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Treatment of Chronic Insomnia by Restriction of Time in Bed

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Summary: A treatment of chronic insomnia is described that is based on the recognition that excessive time spent in bed is one of the important factors that perpetuates insomnia. Thirty-five patients, with a mean age of 46 years and a mean history of insomnia of 15.4 years, were treated initially by marked restriction of time available for sleep, followed by an extension of time in bed contingent upon improved sleep efficiency. At the end of the 8-week treatment program, patients reported an increase in total sleep time ($p < 0.05$) as well as improvement in sleep latency, total wake time, sleep efficiency, and subjective assessment of their insomnia (all $p < 0.0001$). Improvement remained significant for all sleep parameters at a mean of 36 weeks after treatment in 23 subjects participating in a follow-up assessment. Although compliance with the restricted schedule is difficult for some patients, sleep restriction therapy is an effective treatment for common forms of chronic insomnia. **Key Words:** Chronic insomnia—Nondrug treatment—Behavioral treatment.

Insomnia, which has troubled humanity since antiquity, is one of the most common complaints made to physicians and continues to be one of the most difficult to treat (1,2). In the last few years, there has been an increased recognition of the multiplicity of different causes of insomnia, including conditions such as sleep apnea, nocturnal myoclonus, delayed sleep phase syndrome, and drug ingestion, for which specific treatments are now available (3–5). However, the most commonly occurring forms of insomnia are associated with stress, anxiety, mild depression, maladaptive conditioning, and disturbances of the sleep-wake pattern (6,7). The diagnosis of psychophysiological insomnia or a psychiatric condition associated with the insomnia accounts for the majority of these cases (7,8). While hypnotic drug therapy may play a role in management (9) and some success has been reported with behavioral treatments such as relaxation training, stimulus control, biofeedback, and sleep hygiene (see 10), the prevalence of insomnia remains high (6). While family, marital, or occupational stress, psychological conflicts, an erratic sleep schedule, and drugs or alcohol may be responsible for initiating insomnia, even after these factors have resolved the insomnia will fre-

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quently persist (11). Therefore, understanding the mechanisms that sustain the insomnia appears to be essential for effective therapy (12).

We have identified and addressed therapeutically one of the factors that we believe perpetuates insomnia, namely, excessive time in bed (TIB) (13). Methods to determine the amount of time that a patient with chronic insomnia should spend in bed have not previously been reported, nor has any study systematically varied TIB as a treatment strategy. In this report a treatment is described that involves restricting available sleep time and making changes in TIB contingent upon the patient's clinical response.

METHODS

Subjects

Patients evaluated for the complaint of insomnia between September 1982 and August 1983 were eligible for study. In addition, 11 patients with insomnia, previously evaluated and requesting further treatment, were included in the study. Each underwent the standard evaluation, which included the following: completion of a sleep log that records the daily sleep pattern over a 2-week period, a detailed medical history and physical examination by a physician sleep specialist, an unstructured interview by a clinical psychologist who is also a clinical polysomnographer (AJS), and the Minnesota Multiphasic Personality Inventory.

Of the 49 patients who agreed to participate in the study, 35 completed the 8-week treatment and constitute the subject group. Fourteen individuals who started but did not complete the study remained in the treatment for a mean of 19.4 days (SD = 10). Seven (50%) subjects completed $\leq$15 days, 5 (36%) completed between 16 and 28 days, and 2 (14%) remained in the study 32 and 37 days. Two of these 14 subjects improved so rapidly that they did not desire further treatment, and two were removed from the study because they were prescribed psychoactive medicine by their private physicians. One subject withdrew because of a severe medical problem requiring a hysterectomy. Eight subjects withdrew because of a mixture of discouragement and difficulty complying with the rigid schedule. Five of these eight subjects, who could be considered treatment failures, withdrew within 15 days of starting the treatment. One subject could not be contacted to ascertain the reasons for discontinuation.

In the sample of 35 subjects who completed the study, there were 18 men and 17 women, with a mean age of 46 years (SD = 13) and a 15.4-year (SD = 13) mean duration of the complaint of insomnia. Thirty-one patients were willing to have an all-night polysomnographic recording during which there was continuous monitoring of electroencephalogram (C4-A1A2), electrooculogram, chin and anterior tibialis electromyogram, electrocardiogram, nasal and oral air flow measured by thermistors, and bellows recording of thoracoabdominal movement. Polysomnographic records were scored in the standard manner (14). One patient's sleep study was excluded from data analysis because he could not tolerate the recording, discontinuing the study after 16 min. Patients with sleep apnea (more than five apneas per hour of sleep), a major affective disorder, or delayed sleep phase insomnia were excluded from the study. Diagnoses were made at a case conference during which all the information from the evaluation was considered.

Twelve subjects who were taking hypnotic medications nightly were told to continue this drug regimen throughout the treatment program. Likewise, two subjects using alcohol as a hypnotic, one taking amitriptyline, and one occasionally taking a minor

tranquilizer were told to continue the frequency and dosage of their drug intake throughout the study. The remaining 19 patients had been drug-free for at least 6 months and agreed to refrain from commencing hypnotic use during the duration of treatment. Based on daily log data, the 16 subjects taking some sedating substance during baseline continued this regimen at the same frequency and dosage throughout the 8-week treatment period. However, one subject not taking any sedative-hypnotic during the baseline period began taking 25 mg of amitriptyline each night starting the first week of treatment and continuing throughout the 8 weeks.

Procedure

Immediately prior to and at the end of the treatment, patients answered a 13-item insomnia symptom questionnaire. Based on the 2-week sleep log, an individualized sleep-wake schedule was prescribed for each patient as follows. The average subjective total sleep time was used to calculate the initial TIB. For example, if a patient reported a nightly average of 5 h of sleep for the 2-week baseline period, although having spent an average of 8 h in bed, then the nightly TIB prescribed at the start of treatment was 5 h. The time of awakening in the morning was established in accordance with daytime schedule needs such as habitual time of arising to get to work. Retiring time at night was set, to the nearest quarter-hour, so that the time spent in bed equaled the prescribed TIB. Regardless of the baseline total sleep time, no patient was prescribed <4.5 h TIB at the start of treatment.

Over the 8-week treatment period, subjects called a standard telephone-answering machine daily to report retiring time, out-of-bed time in the morning, and their estimate of total sleep time. Total wake time was derived from these reports. Each day sleep efficiency (estimated total sleep time/TIB × 100%) was calculated and combined with the sleep efficiency values for the previous 4 days to yield a mean for 5 days. This value served as the basis for sleep schedule changes. Throughout treatment, changes in TIB were instituted according to the following three criteria: (a) When the mean sleep efficiency over the previous 5 days was ≥90%, then the subject's TIB was increased 15 min by setting the retiring time earlier. TIB increases were always followed by at least 5 days with no change in sleep schedule. (b) When the mean sleep efficiency over the previous 5 days was <85%, the TIB was decreased. Reduction in TIB was not made for at least 10 days from the start of treatment or 10 days following any sleep schedule change. TIB was reduced to the mean sleep time of the previous 5 days. (c) If the mean sleep efficiency over the previous 5 days was <90% and ≥85%, then the TIB was not altered. The only additional sleep schedule recommendation was to prohibit lying down or napping at any time other than during the prescribed TIB. Subjects were not given any instructions regarding coffee, food, or tobacco consumption, exercise, or whether to stay in bed when they were unable to sleep.

A follow-up assessment was conducted a mean of 36 weeks (SD = 20.5, range 13.4–100.3 weeks) after the end of the treatment. The long time to follow-up in the subject studied at 100.3 weeks after treatment was unusual; the next-longest interval from the end of treatment to follow-up was 68 weeks. The variability of time to follow-up was due to subjects' availability and willingness to be reevaluated. Subjects were first asked to rate their current sleep as "better," "no different," or "worse" compared with their sleep prior to restriction therapy. Subjects called the telephone-answering machine daily and filled out sleep logs for 2 weeks. No sleep schedule was prescribed during this period.

RESULTS

Subjects

Clinical features. A combination of sleep onset and maintenance difficulties was the most common complaint (n = 18), a sleep maintenance problem was the next most frequent complaint (n = 13), and sleep onset problems the least frequent (n = 4). Utilizing the Association of Sleep Disorders nosology (8), insomnia was associated with a diagnosable psychiatric disorder in 60% of the cases (n = 13, A.2.a; n = 8, A.2.b). Approximately 34% of the cases were diagnosed as having a persistent psychophysiological insomnia (n = 12, A.1.b). In two subjects an irresistible urge to move the legs while lying awake in bed and periodic movements while asleep were present (A.5.a, b). These two subjects were included for a number of reasons. First, while their primary diagnosis suggested that organic conditions were producing the insomnia, their secondary diagnoses of psychophysiological insomnia and insomnia associated with an affective disorder suggested conditioning and emotional factors were playing some role. Second, both patients had been previously evaluated and treated with medications at other sleep disorders centers with limited success. Therefore, these two subjects were included because we reasoned that a behavioral approach offered some hope of alleviating factors that had not been previously addressed.

Polysomnography. Thirty subjects had a polysomnographic recording prior to treatment to screen for other sleep disorders. Sleep efficiency, a common measure of overall sleep difficulty, was 69%. Mean sleep latency for the group, defined as the time from retiring to the first 10 min of consolidated sleep, was 46 min.

On the morning following the polysomnographic study, patients made subjective estimates of sleep latency and total sleep time. The nighttime polysomnographic record was compared and paired with the morning subjective report for total sleep time (n = 30) and sleep latency (n = 25) using a two-tailed t test and the Pearson product-moment correlation. Sleep latency was based on an n of 25 because one subject refused to make an estimate and the subjective sleep latency was indeterminate in four subjects who reported "no sleep." Compared with the polygraphic recording, patients underestimated total sleep time by a mean of 58 min (SD = 109; $t[29] = 2.90$, $p < 0.01$). Subjects' estimate of sleep latency ($\bar{x}$ = 77 min, SD = 81) was significantly greater than the recorded value ($\bar{x}$ = 42 min, SD = 56; $t[24] = -2.86$, $p < 0.01$). These differences are consistent with those in previous studies (15,16). Although the absolute levels were different, there was a significant association between recorded and reported total sleep time ($r = 0.66$, $t[28] = 4.65$, $p < 0.01$) and sleep latency ($r = 0.66$, $t[23] = 4.21$, $p < 0.01$). These findings that subjective reports reflect the degree of polygraphically measured sleeping difficulty in insomniacs are consistent with those in previous studies (16–18).

Treatment

Reported sleep parameters. A repeated measures analysis of variance with one main factor, condition [baseline (pretreatment) and end of treatment (posttreatment)], was performed on reported TIB, sleep latency, total wake time, total sleep time, and sleep efficiency. Baseline condition was based on 14 days and the end-of-treatment condition on the last 7 days of treatment. On the first night of the treatment, subjects' TIB was restricted to a mean prescribed TIB of 339 min (SD = 65), which represents a reduction of 140 min (SD = 63) from the mean TIB of 479 min (SD = 49) reported in the baseline sleep log ($t[34] = 13.14$, $p < 0.0001$). Sleep latency data were collected from

the sleep log and not reported on the telephone-answering machine. Three subjects did not submit sleep logs for a portion of the baseline or last week of treatment, and therefore the analysis of sleep latency was based on 32 subjects on whom data were available. Mean sleep latency for the group decreased from a baseline value of 48 to 19 min by the end of treatment ($t[31] = 4.81$, $p < 0.0001$) (Table 1). Mean total wake time was sharply decreased at the start of treatment, remained low throughout the 8-week treatment period (Fig. 1), and was reduced by 109 min at the end of treatment ($t[34] = 7.31$, $p < 0.0001$). Sleep efficiency was markedly improved at the onset of treatment and remained elevated, showing a mean increase of 20% by the last week of treatment ($t[34] = -7.05$, $p < 0.0001$). At the end of treatment, mean TIB was 393 min, a reduction of 85 min from baseline ($t[34] = 8.28$, $p < 0.001$).

Total sleep time increased from a pretreatment mean of 320 min to 343 min post treatment ($t[34] = -2.48$, $p < 0.05$). Linear trend was assessed by a Pearson product-moment correlation performed on pairs of total sleep time and treatment day number across 56 days of treatment. Initially total sleep time was reduced below baseline values; however, by the fourteenth day of treatment, it had increased to the pretreatment level and steadily increased throughout treatment ($r[54] = 0.81$, $p < 0.01$) (Fig. 1). Ten of the 35 subjects had at least one nap during the 2-week baseline period. This group of subjects napped a total of 92 times over the 14 pretreatment days, with a mean nap duration of 26.5 min (SD = 24). In the last week of treatment, only 2 of these 10 subjects napped a total of 15 times, with a mean nap duration of 13.3 min (SD = 9). To assess if the substantial decrease in napping may have accounted for the increase in nocturnal total sleep time by the end of treatment, we added the daytime to the nighttime sleep in this subgroup of subjects. Based on this recalculation, the mean total sleep time for the entire group of 35 subjects was 324 min (SD = 90) during baseline and 344 min (SD = 64) during the last week of treatment. A two-tailed paired t test showed that the mean increase of 20 min (SD = 55) was statistically significant ($t[34] = -2.06$, $p < 0.05$).

Variability. The difference between baseline and end-of-treatment night-to-night

TABLE 1. *Reported sleep parameters during the 2 weeks of baseline (pre treatment) and the last week of treatment (post treatment) for 35 subjects*

Sleep parameter		Pre treatment	Post treatment
Time in bed	$\bar{x}$	479	393[b]
	SD	49	63
Sleep latency[a]	$\bar{x}$	48	19[c]
	SD	42	14
Total wake time	$\bar{x}$	159	50[c]
	SD	89	16
Total sleep time	$\bar{x}$	320	343[d]
	SD	89	63
Sleep efficiency	$\bar{x}$	67	87[c]
	SD	18	5

Values are given as minutes, except for sleep efficiency, which is a percentage calculated as follows: total sleep time (min)/time in bed (min) × 100%.

[a] Sleep latency based on n = 32.

[b] $p < 0.001$.

[c] $p < 0.0001$.

[d] $p < 0.05$.

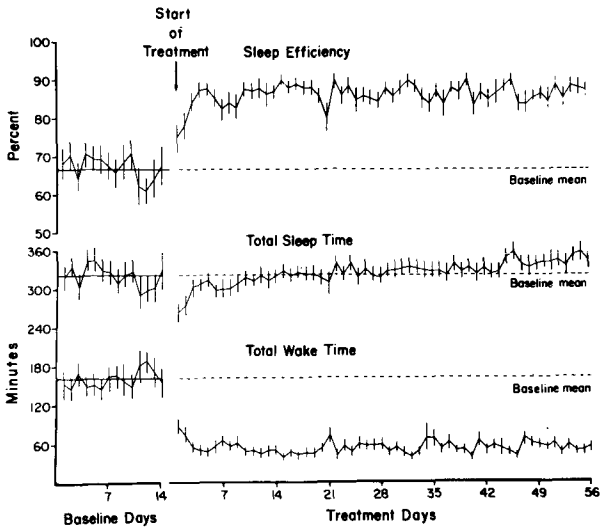

FIG. 1. Mean (±SEM) subjective estimate of nightly total wake time, total sleep time, and sleep efficiency during baseline and treatment periods for 35 subjects.

variability for sleep latency, total wake time, total sleep time, and sleep efficiency was tested as follows: Each subject's SD for each sleep parameter was determined separately across the 14-day baseline period and the last 7 days of treatment. The SDs were normally distributed. The means of these SDs were tested by a two-tailed paired t test. The variability, as measured by the night-to-night SD, of total sleep time was significantly reduced from baseline ($\bar{x}$ = 80.5, SD = 33) to the end of treatment ($\bar{x}$ = 40.8, SD = 22; $t[34] = 6.30$, $p < 0.0001$). The variability of sleep efficiency was also significantly reduced from baseline ($\bar{x}$ = 15.1, SD = 7) to the end of treatment ($\bar{x}$ = 9.3, SD = 6; $t[34] = 5.54$, $p < 0.0001$). Likewise, the variability of total wake time was significantly reduced from baseline ($\bar{x}$ = 75.0, SD = 34) to the end of treatment ($\bar{x}$ = 35.2, SD = 19; $t[34] = 7.51$, $p < 0.0001$), as was that of sleep latency, from baseline ($\bar{x}$ = 37.8, SD = 33) to the end of treatment ($\bar{x}$ = 13.0, SD = 15; $t[31] = 5.05$, $p < 0.0001$).

Insomnia symptom questionnaire. We began administering the insomnia symptom questionnaire after seven subjects had already begun treatment. Therefore, only 28 subjects completed the questionnaire (a) prior to treatment, (b) each time they met the first criterion, and (c) at the end of treatment. The response to each item was "rarely," "sometimes," or "frequently" and given values of 1, 2, or 3, respectively. The questionnaire was analyzed using a Hotelling T^2 statistic. The post hoc tests on individual questions were performed using the Bonferroni criterion. Analysis of the symptom questionnaire revealed an overall improvement ($T^2 = 192.55$, distributed as $F[13,15] = 8.23$, $p < 0.0001$), and individual analysis of each item showed significant improvement (Fig. 2) on all but two questions. At the end of treatment, all 35 subjects compared their sleep problem with the start of treatment and 30 (86%) reported their insomnia was "better," 5 (14%) reported it was "no different," and none reported it was "worse." Adding the 10 dropouts that were either subjectively improved (n = 2) or discouraged (n = 8) to the 35 subjects that completed the treatment, the global subjective response of 32 (71%) subjects was that their insomnia was improved while 13 (29%) reported it was not improved.

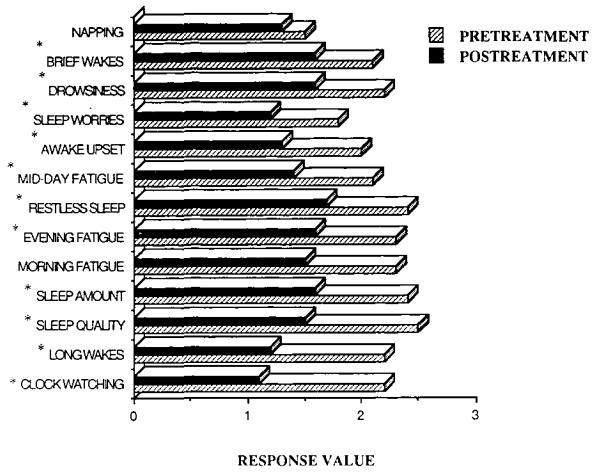

FIG. 2. Insomnia symptom questionnaire asked prior to treatment and at the end of treatment for 28 subjects. Lower values indicate better sleep or functioning. An asterisk indicates $p < 0.05$.

Subgroup analysis. An important relationship to bear in mind when evaluating the differential effects of sleep restriction in subgroups is the strong association between the degree of improvement in a sleep parameter and the initial level of that parameter. For example, the improvement (end of treatment minus baseline) in reported sleep latency is highly correlated with the baseline sleep latency ($r[30] = +0.95$, $p < 0.0001$). This relationship of degree of improvement with initial level also holds for total wake time ($r[33] = +0.98$, $p < 0.0001$), total sleep time ($r[33] = -0.71$, $p < 0.0001$), and sleep efficiency ($r[33] = -0.95$, $p < 0.0001$). Therefore, when subgroups differ in the severity of a particular sleep parameter in baseline, this difference may explain the degree of improvement with treatment.

The 12 subjects with psychophysiological insomnia showed the same pattern of change as the entire sample, with reported improvement at the end of treatment in sleep latency (baseline $\bar{x} = 51.9$ min, SD = 39; end of treatment $\bar{x} = 17.9$ min, SD = 12; $t[9] = 3.43$, $p < 0.01$), total wake time (baseline $\bar{x} = 164.6$ min, SD = 97; end of treatment $\bar{x} = 52.0$ min, SD = 19; $t[11] = 4.18$, $p < 0.005$), and sleep efficiency (baseline $\bar{x} = 66.1\%$, SD = 18; end of treatment $\bar{x} = 86.3\%$, SD = 7; $t[11] = -4.23$, $p < 0.005$). The increase in total sleep time approached but did not reach statistical significance (baseline $\bar{x} = 317.0$ min, SD = 85; end of treatment $\bar{x} = 353.5$ min, SD = 68; $t[11] = -1.95$, $p < 0.08$).

The 21 subjects with insomnia associated with a psychiatric disorder also reported improvement in sleep latency (baseline $\bar{x} = 44.1$ min, SD = 46; end of treatment $\bar{x} = 21.0$ min, SD = 15; $t[19] = 2.91$, $p < 0.01$), total wake time (baseline $\bar{x} = 145.6$ min, SD = 80; end of treatment $\bar{x} = 48.8$ min, SD = 15; $t[20] = 5.45$, $p < 0.0001$), and sleep efficiency (baseline $\bar{x} = 68.2\%$, SD = 18; end of treatment $\bar{x} = 86.6\%$, SD = 4; $t[20] = -4.91$, $p < 0.0005$). However, this group of subjects showed little change in total sleep time (baseline $\bar{x} = 326.4$ min, SD = 95; end of treatment $\bar{x} = 339.3$ min, SD = 64; $t[20] = -1.13$, $p < 0.27$).

The two subjects with restless legs syndrome and periodic movements in sleep also improved with treatment. Sleep latency and total wake time were reduced from baseline means of 64.3 and 212.7 min to end-of-treatment means of 5.4 and 35.4 min, re-

spectively. Mean total sleep time was increased by 61.6 min and sleep efficiency increased by 36.1%.

The 18 subjects taking no sedating drug at any time during the study reported improvement on sleep latency (baseline $\bar{x}$ = 35.3 min, SD = 35; end of treatment $\bar{x}$ = 13.3 min, SD = 11; $t[14] = 2.87$, $p < 0.05$), total wake time (baseline $\bar{x}$ = 135.4 min, SD = 76, end of treatment $\bar{x}$ = 43.1 min, SD = 12; $t[17] = 4.95$, $p < 0.0005$), and sleep efficiency (baseline $\bar{x}$ = 71.6%, SD = 15; end of treatment $\bar{x}$ = 88.9%, SD = 2; $t[17] = -4.90$, $p < 0.0005$). The increase in total sleep time approached but did not reach statistical significance (baseline $\bar{x}$ = 333.9 min, SD = 42; end of treatment $\bar{x}$ = 357.1 min, SD = 15; $t[17] = -1.93$, $p < 0.07$).

The same pattern of results was obtained in the 17 subjects taking some type of sedating drug during the baseline or treatment. Improvement was reported on sleep latency (baseline $\bar{x}$ = 58.8 min, SD = 46; end of treatment $\bar{x}$ = 24.0 min, SD = 15; $t[16] = 3.87$, $p < 0.005$), total wake time (baseline $\bar{x}$ = 183.7 min, SD = 97; end of treatment $\bar{x}$ = 55.5 min, SD = 18; $t[16] = 5.45$, $p < 0.0005$), and sleep efficiency (baseline $\bar{x}$ = 61.4%, SD = 19; end of treatment $\bar{x}$ = 84.4%, SD = 7; $t[16] = -5.14$, $p < 0.0005$). Similarly, the increase in total sleep time approached but did not reach statistical significance (baseline $\bar{x}$ = 303.6 min, SD = 103; end of treatment $\bar{x}$ = 328.1 min, SD = 79; $t[16] = -1.57$, $p < 0.13$).

Follow-up assessment

At the start of the follow-up, 14 subjects reported that their sleep was "better" as compared with the start of treatment, 8 reported it was "no different," and 1 reported it was "worse." Two planned comparisons were performed using paired *t* tests to analyze the differences between baseline and the end of treatment and between baseline and follow-up for all reported sleep parameters (Table 2). Subjects cut 74 min from their habitual TIB by the last week of treatment ($t[22] = -5.75$, $p < 0.0001$). Following treatment, subjects increased their TIB somewhat, but it remained 39 min below the

TABLE 2. *Reported sleep parameters during the 2 weeks of baseline (pre treatment), the last week of treatment (post treatment), and the 2 weeks of follow-up for 23 subjects*

Sleep parameter		Pre treatment	Post treatment	Follow-up
Time in bed	$\bar{x}$	476	402[b]	437[b]
	SD	46	62	51
Sleep latency[a]	$\bar{x}$	53	17[c]	31[d]
	SD	52	14	38
Total wake time	$\bar{x}$	156	48[b]	87[b]
	SD	95	15	60
Total sleep time	$\bar{x}$	320	353[e]	350[e]
	SD	96	63	80
Sleep efficiency	$\bar{x}$	68	88[b]	80[c]
	SD	19	5	14

Values are given as minutes, except for sleep efficiency, which is a percentage calculated as follows: total sleep time (min)/time in bed (min) × 100%. Pretreatment means are statistically compared with posttreatment and follow-up means.

[a] Sleep latency based on n = 21.

[b] $p < 0.0001$.

[c] $p < 0.001$.

[d] $p < 0.005$.

[e] $p < 0.05$.

baseline levels at follow-up ($t[22] = -4.59$, $p < 0.0001$). Compared with baseline total sleep time ($\bar{x} = 320$ min), the significant increase at the end of treatment ($\bar{x} = 353$ min; $t[22] = 2.40$, $p < 0.05$) was maintained at follow-up ($\bar{x} = 350$ min; $t[22] = 2.16$, $p < 0.05$). Total wake time was reduced by 108 min at the end of treatment ($t[22] = -5.33$, $p < 0.0001$) and by 69 min at the later reassessment ($t[22] = -4.74$, $p < 0.0001$). Sleep efficiency improved by 20% by the last week of treatment ($t[22] = 5.37$, $p < 0.0001$), and remained 12% higher than pretreatment levels at the time of follow-up ($t[22] = 4.24$, $p < 0.001$). Sleep latency showed the same pattern with a smaller reduction of 22 min at follow-up ($t[20] = -3.30$, $p < 0.005$) as compared with the reduction of 36 min at the end of treatment ($t[20] = -3.92$, $p < 0.001$).

We performed an analysis to determine if the mild relapse in sleep at the time of follow-up could be accounted for by increases in TIB, one of the mechanisms we have hypothesized is responsible for the perpetuation of insomnia. A Pearson product-moment correlation was performed between the change in TIB and the change in other sleep parameters (change was calculated as the value at follow-up minus the value at the end of treatment). The change in TIB from the end of treatment to follow-up was directly correlated with change in total wake time ($r = 0.78$, $t[21] = 5.70$, $p < 0.0001$) and sleep latency ($r = 0.59$, $t[19] = 3.05$, $p < 0.005$) and inversely correlated with sleep efficiency ($r = -0.71$, $t[21] = -4.58$, $p < 0.0005$). Change in TIB was not related to change in total sleep time ($r = 0.04$, $t[21] = 0.18$, NS).

To determine if the variability in the time to the follow-up assessment could account for the changes seen at follow-up, we performed a Pearson product-moment correlation between time to follow-up and changes in total sleep time or sleep efficiency (calculated as the value at follow-up minus the value at the end of treatment). The time to follow-up was not related to the change in either total sleep time ($r = 0.15$, $t[21] = 0.70$, NS) or sleep efficiency ($r = -0.11$, $t[21] = -0.51$, NS).

DISCUSSION

Most current theories of insomnia emphasize factors that precipitate insomnia and characteristics that predispose individuals to develop a sleep disturbance. In contrast, the present approach is guided by the idea that addressing the factors that perpetuate chronic insomnia is essential for therapeutic success (12). Sleep restriction therapy assumes that excessive TIB is one important factor that sustains insomnia although it may not have initiated the sleep disturbance.

The mild sleep loss produced at the beginning of sleep restriction therapy may be crucial for its effectiveness. The partial sleep deprivation may have consolidated sleep directly (19,20), produced daytime fatigue that dampened the insomniacs' chronic state of hyperarousal (21,22), or reduced maladaptive conditioning because less time was spent lying awake in bed (23). In this regard it should be noted that sleep loss also occurs at the start of stimulus control therapy (10) and may be a factor responsible for the success of that treatment.

Repeatedly experiencing the irregularity and unpredictability of sleep heightens the insomniac's worried anticipation of the upcoming night's sleep (24). Getting into bed early, staying in bed late, and napping are short-sighted strategies that foster the fluctuations in the distribution and amounts of sleep and waking that are characteristic of the sleep of insomniacs (2,25). The present approach of restricting TIB stabilizes sleep. The upper limit on the TIB precludes the opportunity for increased sleep as well as

producing a mild sleep loss at the start of treatment that eliminates the nights with little sleep. Sleep becomes regular and predictable, which reduces the disruptive effects of anticipatory anxiety and may play a role in the favorable outcome.

Sleep efficiency, used as the criterion for making changes in TIB, is calculated by taking into account wake time that occurs both before and after sleep onset. This has the advantage of using a measure that reflects difficulty in falling asleep as well as difficulty staying asleep and is therefore generally applicable to patients with different insomnia complaints. In addition, high sleep efficiency is recognized as one aspect of satisfactory sleep and is commonly used as an outcome measure in treatment studies of insomnia. In the present treatment, titration of TIB is made contingent on the patient's self-report of sleep efficiency. Therefore, only when a patient perceives he or she is getting consolidated sleep with minimal amounts of wakefulness is the time allowed in bed increased. This procedure makes the therapeutic process self-paced and based on the patient's perception of improvement and results in high sleep efficiency consistently maintained throughout the 8 weeks of treatment, which may be another factor contributing to the clinical improvement. In line with this reasoning, studies of chronic insomniacs and normal subjects repeatedly briefly aroused from sleep have concluded that the complaints associated with insomnia and the daytime impairment may not be due to the sleep loss itself but may be the result of nocturnal wakefulness and the fragmentation of sleep resulting from the multiple interruptions (15,16,26,27). Therefore, the clinical efficacy of the current approach may be more related to the reduction of nocturnal wakefulness, which was considerable, than to the increase in total sleep time, which was modest.

At the start of treatment, the two main problems that occurred were difficulty adhering to the prescribed schedule and the effects of sleep loss. First, the struggle to stay awake until the scheduled bedtime was more of a problem than getting up at the scheduled time. Finding an appropriate activity late at night was not easy. Patients complained that they were too tired and sleepy to attempt demanding tasks such as writing and that more passive endeavors such as watching TV were soporific. Second, even though patients were told that they should anticipate mild sleep loss and fatigue, some became alarmed that they felt worse at the start of treatment. They worried that they would not be able to tolerate the fatigue and would be unable to perform on their jobs. As a result of these two problems, a substantial number of patients became discouraged and some dropped out in the first 2–3 weeks of treatment.

The similarities in outcome were more prominent than any differences between the diagnostic groups and between the subjects taking some sedating substance compared with those who were not using drugs. More impressive was the strong association between the baseline level of each reported sleep parameter and the degree of improvement with treatment. The chronic insomniacs whose sleep was more subjectively disturbed prior to treatment benefited most from sleep restriction therapy. The extent to which this law of initial values is a general characteristic of all treatments or more in accord with the present approach awaits the results of further comparative studies.

In addition to the short-term efficacy present at the end of treatment, the improvement maintained in all sleep parameters as well as the subjective rating of sleep at the time of the follow-up demonstrates the long-term efficacy of sleep restriction therapy. Consistent with our hypothesis of the importance of TIB in chronic insomnia is the finding that the degree to which TIB was increased following treatment was related to the degree to which sleep deteriorated. While the results of the present treatment are

consistent with the role of excessive TIB as a mechanism that sustains insomnia, this model of insomnia needs to be tested more directly. Furthermore, what constitutes an excessive amount of TIB for an individual may vary. For example, in a particular individual the same amount of TIB may be either excessive when an insomnia is present or just right when there is no current sleeping problem.

Polygraphic documentation of the sleep parameter changes during the course of treatment would have added convergent validity to the clinical improvement. However, we felt that the subjective response in the patients' own environment was initially the most essential component of a successful treatment. Having established subjective improvement, future studies should include control group comparisons and polysomnographic assessment.

In conclusion, we have described a treatment of chronic insomnia that is based upon restricting TIB. This treatment is effective in several diagnostic categories of insomnia patients and is relatively easy to implement.

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