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Behavioral Treatment of Insomnia: A Clinical Case Series Study

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There is substantial experimental evidence that behavioral treatment of insomnia produces significant clinical improvement and that treatment gains tend to be maintained over time. Less clear is whether behavioral treatment is effective as it is plied in clinical settings. In this clinical case series study, we evaluated 47 patients with primary insomnia. It was found that patients were, on average, 43% improved. This average corresponded to a 65% reduction in sleep latency, a 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 suggest that behavioral treatment for insomnia is as effective in clinical settings as it is as under clinical trial conditions.

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

INTRODUCTION

Over the course of the last three decades, seven nonpharmacologic treatments for insomnia have been developed and subjected to empirical validation. The treatments include two types of relaxation therapy, a paradoxical

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intention intervention, frontalis biofeedback, sleep hygiene, stimulus control, sleep restriction, and cognitive therapy. Recently, the outcome data from the various treatment efficacy studies were reviewed in two meta-analyses: one by Morin and colleagues (1994) and one by Murtagh and Greenwood (1995). The results from these quantitative reviews suggest that behavioral treatment produces significant improvement, that sleep restriction or stimulus control yield the greatest gains, and that treatment effects are maintained or enhanced over follow-up periods ranging from 3 weeks to 3 years. These global trends correspond to an average of 32–41% overall improvement and 39–43% reductions in sleep latency, 30–73% reductions in number of intermittent awakenings, 46% reductions in the duration of intermittent awakenings, and 8–9.5% increases in total sleep time. In actual minutes, pre-post measures show that patients fall asleep about 25 min sooner, have 0.5–1.2 fewer awakenings, and obtain about 30 min more sleep a night.

Not clear from the work that is now available is whether behavioral therapy is as effective as it is efficacious. That is, does behavioral therapy for insomnia, as it is plied in clinical settings, yield results comparable to those obtained in clinical trial research? In routine clinical practice there is 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 specialty training and/or clinical experience). Whether these various factors, on balance, negatively or positively influence treatment outcome is an important question. In the present study, clinical chart data from an active Behavioral Sleep Medicine Clinic are evaluated to determine (1) whether routine cognitive behavioral interventions significantly improve sleep continuity and (2) whether such improvements appear to be comparable in magnitude to those obtained in clinical trial type settings.

METHODS

Data Source

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

Subjects

One hundred sixteen consecutive patients were evaluated and treated from August 1997 to July 1998. Eighty-two percent of the patient sample described themselves as white. The average age was 39.2 ± 17.4 and 57% were female.

Setting

The clinic is housed within an active 10-bed sleep disorders center and was staffed by three psychologists and one physician. The majority of the patients in the sample were treated by one clinician (M.S.A.), who has a Ph.D. in clinical psychology and specialized expertise in behavior therapy, behavioral sleep medicine, and neuropsychology. At the time of this program evaluation, he had completed his clinical internship (which included a rotation at the Rhode Island Hospital Insomnia Clinic) and a 1-year postdoctoral fellowship. Prior to assuming clinical responsibilities, M.S.A. was provided with a 2-week training period during which he was familiarized with the clinical protocol and provided the opportunity to discuss and/or amend the treatment regimen. M.S.A. was peer supervised on a weekly basis at the clinic case conference.

General Protocol

Patients were physician- or self-referred to the center and were triaged by telephone to the clinic. All patients underwent an extensive intake interview to review their clinical history. During this interview we determined whether (1) the patient had primary insomnia or insomnia secondary to stable medical and/or psychiatric conditions, (2) the patient was using hypnotic medications, and (3) the patient was inclined to attempt a behavioral treatment regimen.

If it was suspected that patients had unstable medical or psychiatric conditions, the primary care physicians or psychiatrists were contacted, with the patients' consent, before treatment was initiated. 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 four to nine sessions. Over the course of the first four treatment sessions, four 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; Bootzin and Perlis, 1992). Sessions 5–9 were used for extended monitoring, prolonged sleep restriction therapy, or 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 2-Week Baseline). The intake evaluation session was typically 90 to 120 min. During the session, the clinician reviewed the patient's medical, psychiatric, and sleep disorder 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, the Beck Depression Inventory (BDI), the Beck Anxiety Inventory (BAI), and a demographic questionnaire. By the end of this session, the clinician determined if a referral was required (for unstable primary medical or psychiatric problems), whether the patient needed to be withdrawn from medication, and/or whether 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 "counter-strategy." 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, i.e., 90% of the time spent in bed must be spent asleep. We did not restrict lower than 4 hr 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 as follows: (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 (1972) original formulation, we did not specify a time increment after which the patient should get out of bed and leave the bedroom. Instead we recommended 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 which 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 (1992)-style approach to "decatastrophization." 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 at 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' (1988, 1993) work on cognitive therapy for insomnia.

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 strategies 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 was made: therapy was continued for one to four sessions, a referral for a sleep study was made, or further diagnostic work was recommended.

Sleep Diaries

Our clinic used a scannable sleep diary 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 (1) prospectively to evaluate sleep disturbance complaints, (2) to tailor sleep restriction therapy to the individual, and (3) to track treatment outcome. Each week, sleep diary data were summarized at the beginning of the session. Measures of sleep latency, number of intermittent awakenings, duration of intermittent 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 compared to 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). Two sets of analyses were undertaken (absolute

change and percentage change). In both, baseline data were compared to end-of-treatment data using paired *t* tests. Bonferonni corrected *p* values are provided. The correction was calculated by multiplying the uncorrected *p* values by the number of tests in each set of analyses.

Outcome measures were change scores and percentage change, calculated individually for sleep latency (SL), number of awakenings (#WTS), duration of awakenings (WASO), total sleep time (TST), and average percentage improvement (Global). Change scores were calculated by subtracting baseline values from end-of-treatment values. Percentage improvement was calculated by determining the ratio of change from beginning to end of treatment compared to the original baseline values. Also calculated was a variable that represented overall change. This variable was constructed by averaging the percentage improvement scores for each sleep parameter. Inverse values were used for percentage change for total sleep time so that the direction of change for all parameters was constant. In addition to the percentage outcome measures, effect size values are also provided.

RESULTS

Description of Sample

One hundred sixteen consecutive patients were evaluated and treated from August 1997 to July 1998. Of these patients, 73% were diagnosed with primary insomnia, 18% with circadian rhythm disorders, and 9% with other sleep disorders (e.g., inadequate sleep hygiene). Of the 85 patients with primary insomnia, 16 patients were referred and/or elected not to undertake treatment following the intake interview, 17 patients discontinued prior to the fourth session, and 5 patients had incomplete data. Thus 47 of the original 85 patients with primary insomnia had a minimum adequate trial of therapy and complete data. The average number of sessions for this group was 8.2 ± 3.6. Comparisons between the 33 patients who did not complete treatment and the 52 patients that did (47 with and 5 without complete data) revealed that the groups did not significantly differ with respect to age, sex, race, prevalence of medical or psychiatric disorders, BDI scores, BAI scores, or marital status. The groups did, however, differ significantly with respect to some of their retrospective assessments of sleep continuity (data from Intake Sleep Questionnaire). The patients that withdrew from treatment reported a greater number of awakenings (4.0 vs 2.3; $p < 0.02$) and less total sleep time (245.5 vs 292.4; $p < 0.03$). The groups did not differ on their retrospective estimates of sleep latency or wake after sleep onset time.

Of the 47 subjects categorized using the adequate trial definition, 63% were female and the average age was 43.0 (15.6) years. Fifty-three percent reported primary psychiatric and/or medical disorders. The average BDI was 13.5 (9.1). The average BAI was 14.4 (9.9). Both scores are within the clinically significant range and represent mild depression and anxiety levels. The most common medical problems were related to gastrointestinal, musculoskeletal, and headache disorders. The most common psychiatric disorders were mood, anxiety, and attention deficit disorders. Prior to treatment, the average sleep profile as derived from the baseline sleep diary data was as follows: sleep latency = 67.0 ± 51.3 min, number of intermittent awakenings = 3.0 ± 2.0, wake time after sleep onset = 87.9 ± 53.3 min, and total sleep time = 340.4 ± 80.5 min.

Treatment Effectiveness

All pre-post comparisons for this subsample of our clinical case series were significant at $p < .01$. See Table I for details. On average, subjects who completed a minimum adequate trial were about 43% improved. This average corresponded to an average 65.4% reduction in sleep latency (effect size, 1.25), a 46.2% decrease in number of awakenings per night (effect size, 1.06), a 47.5% reduction in wake time after sleep onset (effect size, 1.42), and a 13% increase in total sleep time (effect size, 0.41). In actual minutes, patients, on average, fell asleep about 48 min more quickly, work up some 1.7 fewer times, and obtained about 33.8 min more sleep a night. When a Bonferonni correction was applied to these data (a x No. of tests per group), all of the pre-post changes remained significant at $p < .05$.

Table I. Pre–Post Changes in Sleep Parameters

Variable	Mean	*N*	SD	*t*	*p*	*A–p*[a]
		Average percentage change				
Global	43.00	47	34.40	8.60	.0001	.0008
		Change in Real numbers				
SL	–47.98	47	47.09	–6.99	.0001	.0004
#IWT	–1.67	47	2.16	–5.29	.0001	.0004
WASO	–61.14	47	46.54	–9.01	.0001	.0004
TST	+33.78	47	83.36	2.78	.0079	.0316
		Percentage change * Parameter				
SL	–65.37	47	26.2	–17.10	.0001	.0004
#IWT	–46.18	47	40.5	–7.81	.0001	.0004
WASO	–47.52	47	117.5	–2.77	.0080	.0032
TST	–13.01	47	29.3	–3.04	.0039	.0156

[a] *p* values are Bonferonni corrected for each set of analyses.

DISCUSSION

The purpose of this study was to evaluate the effectiveness of behavioral treatment as it is delivered in a clinical setting. Treatment outcome was defined using an adequate minimum trial definition. Using this formulation, 61% of patients completed therapy and exhibited significant global improvement. The most robust clinical gains were centered on sleep latency and wake after sleep onset time.

Attrition

Thirty-three of our patients (39%) were either referred or elected to discontinue treatment before obtaining an adequate trial of therapy. The question that this gives rise to is, "How does this rate of attrition compare to the drop out rate for the clinical efficicy studies?" Recently, we reviewed the behavioral literature on insomnia and found 20 studies that provided specific information on attrition (Morin *et al*., 1993, 1999; Woolfolk and McNulty, 1983; Turner and Ascher, 1979; Nicassio and Bootzin, 1974; Steinmark and Borkovec, 1974; Zwart and Lisman, 1979; McClusky *et al*., 1991; Lacks *et al*., 1983a,b; Edinger *et al*., 1992; Jacobs *et al*., 1993; Dahl *et al*., 1991; Davies *et al*., 1986; Johnson and Magaro, 1987; Guilleminault *et al*., 1995; Alperson and Biglan, 1979; Spielman *et al*., 1987a; Bliwise *et al*., 1995; Riedel *et al*., 1995). The average dropout rate of subjects receiving active treatment was 15.3% (range, 0–42%). Thus, the attrition rate in our study appears to represent the high end of the spectrum. One interpretation of why our dropout rate is higher might be that patients who are participating in research are simply more willing to accede to the demands of an experimental protocol. In contrast, clinical patients may be less willing to engage in, and *pay* for, treatment that involves

(1) discontinuing hypnotics indefinitely,
(2) engaging in and/or being billed for a form of therapy that falls under the rubric of "mental health" services (i.e., patient's may prefer medical treatment),
(3) a form of therapy that is time and labor intensive, and/or
(4) a fundamental paradox.

Regarding the last point, it is fair to characterize behavior therapy as paradoxical. After all, the core prescription is ultimately that patients with insomnia should sleep less. Not every patient can, within the clinical setting, be persuaded that the prescription is short term and that the transient worsening of insomnia symptoms ultimately serves the cure. Finally, it should be

noted that we elected to use a conservative estimate with respect to patient attrition. A proportion of the patients designated as "dropouts" may have (1) been referred because of unstable medical or psychiatric conditions or (2) discontinued therapy because of adequate gains *prior to* the fourth session. Since the data to make these distinctions were not available, the most conservative estimate was used.

Effectiveness

As noted in the Introduction, there is a variety of factors that are likely to influence treatment outcome. Some may negatively impact on treatment effectiveness; others might actually increase the likelihood of a positive outcome. Given the present findings, and the fact that they appear comparable to the treatment efficacy data, this suggests that there is a balance between the factors that detract from and those that contribute to a positive treatment outcome in the clinic setting.

With respect to specific outcomes, it is interesting to note that the parameters least affected by behavior therapy are number of awakenings and total sleep time. It may be the case that *number of awakenings* is the sleep parameter that is the most difficult for patients to estimate reliably, and thus moderate changes within this domain may be more difficult to detect. Alternatively, behavior therapy may not so much alter the number of awakenings as diminish their duration. Recent preliminary work from our group provides data that are consistent with this point of view. Using polysomnographic data, we found that patients with insomnia secondary to depression and good sleeper controls awaken during the night at a relatively fixed rate and that what distinguished patients from depressed controls was the amount of time awake following the arousal (Perlis *et al*., 1998).

As for *total sleep time*, it is not surprising to find that this parameter is the least affected by behavior therapy. The two major components of treatment (sleep restriction and stimulus control) both serve mildly to 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 insomnia are consistent with this perspective (Morin *et al*., 1994; Murtagh and Greenwood, 1995). For example, Morin and colleagues (p. 1178, Table 5) show that total sleep time increases from a post treatment value of 377.9 min to 395.0 min at follow-up.

Effect Size

The results of this study appear to be comparable with those of the meta-analytic studies on behavioral treatment for insomnia (Morin *et al*., 1994; Murtagh and Greenwood, 1995). Expressed in terms of average effect size, our results *may* be better (Perlis *et al*. = 1.04; Murtagh *et al*. = 0.66; Morin *et al*. = 0.61). This increased effect may correspond to the quality of care at our clinic or to differences related to the clinical setting, or it may simply represent a regression-to-the-mean artifact. With respect to the latter possibility, the data in the meta-analyses represent grand averages. These values are constructed by calculating means for individual samples as well as for multiple samples. Such averaging is likely to produce reliable estimates of the population mean, where data from a single study such as ours may be skewed.

FINAL COMMENTS

The strengths and the weaknesses of the present study both derive from the fact that it is based on case series and self-report data. The strength of the study is that it demonstrates that behavioral interventions, as applied in clinical settings, are effective. The weakness of the study is that it lacks objective measures (e.g., polysomnographic and/or actigraphic data) and traditional controls (e.g., a wait-list control or control treatment groups).

As for objective measures, 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) (e.g., Perlis *et al*., 1997), the issue itself may be moot in context. The present investigation is a treatment effectiveness study. The central question is, "Is behavioral treatment effective in the clinical setting?" Since prospective self-report measures constitute the standard of practice for behavioral sleep medicine, they are the appropriate measure for this type of investigation.

As for the inclusion of traditional controls, there is no doubt that this would strengthen the present study. Traditional controls would allow us to rule out the possibilities that (1) time alone produced the observed results and/or (2) nonspecific factors within our treatment regimen produced the treatment gains. These potential confounds, however, have been extensively investigated. The data from the treatment efficacy studies suggest that treatment gains are related to the active components of therapy (Morin *et al*.,

1994; Murtagh and Greenwood, 1995). Data from Mendelson (1995) convincingly suggest that patients do not simply get better with time. Thus, one may be reasonably confident that the effects observed in this study (1) are likely to be the result of the behavioral intervention and (2) appear to be equal to, or better than, those observed under controlled and experimental conditions.

Finally, as noted in the Introduction, it is important that more treatment effectiveness research is undertaken. In part, additional research will determine whether the treatment effect findings of this study are reliable. 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 as part of an American Academy of Sleep Medicine-supported multisite study on the treatment of insomnia.

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