

The mesograde amnesia of sleep may be attenuated in subjects with primary insomnia

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Abstract

In this study, we pilot tested one of the more controversial components of the Neurocognitive Model of Insomnia; the proposition that subjects with chronic primary insomnia are better able to recall and/or recognize information from sleep onset intervals than good sleeper controls. Nine subjects participated in this pilot study, five of whom had a complaint of insomnia. The remaining four subjects were self-reported good sleeper controls. Subjects were matched for age, sex, and body mass. All subjects spent two nights in the sleep laboratory. The first night served as an adaptation night. The second night served as the experimental night during which a forced awakening and memory task was deployed. In this procedure, subjects were played single-word stimuli across four time periods: at natural sleep onset (Trial 1) and at the sleep onset transitions following three forced awakenings (Trials 2–4 from Stage 2 sleep). All subjects were awakened after about 6 h had elapsed from lights out and were tested for free recall and recognition memory for the word stimuli. The insomnia subjects, tended to identify more of the word stimuli on the recognition task (average for the four trials) and recognized significantly more of the words that were presented at sleep onset proper (Trial 1). This finding suggests that the natural mesograde amnesia of sleep may be attenuated in subjects with insomnia. © 2001 Elsevier Science Inc. All rights reserved.

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1. Introduction

Recently, we proposed that primary insomnia involves not only difficulty initiating and maintaining sleep but also two neurocognitive abnormalities that may account for several of the paradoxes observed in insomnia (e.g., sleep state misperception) [1]. The first abnormality is related to the persistence of high-frequency EEG activity at peri-sleep onset intervals. To date, at least three studies have found that subjects with chronic insomnia exhibit increased beta/gamma EEG activity at or around sleep onset [2–4]. The second abnormality is related to the attenuation and/or suppression of the mesograde

amnesia¹, which is normally attendant upon sleep [5]. To date, no studies have evaluated peri-sleep onset memory function in patients with insomnia. Several studies, however, have clearly shown that good sleeper subjects cannot recall information from periods *immediately prior* to sleep (cf., Refs. [6–8], *during sleep* (cf. Refs. [9–13]), or *from brief arousals*, which occur during the night (cf., Refs. [14,15]).

Our model (the Neurocognitive Model of Insomnia) [1] proposes that as one develops chronic insomnia via behavioral contingencies [16], there is an increase in high-frequency EEG activity (i.e., beta/gamma activity) at or around sleep onset as a result of classical conditioning. That is, beta/gamma EEG is elicited in response to the visual

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¹ The term “mesograde” is used to denote a form of amnesia that is not clearly either anterograde or retrograde, i.e., memory loss that is due to either a failure to consolidate information or the inability to retrieve consolidated information from long-term stores.

and/or temporal cues usually associated with sleepiness and sleep (e.g., bedroom, bed, bedtime) and this occurs in the absence of situational stressors. High-frequency EEG activity, in turn, allows for increased *sensory processing*, *information processing*, and the *formation of long-term memory*. The latter two phenomenon, it is hypothesized, account for sleep state misperception (cf., Refs. [17–21]) and the tendency to retrospectively overestimate sleep latency and under estimate total sleep time relative to polysomnography [22–29].

In the present study, we tested, in a preliminary way, one component of Neurocognitive Model; the proposition that subjects with chronic primary insomnia are better able to recall and/or recognize information from sleep onset intervals than good sleeper controls.

2. Methods

2.1. Subjects

Subjects were recruited from advertisements posted at the University of Rochester. Advertisements included flyers, posters, and an ad in the local faculty/staff newsletter. All subjects underwent an initial phone interview to document sleep quality and to rule out known medical or psychiatric illness. Following the phone assessment, eligible subjects were scheduled for an extensive evaluation, which included the Pittsburgh Sleep Quality Index (PSQI) [30,31], the Schedule for Affective Disorders — Lifetime Version (SADS-L) [32], the Hamilton Rating Scale for Depression (HRSD) [33,34], the Beck Depression Inventory (BDI) [35–37], the Beck Anxiety Inventory (BAI) [38,39], the California Verbal Learning Test (CVLT) [40,41], and a physical symptoms checklist.

Subjects who were not currently psychiatrically or medically ill were scheduled for two nights of in-laboratory polysomnography, signed a consent form, asked to keep a stable sleep–wake schedule, and required to complete sleep diaries for a period of 1 week. Subjects were provided US\$100 remuneration per night for their participation in this study.

Nine subjects participated in the current study, seven of which were part of a larger investigation on high-frequency EEG activity in subjects with primary and secondary insomnia and in good sleeper controls [42]. Five subjects had a complaint of insomnia and four subjects were self-reported good sleeper controls. Subjects were matched for age, sex, height, and weight (body mass index). Table 1 contains group information from the evaluation sessions and the average baseline sleep diary data.

Subjects with the complaint of insomnia met the diagnostic criteria for psychophysiological insomnia according to the International Classification of Sleep Disorders [43]. Criteria were: the complaint of insomnia and impaired daytime function, which is an indication of learned sleep-preventing associations, evidence of somaticized tension or “cognitive hyperarousal,” and active help-seeking behavior. The complaint of disturbed sleep also had one or more of the following characteristics: ≥ 30 min to fall asleep and/or ≥ 2 awakenings per night and/or wake after sleep onset time of ≥ 30 min, problem frequency ≥ 4 nights/week, and problem duration ≥ 6 months.

Exclusion criteria for all subjects were as follows: (1) significant current medical or psychiatric illness, (2) history of significant medical or psychiatric illness (within the last 5 years), (3) sleep disorders other than primary insomnia, (4) history of head injury, (5) any prescription medications that might interfere with the PSG or sleep diary assessments, (6) recreational drug use within 4 weeks

Table 1
Sample characteristics of subjects with insomnia and good sleeper controls

	Controls	Subjects with insomnia	P value
Age (years)	32.3 (11.5)	30.6 (8.9)	ns
Sex (% female)	50	60	ns
Height (in.)	67 (3.2)	65.0 (4.6)	ns
Weight (lb)	140 (23.5)	133.6 (29.2)	ns
Income (four-point Likert Scale) ^a	2.8 (0.9)	2.4 (0.9)	ns
BDI	0.5 (0.6)	7.2 (8.3)	0.049*
BAI	0.7 (0.9)	3.6 (2.3)	0.145
HRSD	0.5 (1.0)	2.4 (2.6)	0.191
PSQI	2.7 (1.3)	11.0 (0.71)	0.0002*
Sleep diary data			
Sleep latency (min)	12.9 (4.0)	43.7 (21.1)	0.029*
Number of awakenings	1.1 (1.5)	2.2 (1.3)	ns
Wake after sleep onset (min)	7.7 (8.7)	25.3 (12.7)	0.044*
Sleep efficiency (TST/TIB) (%)	83.2 (0.7)	69.4 (0.12)	0.091

All comparisons were undertaken using *t* test statistic.

^a Higher values indicate large income.

* Significant at $P < 0.05$.

ns = not significant.

of study intake, (7) use of SSRIs within 6 months of the in-laboratory study.

2.2. Protocol

All subjects spent two nights in the sleep laboratory. The first night served as an adaptation night. The second night served as the experimental night during which a forced awakening and memory task was deployed. In this procedure, subjects were played single-word stimuli (delivered at 1-min intervals) across four time intervals: at natural sleep onset and at the sleep onset transitions following three forced awakenings. Each forced awakening occurred either during the first 5 min of Stage 2 sleep following REM sleep offset(s) or after 90 min of sleep had accumulated since the last awakening. Stimulus administration started at “lights off” (and/or following the forced awakening) and ended 5 min *after* the appearance of the first sleep spindle or K complex. The maximum number of words presented over the course of the night was 100 (40 at natural sleep onset and 20 for each of the three subsequent trials). In order to ensure that the word stimuli were encoded, subjects were required to repeat aloud each of the word stimuli upon presentation. Failure to demonstrate encoding was noted. All subjects were awakened for the memory tests after 6 h had elapsed from lights out (± 25 min).

In the present study, only peri-sleep onset intervals were evaluated based on the perspective that it is during these intervals that patients with insomnia are most likely to suffer an attenuation of the normal mesograde amnesia of sleep [1]. We elected to use a “forced awakening paradigm” (rather than capitalize on natural awakenings across the night) so that the number of presentations, the stages from which the awakenings occurred, and the time elapsed between stimulus presentations would be standardized. Finally, the memory tests were performed in the morning so that we could assess the kind of memory that is most likely to interfere with morning attributions of sleep quality and quantity (i.e., morning recollections of events that occurred over the course of the prior night).

2.3. Word stimuli properties and administration

The word stimuli were proper nouns matched for concreteness, usage frequency, and utterance duration. The word stimuli, spoken by one speaker (female), were digitally matched for loudness, standardized for volume of delivery, and delivered via speakers mounted on either side of the bed headboard. Word randomization, presentation, recognition testing, and report generation were accomplished by a beta software routine (“ComputerSez”) written to interface with a PC using a Windows 95 operating system. It should be noted that: (1) the word stimuli were originally selected by a cognitive neuroscience investigator and that the mode of presentation and the memory test

format have been used in a variety of published studies regarding implicit and explicit memory for auditory stimuli (cf., Refs. [44,45]) The method of administration and the interstimulus interval have been previously employed in three sleep and memory studies [7,12,13,46]. Stimuli were delivered via speakers mounted on either side of the bed headboard (rather than via ear phones or pillow speakers based on the belief that “airborne” stimuli would more closely approximate normal auditory stimulation.

2.4. Memory tests

For the *free recall* test, subjects were asked to write down all the words they could remember that were presented during the night. These words were then read aloud and keyed into the program that governs our stimulus presentation, test administration, and report generation (“ComputerSez”). For the *recognition* test, subjects were played all the words administered on the prior night. These words were randomly mixed with an equal number of words that had not been presented. Upon administration of the words, subjects were required to press a “yes” or “no” button on a switch box to indicate whether they recognized the word. In addition to assessing recognition, this procedure measured reaction time.

The free recall memory test was scored for total percent correct for all the words administered over the course of the night. For example, 5 words correctly recalled out of 100 administered would equal 5%. The recognition test was scored for total percent correct, for percent correct per trial (10-min window pre–post-sleep onset), and for percent of “false-positives” (items identified as presented but were not). The latter percentage was used to account for a “yes” response bias. Words for which there was no evidence of encoding (no repetition of word by the subject) were not included in the calculation of the final percentages.

2.5. Polysomnographic measures and recording parameters

The recording montage for the two consecutive nights in the sleep laboratory consisted of a minimum nine electrophysiologic signals. The basic montage included two EOGs referenced to a supramedial electrode (LOC and ROC), six EEGs referenced to linked mastoids (F3, F4, C3, C4, O1, and O2), and a bipolar mentalis EMG. In addition to the basic montage, several additional measures were obtained. On Night 1, these included one channel of nasal/oral airflow and two channels of leg-related motor activity (right and left tibial EMGs). The airflow and tibial data were used to rule out sleep apnea and/or periodic leg movement disorder. On Night 2, these included an audio signal marker and voice marker (trachea microphone). The signal marker was used to precisely mark on the digital record when the word stimuli were presented. The voice marker was utilized to precisely mark on the digital record when the subject verbalized the presented word stimuli. The signal mark

followed by a voice mark served to document that the subject heard and repeated the presented word. This, along with the technicians' observation that the word was spoken aloud and correctly identified, served as the evidence for whether the subject encoded the word stimuli.

All the electrophysiologic signals were initially acquired using a Grass Model 8-21 electroencephalograph. Analog signals were then digitized for on-line and off-line display on a 21-in. super VGA screen (1280*1064). Digital acquisition was governed by Stellate Harmonie software and accomplished by a BSMI 519 AD board. The final digital display was additionally modified by digital filtering for optimal on-screen display.

3. Results

3.1. Demographic, and clinical measures

The groups did not significantly differ on CVLT measures of waking memory function and, as can be seen in Table 1, the groups did not significantly differ in relation to age, sex, height, weight, or income. Income was assessed using a four-point Likert scale ranging from <15,000 to >50,000 per annum. Interestingly, although neither group scored within the clinical range, the insomnia group reported significantly more depression type symptoms as measured by the BDI. This may be due to increased scores on the sleep disturbance and fatigue items on this instrument or may reflect a genuine increase in depressive type symptomatology in patients with primary insomnia. As expected, the groups significantly differed on self-reported sleep disturbance (PSQI) and on prospectively assessed sleep latency and wake after sleep onset times (average sleep diary data for the 1-week baseline period prior to the in-lab study). The patients with insomnia also tended exhibit lower sleep efficiency on the baseline diary measures.

3.2. Long-term memory for the word stimuli and sleep latency data

As can be seen in Table 2, the groups did not significantly differ with respect to response bias (false-positive recognition), on our measures of reaction time, or with respect to the number of words heard and repeated during stimuli presentation for the four trials. It should also be noted that the groups did not significantly differ for the amount of time they took to fall asleep over the course of the four sleep onset periods. On average, the patients with insomnia took 19.6 ± 3.64 min to fall asleep while the controls took 15.6 ± 6.5 min to fall asleep. The natural sleep onset period showed the greatest distinction between the groups (30.7 ± 7.5 vs. 20.5 ± 12.1), but this also failed to reach significance ($P < .20$). When these data are considered together, they suggest that both groups encoded the same amount of information peri-sleep onset, were not subject to significantly different response biases, and were awake for comparable periods of time during the four trials.

As for retrieval, the groups did not differ on free recall but the subjects with insomnia tended to identify more of the word stimuli on the recognition task (average for the four trials) and recognized significantly more of the words that were presented at sleep onset proper (Trial 1). Subsequent analyses revealed that subjects with insomnia not only recognized more of the words from the first trial, but also significantly more words from the 5-min interval immediately prior to sleep onset (53% vs. 19%, $P < .05$).

4. Discussion

The preliminary data acquired in this study suggest that subjects with insomnia may be better able to retrieve information from peri-sleep onset intervals and that this effect exists primarily for recognition memory for information from the early sleep phase sleep onset. There may be a

Table 2
Memory performance in subjects with insomnia and good sleeper controls

	Controls	SBJS with insomnia	P value
Number of words encoded	43 (20.0)	49 (18.3)	0.61
Recognition (false-positive, %)	8.9 (7.2)	20 (19.1)	0.27
Free recall (all trials, %)	4.6 (3.2)	9.8 (10.7)	0.35
Recognition (all trials, %)	45.2 (11.8)	68.6 (19.9)	0.08
First trial	46.2 (12.6)	75.8 (12.7)	0.01*
Second trial	42.0 (17.6)	58.0 (38.1)	0.55
Third trial	42.5 (26.9)	59.7 (25.5)	0.38
Fourth trial	51.6 (8.6)	59.3 (29.9)	0.69
Recognition speed (true-positive, ms)	2753 (820)	2137 (761)	0.29
Recognition speed (false-positive)	3358 (510)	2664 (903)	0.19
Speed (false/true-positive)	1.259 (0.20)	1.261 (0.17)	0.99

All comparisons undertaken using *t* test statistic.

* Significant at $P < 0.05$.

combination of factors, however, that when taken into account may show the memory effect to (1) include free recall memory and (2) to span beyond the initial sleep onset interval. The factors include recruitment, technical, statistical, and methodologic factors.

With respect to recruitment, three of our five subjects with insomnia were young and/or had insomnia for only a relatively short period of time (6–12 months). Inclusion of subjects with more chronic insomnia may serve to enhance our ability to detect effects that are likely to be associated with chronicity. With respect to technical issues, several components of our stimulus delivery system could be improved. For example, the sound quality of the original recordings were not uniformly free from “noise” and the speaker and amplification system themselves could be improved. These technical problems may have served to diminish the number of usable stimuli for both groups, stimuli that may have, potentially, been encoded and recalled by the patients with insomnia. With respect to statistical issues, because our study was preliminary and included relatively few subjects, our power to detect subtle effects was reduced. Thus, we were able to only detect the most robust effect: enhanced recognition memory for information presented at the initial sleep onset period. Taking into account these issues may make it possible to detect subtler effects in subsequent studies. Finally, the use of the forced awakening paradigm itself may not have been ideal. It is plausible that sensory and information processing are high for waking intervals across the night, but only for awakenings that occur naturally.

5. Concluding remarks

Although the present data are consistent with the Neurocognitive Model [1] much remains to be determined. Assuming that the findings of the present study are reliable, the next step toward evaluating the role of abnormal sensory and information processing in primary insomnia will be to show that these phenomena are related to both the occurrence of beta/gamma EEG activity at or around sleep onset and to patient’s subjective judgments about their sleep quality and quantity. Such data are likely to allow us a deeper understanding of the pathophysiology of primary insomnia.

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