin *et al.*, 1993; Morin and Azrin, 1988).

Session 6 (Sleep Restriction Therapy Adjustments and Relapse Prevention)

If adequate clinical gains had been made, relapse prevention issues were reviewed. Typically, this entailed a review of (1) "how insomnia gets started" and the behaviors that maintain poor sleep and (2) the strategies that are likely to abort an extended episode of insomnia. If treatment had not been

successful, one of three recommendations were made: therapy was continued for 1— 4 sessions, a referral for a sleep study was made, or further diagnostic work was recommended.

Sleep Diaries

Our clinic used scannable sleep diaries produced by Clearview Printing. This instrument contains two major color-keyed sections. The first was completed prior to bedtime and was composed of 14 questions to assess daytime behavior and mood. The second was completed upon awakening and was composed of 16 questions to assess standard parameters such as subjective perception of sleep latency, number of awakenings, time awake during the night, and total sleep time. The sleep diaries allowed the clinician to (1) prospectively evaluate sleep disturbance complaints, (2) tailor sleep restriction therapy to the individual, and (3) track treatment outcome. Each week, sleep diary data were summarized at the beginning of the session. Measures of sleep latency, number of awakenings, duration of awakenings, total sleep time, and sleep efficiency were calculated. All self-report sleep variables were coded onto a simple form for the clinic chart and were graphed. The chart supplemented the clinic progress notes and the graphs allowed the patient and clinician to have a visual representation of the treatment course as compared with baseline measures.

ANALYSES

Treatment Effectiveness

Treatment completion was defined using a minimum adequate trial cutoff. The cutoff was four or more sessions (intake and three treatment sessions). Analyses were undertaken using change score values and one measure of overall change in terms of global percent improvement. Baseline data were compared with end-of-treatment data using paired *t* tests. Bonferroni corrected *p* values were used to determine significance. The correction was calculated by multiplying the uncorrected *p* values by the number of tests in the analyses (e.g., 0.07 x 4 = 0.28). Outcome measures were change scores calculated individually for sleep latency (SL), number of awakenings (NA), duration of awakenings (WASO), total sleep time (TST), and average percent improvement (Global). Change scores were calculated by subtracting baseline values from end-of-treatment values. Also calculated was a variable that represented overall change. This variable was constructed by averaging the percent improvement scores for each sleep parameter. Inverse values

were used for percent change for total sleep time so that the direction of change for all parameters was constant.

Treatment Outcome Compared with Literature Norms

In order to facilitate comparison, our data were formally compared with average literature norms using one-sample *t* tests. The standardized norms for these analyses were taken from a comparative meta-analyses undertaken by our group. Detailed information about this data set may be found elsewhere (Smith *et al.,* 2000). In short, an initial pool of 190 treatment outcome studies of primary insomnia were identified by a Medline and PsychInfo search (1966 — present) and from reference lists provided by the authors from two of the previous meta-analyses (Nowell *et al.,* 1997; Morin *et al.,* 1994). The 190 studies were reviewed for the following inclusion and exclusion criteria. *Inclusion criteria*: (1) duration of insomnia >3 months, (2) cognitive—behavioral treatments must include either stimulus control or sleep restriction, (3) pharmacological treatments must be either benzodiazepines or like GABAergic agents (e.g., zolpidem, zopiclone), (4) studies must use sleep diary measures and have pre/post data. *Exclusion* criteria were (1) sleep continuity variables were presented as ordinal data, and (2) no mean or standard deviation data. Pre—post treatment means and standard deviations were calculated for major sleep continuity variables. Fourteen cognitive—behavioral (CBT) studies involving 250 subjects and 8 pharmacotherapy (PT) studies involving 286 subjects met the inclusion/exclusion criteria. The two groups did not differ with respect to gender or age. The average number of CBT sessions was 4.9 ± 2 over an average period of 5.3 ± 2.1 weeks. The average length of PT was 2 ± 2 weeks. Our treatment outcome data were compared with the average sleep continuity variables for both the pharmacologic and CBT literature. Independent comparisons were made for sleep latency, number of awakenings, wake after sleep onset time, and total sleep time. Bonferroni corrections were also utilized for these analyses.

Treatment Effectiveness Relative to Medical and Psychiatric Comorbidity

As indicated in the introduction, one of our central questions was to assess whether medical and/or psychiatric morbidity influence treatment outcome. To accomplish this, we segregated our sample into two groups based on whether the subjects had formal medical or psychiatric co-morbid diagnoses. The groups were then compared for age, sex, and pre—post change for each of the four sleep continuity variables (SL, NA, WASO, TST). A set of four

t tests were used to assess whether patients with *medical* comorbidity exhibited divergent treatment outcomes. A set of four *t* tests were used to assess whether patients with *psychiatric* comorbidity exhibited divergent treatment outcomes. Bonferroni corrections were applied for each set of analyses.

RESULTS

Description of Sample

From October 1998 to November 1999, 89 consecutive patients were evaluated and treated. Of these patients, 30% of the subjects were diagnosed with primary insomnia, 30% with insomnia secondary to Major Depression, 35% with insomnia related to circadian rhythm disturbances or other intrinsic sleep disorders, and 5% with insomnia secondary to psychiatric disorders other than depression.

Of the original 89 patients, 28 had a minimum adequate trial of therapy and complete data. Average number of sessions for this group was 7.0 (±1.76). Comparisons between the 27 patients who discontinued treatment with the 29 patients that met the adequate trial definition for completion revealed that the groups did not significantly differ with respect to age, sex, race, BDI or BAI scores, and prevalence of medical or psychiatric disorders. Patients who completed treatment, however, were more likely to be diagnosed with primary insomnia and delayed sleep phase syndrome.

Of the 28 subjects categorized using the adequate trial definition, 65% were female and the average age was 46.5 (±16) years; 50% reported primary psychiatric disorders; 53% reported primary medical disorders. The average BDI was 8.8 (±5.8), and the average BAI was 7.6 (±4.6). Although these average scores are not in the clinically significant range, they do represent subclinical levels of depression and anxiety. The most common medical problems were related to musculoskeletal disorders, headache, and gastrointestinal disorders. The most common psychiatric disorders were mood and anxiety disorders. Prior to treatment, the average sleep profile as derived from the baseline sleep diary data was as follows: sleep latency = 55.0 min (±51.8), number of awakenings = 2.7 (±1.8), wake time after sleep onset = 83.1 min (±62.8), total sleep time = 292.0 min (±84.2).

Treatment Effectiveness

All pre-post comparisons for this clinical case series were significant at $p < .05$ after Bonferroni correction was applied ([Alpha] x [# of tests

per group] = 0.07 x 4 = 0.28) except for total sleep time (see Table I for details). On average, subjects who completed a minimum adequate trial were about 33% improved. This average corresponded to a 34% reduction in sleep latency (effect size = 0.85), 13% decrease in number of awakenings per night (effect size = 0.54), a 56% reduction in wake time after sleep onset (effect size = 1.14), and a 29% increase in total sleep time (effect size = 0.55). In actual minutes, patients, on average, fell asleep about 33 min quicker, woke up about 1 fewer times and obtained about 50 more minutes of sleep a night.

Treatment Outcome Compared with Literature Norms

As can be seen in Table I, average values for four sleep continuity variables, for both the pharmacologic and CBT literatures, are provided. Independent comparisons for sleep latency, number of awakenings, wake after sleep onset time, and total sleep time revealed that our treatment outcomes did not significantly differ from the literature norms for CBT except in one instance: total sleep time in our sample was increased by more than 2.5 times the amount typically produced by CBT. Our treatment outcomes, when compared with the average pharmacologic intervention, produced comparable results for three of the four sleep continuity variables and superior results for wake after sleep onset time.

Treatment Effectiveness Relative to Medical and Psychiatric Comorbidity

After the sample was dichotomized, it was found that the Medical (Med Dx [*n* = 16] vs. No Med Dx [*n* = 12]) and Psychiatric groupings (Psych Dx [*n* = 12] vs. No Psych Dx *[n* = 16]) did not significantly differ by age and sex. The groups also did not significantly differ for their respective treatment outcomes. For specific sleep continuity comparisons, see Table II.

DISCUSSION

The present study had three goals: (1) to determine whether patients in our clinical service exhibit significant improvement, (2) to compare our clinical outcome data to literature based norms, and (3) to assess whether medical and/or psychiatric morbidity is associated with treatment outcome. It was found that patients who completed a minimum adequate trial were significantly improved overall and this improvement corresponded to a 34% reduction in sleep latency, 13% reduction in number of awakenings, a 56% decrease in wake after sleep onset time, and a 29% increase in total sleep

Table I. Treatment Outcome Data From the 1998–1999 Case Series and Normative Values for Pharmacotherapy & CBT

Subjective sleep continuity measure (sleep diary)	Pretreatment value, M (SD)	Posttreatment value, M (SD)	Pre- to posttreatment change (difference)	%	p[a]	*p*[b]
Sleep latency (min)[a]						
BSMC 98—99	55.0 (51.8)	21.7 (19.4)	-33.3 (44.8)	34	.0005	.002
*Pharmacotherapy (PT)	48.8 (29.7)	34.4 (26.3)	-14.5 (9.0)	30		
*Cognitive-Behavioral TX (CBT)	54.2 (28.5)	30.9 (16.0)	-23.3 (15.0)	43		
Number of awakenings[b]						
BSMC 98—99	2.7 (1.8)	1.9 (1.1)	-0.8 (1.4)	13	.0054	.022
*Pharmacotherapy	3.0 (2.0)	1.8 (1.0)	-1.2 (0.1)	39		
*Cognitive-Behavioral TX	2.4 (1.8)	1.7 (1.6)	-0.8 (0.8)	31		
Wake After Sleep Onset (min)[c]						
BSMC 98—99	83.1 (62.8)	27.7 (27.6)	-55.4 (50.3)	56	.0001	.0004
*Pharmacotherapy	55.1 (37.8)	29.5 (19.5)	-25.6 (N/A)[C]	46		
*Cognitive-Behavioral TX	69.4 (44.8)	30.2 (24.0)	-39.2 (14.1)	56		
Total Sleep Time (min)[d]						
BSMC 9—99	291.9 (84.2)	341.7 (95.1)	+49.8 (114.3)	29	.0263	.105
*Pharmacotherapy	332.2 (55.3)	372.6 (49.0)	+40.5 (23.7)	12		
*Cognitive-Behavioral TX	333.3 (63.3)	352.9 (44.2)	+19.6 (22.8)	6		

Note. Asterisk indicates values from a comparative meta-analyses (see Smith *et al.*, 2000; Perlis, *et al.*, 2000). Superscripts *a, b, c*, and *d* indicate one-sample *t* test for pre-post change. [a]BSMC vs. PT $p = 0.035$; BSMC vs. CBT p = 0.25. [b]BSMC vs. PT $p = 0.187$; BSMC vs. CBT $p = 0.858$. [c]BSMC vs. PT $p = 0.004$; BSMC vs. CBT $p = 0.099$. [d]BSMC vs. PT $p = 0.905$; BSMC vs. CBT $p = .006$.

[a]Paired *t* tests for change in minutes without Bonferonni correction.

[b]Paired *t* tests for change in minutes with Bonferonni correction (e.g., 0.05 x 4 = 0.20).

[c]sample size = 1.

Table II. Contrast Between Participants With and Without Psychiatric and Medical Comorbitity

Subjective sleep continuity measure (sleep diary)	Pre- to posttreatment change (difference)	p^a
Psychiatric comorbidity[b]		
Sleep latency (min)		.422
No psychiatric DX	- 39.3 (53.2)	
Psychiatric DX	- 25.3 (38.1)	
# Awakenings		.681
No psychiatric DX	- 0.7 (1.4)	
Psychiatric DX	- 0.9 (1.5)	
WASO (min)		.845
No psychiatric DX	- 57.0 (41.9)	
Psychiatric DX	- 53.29 (61.7)	
TST (min)		.304
No psychiatric DX	+ 68.4 (142.0)	
Psychiatric DX	+ 23.4 (51.3)	
Medical comorbidity[c]		
Sleep latency (min)		.312
No medical DX	- 43.4 (45.8)	
Medical DX	- 25.8 (44.1)	
# Awakenings		.487
No medical DX	- 1.0 (1.3)	
Medical DX	- 0.6 (1.6)	
WASO (min)		.248
No medical DX	- 68.3 (29.6)	
Medical DX	- 45.7 (60.6)	
TST (min)		.822
No medical DX	+ 55.2 (86.4)	
Medical DX	+ 45.4 (135.6)	

[a] Paired *t* tests for change in minutes.
[b] with Psych Dx, $n = 12$; Without Psych Dx, $n = 16$.
[C] With Med Dx, $n = 16$; Without Med Dx, $n = 12$.

time. These gains substantially parallel the average outcomes reported in the cognitive-behavioral treatment and pharmacologic treatment literatures. Finally, medical and/or psychiatric comorbidity was not found to substantially influence treatment outcome.

Treatment Effectiveness

As with our previous case series study (Perlis *et al.*, 2000), we found that patients' sleep continuity significantly improved given an adequate therapeutic trial. The only sleep parameter not significantly improved was total sleep time. Although this was found to be significantly increased in the prior study, this parameter was, in both cases, the least affected by behavior therapy. This is not surprising. The two major components of treatment

(Sleep Restriction and Stimulus Control) both serve to mildly sleep deprive the patient. This deprivation, at least in acute therapy, increases the homeostatic drive to sleep and thereby leads to a reduction in sleep latency and wake after sleep onset time. Presumably, total sleep time increases as the insomnia is counter-conditioned. Long-term follow-up data from both meta-analyses on the behavioral treatment of insomnia are consistent with this perspective (Morin *et al*., 1994; Murtagh and Greenwood, 1995). For example, Morin and colleagues show that total sleep time increases from a posttreatment value of 377.9 to 395.0 min at follow-up (1994, p. 1178, Table V).

Treatment Outcome Compared with Literature Norms

As indicated in the results section, our treatment outcomes were largely comparable to those reported for CBT in the literature. The one instance where this appears not to be the case is with respect to total sleep time: patients in our program appeared to accrue more total sleep within the acute treatment phase. The most likely explanation for this is related to duration of treatment. In our program, the average number of weeks in treatment was 7.0 ± 1.8. In the CBT sample in our meta-analysis, the average number of weeks in treatment was 5.3 ± 2.1. Thus, our patients continued in treatment for, on average, two more weeks. Given good sleep efficiency over this time interval, which was likely at this point in treatment, total sleep time would therefore be expected to increase by an additional 30 min. The 30-min difference between our sample and the literature norms suggests that the extended therapy period produced additional gains. This conclusion, however, must be considered tentative as there is evidence that total sleep time increases after successful therapy regardless of additional active treatment (Morin *et al*., 1994).

The comparison between our treatment outcomes and the pharmacologic data from our meta-analyses revealed that CBT produced comparable gains for three of the four sleep continuity variables and superior outcome for wake after sleep onset time. This result may be related to our rigorous use of sleep restriction therapy that was specifically developed to address sleep maintenance issues, that is, to minimize duration of nocturnal awakenings (Spielman *et al*., 1987a,b).

Treatment Effectiveness Relative to Medical and Psychiatric Comorbidity

Our data suggest that medical and/or psychiatric morbidity do not significantly influence treatment outcome. While this directly supports our contention that behavioral sleep medicine interventions are as effective as they

are efficacious, it is important to bear in mind that patients with significant acute illness were referred and thus not represented in this analyses. There are, however, two studies that provide direct support for the possibility that psychiatrically and/or medically ill patients may significantly benefit from behavioral treatment for insomnia (one study by Dashevsky and Kramer (1998), and one study by Lichstein *et al.* (2000)).

In the study by Dashevsky and Kramer (1998), it was found in a sample of 48 subjects (75% female) with insomnia secondary to psychiatric illnesses (mean age 47.3 ± 12.4) that behavioral interventions produced significant pre—post changes in sleep continuity. They found, after 8 weeks of treatment, that sleep latency was reduced by about 38 min (61% reduction), number of awakenings by about 1 episode (52% reduction), wake after sleep onset by about 42 min (68% reduction) and that total sleep time was increased by approximately 36 min (10% increase). Although these data are suggestive, it is not clear whether the patients in this study were in remission or still actively ill. A further limitation of the study is that 96% of the sample were treated with dual therapy (pharmacotherapy and CBT). Thus, it remains possible that significant acute illness may undercut patients' ability to participate in, and/or benefit from, monotherapy by using only cognitive behavioral treatment for insomnia.

In the study by Lichstein *et al.* (2000), in a sample of 23 subjects with insomnia secondary to both psychiatric and medical illness (ages> 58 years old) it was found that behavioral interventions also produced significant pre-post changes in sleep continuity. They found, after 4 weeks of treatment, that sleep latency was reduced by about 17 min (35% reduction), number of awakenings by about 0.5 episodes (14% reduction), wake after sleep onset by about 26 min (30% reduction) and that total sleep time was increased by approximately 46 min (14% increase). A strength of this study was that it had a control for the treatment condition. A limitation of this study was that the treatment regimen did not include sleep restriction therapy.

The results from these two studies, as well as the data from the present study, suggest that patients with secondary insomnia may significantly benefit from standard behavioral treatments for insomnia.

FINAL COMMENTS

As with our prior study, some may argue that a weakness of the present study is that it is based on self-report pre–post data. Serial measures, showing progressive improvements would certainly enhance our confidence that the clinical outcomes are systematically related to treatment. As for the self-report nature of the data, it has been argued that polysomnographic and/or

actigraphic data are required for a complete understanding of pre–post treatment change (e.g., Stepanski, 1989). While the assumption that underlies this point of view may be challenged (i.e., that polysomnography is a more valid measure than prospective self-report; e.g., Perlis *et al.*, 1997), the issue itself may be moot in context. The present investigation is a treatment effectiveness study. Since prospective self-report measures constitute the standard of practice for behavioral sleep medicine, they are the appropriate measure for this type of investigation.

The major strength of the present study is that we are (1) able to formally compare our clinical results with those that are reported in the literature and (2) able to assess the impact of medical and psychiatric morbidity on treatment. These components of this study allow us to conclude that the treatment delivered in our clinic does indeed appear to meet and exceed the published standards *for both* CBT and pharmacologic interventions and that such gains may be obtained in patients with moderate comorbid illness.

Finally, as noted in our prior report, it is important that more treatment effectiveness research be undertaken. In part, additional research will determine whether the treatment effect findings of this study are reliable across time and across treatment centers. More important, however, is that further research is needed to address (1) what variables are related to attrition and acute and long-term treatment outcome and (2) how treatment outcome is related to quality of life and health care utilization. These issues are presently under investigation.

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