Training Subjective Insomniacs to Accurately Perceive Sleep Onset

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Summary: Subjective insomniacs overestimate sleep latency at the beginning of their nocturnal sleep period. It was hypothesized that subjective insomniacs could be trained to accurately estimate sleep latency by learning to differentiate wakefulness from sleep. Ten subjective insomniacs were randomly assigned to one of two groups. Group 1 subjects participated in both a control and a training week; group 2 subjects participated only during a training week. Each week consisted of a baseline lab night, a training lab night (treatment or control), a home (unmonitored night) and a recovery lab night. During training, subjects were taught to use sleep markers (A, B or C) to help them more accurately estimate sleep latency and were given feedback about the accuracy of their estimates. Marker A corresponded to an electroencephalographic level of wakefulness; marker B corresponded to the initial sleep spindle; marker C corresponded to 5 minutes of continuous sleep after the first sleep spindle. In the control condition, subjects had no feedback and were not taught to use markers to help them judge sleep from wakefulness. Total sleep time and percent stage 3 sleep increased, and objective sleep latency decreased on recovery nights. After training, subjective sleep latency, correctness of estimates of sleep versus wakefulness and perceived ability to fall asleep significantly improved. This study helps to establish that subjective insomniacs can learn to more accurately estimate sleep from wakefulness with the use of sleep-wake markers. Key Words: Sleep—Sleep disorders—Insomnia—Subjective insomnia—Behavior.

Subjective insomniacs characteristically overestimate the time taken to fall asleep in the absence of identifiable psychopathology and despite a normal electroencephalographic (EEG) sleep latency and sleep pattern (1). A literature review by Moore (2) indicated that subjective insomniacs overestimated nocturnal sleep latency by an average of 42.8 minutes (p. 16). In sharp contrast to insomniacs, normal sleepers were quite accurate in their estimations of sleep latency; normal sleepers overestimated sleep latency by an average of only 1.4 minutes.

Increased cognition around sleep onset for subjective insomniacs may serve to make the distinction between sleep and wake more cloudy (3–10) and may lead to misperception about when sleep begins. Most studies that have examined this issue have used subjects with objectively verifiable sleep latency difficulty. Thus, few data exist to directly support a relation between increased sleep onset cognition and subjective insomnia.

Although few data exist to support a role for increased cognition in sleep-wake misperception, normal sleepers have been taught to discriminate between sleep states. After receiving feedback regarding the sleep stage they were in after awakening, normal sleepers improved in their discrimination of stage 1 and stage 2 sleep (11). Antrobus and Antrobus (12) found that three normal sleepers were able to correctly discriminate between sleep stages when they were encouraged to identify stage 1—rapid eye movement (REM) sleep as A and stage 2 sleep as B.

The present study was an attempt to train subjective insomniacs to accurately detect sleep onset. The training involved the provision of sleep markers to help them estimate sleep onset latency more accurately and gain feedback about their accuracy at estimating sleep from wake. It was hypothesized that 1) training normalizes sleep latency estimates (<30 minutes); 2) training augments subjective insomniacs’ perceived efficacy for falling asleep; 3) training changes presleep...
TABLE 1. Percentage of subjects who were screened for the study by stage of entry

<table>
<thead>
<tr>
<th>Stage</th>
<th>% Who failed at each stage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Phone in</td>
<td>28.5% (10/35)</td>
</tr>
<tr>
<td>MMPI</td>
<td>28.0 (7/25)</td>
</tr>
<tr>
<td>Lab screen</td>
<td>25.7 (7/28)</td>
</tr>
<tr>
<td>Completed study</td>
<td>09.0 (1/11)</td>
</tr>
</tbody>
</table>

* Number failed/total number of subjects remaining; cumulative total.
* Of those rejected at the lab screen stage, 25% were found to have over 100 leg movements, 25% met the criteria for psychophysiological insomnia and 20% had subjective estimates of sleep latency > 30 minutes or had a subjective to objective sleep latency ratio of < 1.5.
* One subject completed, but data on her were not used.

cognition; and 4) training normalizes the ratio of subjective to objective sleep onset latency (< 1.5).

METHODS

Subjects

Ten subjects, seven females and three males, completed the study. Accepted subjects met the screening criteria outlined below.

Subject recruitment and screening criteria

Accepted subjects were required to be between 18 and 40 years of age and to report chronic insomnia (at least 1 year) with sleep latencies exceeding 30 minutes at least four times per week. Sleep histories were used to help screen for sleep disorders and the Minnesota Multiphasic Personality Inventory (MMPI) was used to screen for psychopathology.

Exclusion criteria

Subjects were excluded if they 1) had psychopathology as determined by personal interview and MMPI scores (MMPI scores greater than 97th percentile were exclusionary, except on scales MF and Pd); 2) used hypnotic medications (more than three times per week), central nervous system (CNS) stimulants, CNS depressants or psychotropic medications; or 3) reported uncontrolled medical disorders such as chronic pain, stroke, CNS infection or trauma, multiple sclerosis, Parkinson's disease, muscular and peripheral nerve problems, metabolic diseases, diabetes mellitus, heart disease or gastrointestinal disorders. Those subjects who met the above criteria were then scheduled to complete one night of polysomnography to further assess their sleep.

Sleep laboratory inclusion criteria

Subjects were accepted if they had 1) no other sleep disorder apparent by polysomnography, including alpha–delta sleep; 2) a sleep latency report of greater than 30 minutes upon awakening in the morning following the adaptation night; 3) a subjective to objective sleep latency ratio > 1.5, with sleep latency defined as the first epoch of stage 1; and 4) a sleep efficiency > 85%.

Eleven subjects ultimately met the inclusion criteria, and data from 10 subjects were used in the data analysis; one subject was sleepless during the training night, and these data could not be used. The frequency of rejection at each screening stage is outlined in Table 1.

Design

Subjects were randomly assigned to one of two groups (see Table 2). Group 1 subjects participated in both control and training, with the training week always following the control week. Treatment or training consisted of feedback about sleep–wake and rule (A,B,C) estimations. The subjects in group 1 are labeled as group 1 control (G1C) and group 1 treatment (G1T), respectively, for the two conditions in the study. Group 2 subjects only served in the treatment condition (it was clearly not possible for a control week to follow the treatment week); the subjects in this group were labeled group 2 treatment (G2T). G1T and G1C means were analyzed using a 2 × 2 repeated measures ANOVA. Repeated measures factors included: condition (treatment vs. control), night (baseline vs. recovery) and the interaction between condition and night. A second series of analyses was completed to help rule out adaptation effects. A 2 × 2 ANOVA was used with

TABLE 2. Basic design of the study proper

<table>
<thead>
<tr>
<th>Night*</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
<th>7</th>
<th>8</th>
<th>9</th>
<th>10</th>
<th>11</th>
<th>12</th>
<th>13</th>
<th>14</th>
</tr>
</thead>
<tbody>
<tr>
<td>Group 1</td>
<td>A</td>
<td>H</td>
<td>BL</td>
<td>D</td>
<td>H</td>
<td>R</td>
<td>H</td>
<td>H</td>
<td>H</td>
<td>H</td>
<td>BL</td>
<td>DT</td>
<td>H</td>
<td>R</td>
</tr>
<tr>
<td>Group 2</td>
<td>A</td>
<td>BL</td>
<td>DT</td>
<td>H</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>R</td>
<td>R</td>
</tr>
</tbody>
</table>

* A = adaptation night; H = night spent at home unmonitored; BL = baseline, undisturbed sleep; D = disruption night without feedback as to accuracy of subject's sleep–wake estimations (control); R = recovery night, undisturbed sleep; DT = disruption night with feedback as to accuracy of subject's sleep–wake estimations (treatment). Control week (group 1 only) is nights 2–6 shown above. Treatment week (groups 1 and 2) is nights 10–14 above.
TABLE 3. Sleep stage and sleep latency estimation data

<table>
<thead>
<tr>
<th>Variable</th>
<th>G1C Baseline</th>
<th>G1C Recovery</th>
<th>GIT Baseline</th>
<th>GIT Recovery</th>
<th>G1C vs. GIT F (cond)</th>
<th>G1C vs. GIT F (nt)</th>
<th>G2T Baseline</th>
<th>G2T Recovery</th>
<th>G1T vs. G2T F (cond)</th>
<th>G1T vs. G2T F (nt)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total sleep recording time</td>
<td>289.0 (66.1)</td>
<td>339.4 (28.6)</td>
<td>325.2 (44.9)</td>
<td>342.0 (34.0)</td>
<td>1.31</td>
<td>3.21</td>
<td>296.8 (57.6)</td>
<td>390.7 (42.6)</td>
<td>0.22</td>
<td>5.80*</td>
</tr>
<tr>
<td>Sleep efficiency</td>
<td>83.3 (13.3)</td>
<td>90.7 (6.5)</td>
<td>85.8 (8.5)</td>
<td>93.7 (3.4)</td>
<td>0.24</td>
<td>2.76</td>
<td>78.1 (19.4)</td>
<td>93.3 (5.2)</td>
<td>0.90</td>
<td>4.12</td>
</tr>
<tr>
<td>% stage 1</td>
<td>7.0 (3.5)</td>
<td>6.5 (4.7)</td>
<td>8.2 (5.1)</td>
<td>7.0 (3.6)</td>
<td>1.89</td>
<td>0.38</td>
<td>8.2 (4.2)</td>
<td>7.9 (3.2)</td>
<td>0.02</td>
<td>0.29</td>
</tr>
<tr>
<td>% stage 2</td>
<td>40.8 (17.9)</td>
<td>45.7 (6.9)</td>
<td>49.3 (12.6)</td>
<td>53.4 (1.2)</td>
<td>6.50</td>
<td>0.78</td>
<td>34.8 (9.9)</td>
<td>43.3 (7.6)</td>
<td>0.58</td>
<td>2.18</td>
</tr>
<tr>
<td>% stage 3</td>
<td>4.2 (3.2)</td>
<td>3.4 (2.6)</td>
<td>3.9 (1.4)</td>
<td>3.5 (2.5)</td>
<td>0.50</td>
<td>12.07*</td>
<td>4.7 (2.8)</td>
<td>4.7 (3.8)</td>
<td>0.28</td>
<td>0.09</td>
</tr>
<tr>
<td>% stage 4</td>
<td>17.4 (6.7)</td>
<td>17.7 (8.2)</td>
<td>12.9 (9.5)</td>
<td>15.8 (8.5)</td>
<td>1.80</td>
<td>0.96</td>
<td>17.9 (5.4)</td>
<td>16.9 (9.7)</td>
<td>0.38</td>
<td>0.13</td>
</tr>
<tr>
<td>% stage REM</td>
<td>14.7 (4.2)</td>
<td>17.8 (5.2)</td>
<td>13.3 (6.2)</td>
<td>13.9 (3.4)</td>
<td>2.55</td>
<td>1.00</td>
<td>12.4 (6.6)</td>
<td>19.9 (8.0)</td>
<td>0.41</td>
<td>5.11</td>
</tr>
<tr>
<td>Latency REM</td>
<td>172.8 (76.7)</td>
<td>126.3 (126.4)</td>
<td>120.6 (56.5)</td>
<td>131.0 (59.5)</td>
<td>3.08</td>
<td>0.15</td>
<td>213.8 (113.1)</td>
<td>171.1 (78.4)</td>
<td>1.89</td>
<td>0.37</td>
</tr>
<tr>
<td>Wake in sleep</td>
<td>58.3 (46.7)</td>
<td>35.0 (25.0)</td>
<td>45.7 (25.7)</td>
<td>23.1 (13.9)</td>
<td>0.39</td>
<td>3.38</td>
<td>92.3 (89.5)</td>
<td>28.3 (21.3)</td>
<td>1.12</td>
<td>3.42</td>
</tr>
<tr>
<td>No. awakenings</td>
<td>16.7 (7.1)</td>
<td>20.2 (6.9)</td>
<td>20.2 (5.8)</td>
<td>16.8 (4.0)</td>
<td>9.09</td>
<td>0.09</td>
<td>12.4 (2.6)</td>
<td>18.2 (9.0)</td>
<td>0.34</td>
<td>0.62</td>
</tr>
<tr>
<td>% movement</td>
<td>0.00 (0.0)</td>
<td>0.1 (0.2)</td>
<td>0.02 (0.2)</td>
<td>0.0 (0.0)</td>
<td>0.00</td>
<td>3.64*</td>
<td>0.0 (0.0)</td>
<td>0.3 (8.4)</td>
<td>0.45</td>
<td>0.45</td>
</tr>
<tr>
<td>Stage changes</td>
<td>90.7 (18.2)</td>
<td>120.0 (45.4)</td>
<td>111.2 (20.2)</td>
<td>104.2 (31.7)</td>
<td>1.40</td>
<td>2.47</td>
<td>89.4 (31.0)</td>
<td>108.4 (26.5)</td>
<td>0.26</td>
<td>0.67</td>
</tr>
<tr>
<td>Sleep latency</td>
<td>17.6 (15.5)</td>
<td>14.5 (7.7)</td>
<td>25.7 (18.0)</td>
<td>16.0 (13.0)</td>
<td>1.36</td>
<td>5.49</td>
<td>26.4 (12.4)</td>
<td>9.9 (1.2)</td>
<td>0.16</td>
<td>6.65*</td>
</tr>
<tr>
<td>Latency estimate</td>
<td>82.0 (66.0)</td>
<td>64.0 (55.8)</td>
<td>67.0 (81.9)</td>
<td>27.4 (19.6)</td>
<td>7.80</td>
<td>4.21</td>
<td>81.0 (80.3)</td>
<td>23.0 (4.0)</td>
<td>0.10</td>
<td>2.68</td>
</tr>
<tr>
<td>Sleep latency ratio</td>
<td>5.5 (2.4)</td>
<td>3.9 (1.6)</td>
<td>2.5 (1.3)</td>
<td>2.1 (0.9)</td>
<td>11.81</td>
<td>9.57*</td>
<td>2.9 (1.8)</td>
<td>2.4 (0.6)</td>
<td>0.40</td>
<td>0.67</td>
</tr>
</tbody>
</table>

* G1C = group 1 control; GIT = group 1 treatment; G2T = group 2 treatment; standard deviations in parentheses. Wake in sleep measured in minutes.
+ p ≤ 0.05.

a repeated measures factor for night (baseline vs. recovery night), and a between-subjects factor for condition (GIT vs. G2T).

After training nights, subjects were allowed to sleep at home for a night to recover from any sleep loss effects (e.g. sleepiness) that might have occurred during the training procedure. This was followed by an undisturbed night of sleep in the laboratory.

Awakening conditions

At each awakening, subjects were required to make a verbal response regarding whether they thought they were awake or whether they thought they were asleep prior to hearing the tone and to estimate the amount of time that had passed since the last awakening.

During each training night, 27 awakenings were scheduled for each subject, and these awakenings randomly occurred and were separated into 9 blocks of 3 awakenings each. Markers A, B or C constituted a block. For example, awakening no. 1 may have used marker A, no. 2 may have used marker B, and no. 3 may have used marker C. The next block may have been organized as awakening no. 1 (no. 4 for the night) using marker C; awakening no. 2 (no. 5 for the night) using marker B; and awakening no. 3 using marker A (no. 6 for the night), and so on. Subjects were taught to use the markers to help judge time between awakenings and to discriminate wakefulness (marker A) from sleep (markers B and C).

Marker A was defined as the presence of alpha rhythm for at least 1 minute (i.e. wakefulness).

Marker B was defined as an awakening made as close to the first well-defined sleep spindle as possible. Marker B was considered to be the point of sleep onset.

Marker C was defined as an awakening 5 minutes after the first well-defined sleep spindle. If an arousal occurred before the end of the 5-minute period, an
estimate was gathered at that point. The subject was then allowed to return to sleep and the condition was repeated.

RESULTS

Ratio of subjective to objective sleep latency

The ratio of subjective to objective sleep onset latency changed significantly between G1C and G1T, with G1T having lower ratios (mean for G1C recovery was 3.9 and mean for G1T recovery was 2.1; see Table 3). The average ratio of subjective to objective sleep onset latency after treatment was still above 1.5, the definitive prestudy level; two subjects fell below the 1.5 criterion.

The ratio of subjective to objective sleep onset latency improved as a function of the number of nights spent in the laboratory ($r = -0.935$, using Fisher's $r$ to $z$ transformation). Individual subject correlations for this variable ranged between $-0.49$ and $-0.98$ (significance at the $p < 0.05$ level = $-0.87$ for 4 df). Individual correlations were used in a dependent groups $t$ test, and the correlations differed significantly from zero [$t(9) = -8.71$, $p < 0.001$].

Subjective insomniac estimates of awake/asleep discrimination

In order to assess sleep–wake discrimination, the percentage of correct sleep–wake estimations were compared with independent samples $t$ test. These results are displayed in Fig. 1. G1T subjects were different on their awake–asleep estimations on training nights than G2T subjects [$t(8) = 3.46$, $p < 0.008$; mean = 93% correct, SD = 7% for G1T and mean = 70% correct, SD = 13% for G2T] (see Fig. 1).

Using independent samples $t$ test, as above, G1C and G2T subjects differed significantly for the percentage of correct sleep–wake estimations, with G2T subjects estimating sleep and wake more accurately [$t(8) = -3.265$, $p < 0.01$ mean = 46% correct, SD = 9% for G1C, mean = 70% correct, SD = 13% correct for G2T] (see Fig. 1). Using a dependent groups $t$ test, G1T and G1C subjects differed significantly, with G1T estimating sleep and wake more accurately [$t(9) = -9.919$, $p < 0.001$].

ANOVA [3 (rules) × 2 (G1T, G2T)] was used to analyze which rules (i.e. markers A, B or C) were used most effectively by subjects to judge sleep from wakefulness (% correct response). Pairwise comparisons were analyzed using the Newman–Keuls test. A significant interaction between rule and treatment group was found [$F(2,16) = 3.47$, $p = 0.05$]. G1T subjects estimated sleep significantly better than G1C subjects, except for rule A. (G1T rule C mean = 96%; G1T rule A mean = 90%; G1T rule B mean = 84%; G1C rule A mean = 84%; significantly more accurate than G1C rule C mean = 35%; G1C rule B mean = 26%).

ANOVA [3 (rules) × 2 (G1T vs. G2T)] was used to analyze which rules (A, B or C) were used most effectively by subjects to judge sleep from wakefulness (% correct response). A significant interaction was found between group (G1T and G2T) and rule (A, B or C) [$F(2,8) = 9.31$, $p < 0.008$]. G2T subjects estimated sleep and wakefulness significantly better than G1T subjects, except for rule A. (G1T means are stated in the above paragraph; rules C, B and A were not different from G2T rule A mean = 86% but were significantly more accurate at estimating sleep and wakefulness than G2T rule B mean = 69%; G2T rule C mean = 56%).

To determine if arousal thresholds were used as cues to discriminate wakefulness from sleep, point biserial correlations between awake or asleep response (coded as 0 or 1, respectively) and arousal threshold level (range: 0–100 dB) were calculated for group 1 and G2T subjects. The point biserial correlations were averaged using Fisher's $r$ to $z$ transformation. The level of arousal threshold was nonsignificantly related to awake–asleep response ($r = 0.41$, df = 8, $p = n.s.$). The correlation between percentage correct awake–asleep response during the training nights was not significantly related to perceived efficacy for falling asleep within 60 minutes upon estimation in the morning following recovery sleep.

Sleep latency estimates

G1T and G2T subjects estimated that they had fallen asleep within 30 minutes following training [$F(1,4) = 7.80$, $p < 0.05$], which represents a significant change from baseline nights (see Table 3).
Frequency and quality of presleep cognition

Before lights out, subjects filled out a questionnaire that asked them to report, on a 1–5 scale, the content of their thoughts from lights out to sleep onset. Although the amount of presleep cognition did not change after training \(F(1,4) = 0.18, p = \text{n.s.}\) the content of thoughts did change significantly. Awareness of surroundings decreased after training nights for the G1C vs. G1T comparison \(F(1,4) = 9.00, p < 0.05\) but was not significant when G1T subjects were compared with G2T subjects \(F(1,4) = 2.66, p = \text{n.s.}\). A significant interaction for negative thoughts was found between G1T and G2T for the baseline to recovery night comparison \(F(1,4) = 6.40, p < 0.05\). Negative thoughts were more apparent for G2T subjects than for G1T subjects at baseline. Training had less effect on negative thoughts in the G2T subjects. Subjects’ future thoughts decreased \(F(1,4) = 27.00, p < 0.05\) when G1C was compared to G1T subjects.

Sleep EEG parameters

Sleep EEG parameters were statistically analyzed in the same manner as the other variables in this study (see Table 3). G2T subjects had an increased percentage of stage 2 sleep compared to G1T subjects \(F(1,4) = 8.58, p < 0.05\). Percentage of stage 3 sleep decreased from baseline to recovery in both G1C and G1T subjects \(F(1,4) = 12.07, p < 0.05\). Sleep latency to stage 1 (EEG-based sleep latency, not estimated sleep latency) decreased on recovery when compared to baseline for both groups \(F(1,4) = 6.65, p < 0.05\), but no condition effects or interaction effects (G1C vs. G1T subjects vs. G2T subjects) were found. No differences were noted with any comparison for any other sleep variable. The number of EEG arousals was within normal limits (13).

DISCUSSION

Perhaps the most significant finding of this study is that chronic subjective insomnics can learn to discriminate sleep from wakefulness well during a single sleep–wake discrimination training night in the sleep center, following 27 estimations of sleep and wakefulness.

Further it was found that self-efficacy for falling asleep within 60 minutes was significantly improved, that presleep mentation quality changed and that subjective estimates of sleep latency averaged less than 30 minutes following training. The ratio of subjective to objective sleep latency also decreased significantly across the nights subjects spent in the laboratory.

The fact that one of the major criterion variables for entry into the study, namely, the ratio of subjective to objective sleep latency, did not change significantly between G1T and G2T merits further discussion. First, the ratio of subjective to objective sleep latency changes noted might be explained by laboratory adaptation effects. This idea is substantiated by the strong negative correlation between the number of nights subjects spent in the study and the ratio of subjective to objective sleep latency. The adaptation idea helps to explain the G1C and G1T data. The adaptation explanation is weakened by the fact that G2T subjects did not differ significantly from G1T subjects for the ratio of subjective to objective sleep latency, despite the fact that G2T subjects spent fewer nights in the sleep laboratory.

A second plausible explanation is that the ratio of subjective to objective sleep latency is not a critical variable in measuring responsivity to the treatment procedures used in this study. This conclusion is based on the finding that rankings of subjects’ ratios of subjective to objective sleep latency at baseline were not related to rankings of subjects’ ratios of subjective to objective sleep latency at the end of the experiment. Some subjects may have learned better than other subjects, and the absolute ratio may not reflect such a difference.

To document improvement, sleep diaries were considered. However, it was felt that sleep diaries themselves provide subjective feedback and may have altered the results of this study. Future work may look at the use of sleep diaries alone as either a treatment or as an adjunct to the treatment described in this paper.

Subject follow-up

Eight of 10 subjects have been contacted regarding their current status. Two of the eight contacted subjects have returned to complete a night where they were awakened and asked to estimate sleep from wake, using the rules A, B and C. Both of these subjects estimated sleep from wake with more than 85% accuracy. All the other subjects reported they were sleeping better and felt the study was worth the effort, but not all the subjects believed their insomnia had been completely ameliorated. Two subjects are using hypnotic medication on an as-needed basis; both of these subjects have subclinical levels of leg movements, 70–90 per night. However, they reported improvement. As a result of the training they both estimated that they can use the medications on bad nights, as they have found their insomnia to be quite variable in presentation. However, the use of sleep onset feedback as a primary therapy for subjective insomnia is guarded until more data are available to judge the long-term efficacy.

Several questions remain: What is the mechanism
underlying the changes noted in this study? That is, do estimates improve based upon this EEG training procedure, or is similar improvement possible by simply paying attention to sleep by using logs or other devices? Can the method described in this paper be applied to modify the sleep latency perception of older groups and other types of insomniacs? Can this method be effectively applied to patients with periodic limb movement disorder, whose limb movements develop into an insomnia complaint? Patients with periodic limb movement disorder are often helped by hypnotic medications, perhaps because hypnotic medications decrease their sensitivity to the leg movements (14). The phenomenon itself can vary from night to night in these patients. If these individuals cannot discriminate a good night of sleep from a bad night of sleep and take medications nightly, then perhaps they can be taught to discriminate sleep from wakefulness using the techniques described, and take medications to help them sleep only on bad nights.

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REFERENCES


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