Reported Chronic Insomnia Is Independent of Poor Sleep as Measured by Electroencephalography

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Objective: Several behavioral, physiological, and subjective variables were examined in subjects reporting chronic insomnia (IN group) and subjects with no complaint of insomnia (NC group) to determine factors predictive of poor sleep as measured by electroencephalography (EEG sleep). **Methods:** A total of 177 subjects (121 in the IN group and 56 in the NC group) were evaluated on the basis of EEG sleep, subjective sleep, sleepiness, performance, mood, personality, and metabolic parameters during a 36-hour laboratory stay. **Results:** Equal percentages of subjects in each group had 0, 1, or 2 nights of poor EEG sleep, indicating that the IN group was not more likely to have impaired sleep in the laboratory. Results of the Minnesota Multiphasic Personality Inventory showed that subjects in the IN group had more pathological personality profiles, and results of laboratory studies showed that these subjects had worse mood ratings, less subjective sleepiness, poorer memory performance, and longer midafternoon sleep latencies. Subjects in the IN group also rated their laboratory sleep as poorer in quality with more time awake after sleep onset and longer sleep latencies, but no differences in EEG sleep were observed. Poor nights of EEG sleep were associated with being male, increasing age, and a history of more time awake after sleep onset; among the laboratory tests, poor EEG sleep was associated with worse mood ratings, poorer memory performance, longer sleep latencies (as indicated by higher scores on the Multiple Sleep Latency Test), higher sleep/wake ratios for metabolic parameters, lower ratings of sleep quality, and longer perceived sleep latencies. **Conclusions:** A history of chronic insomnia does not predict poor EEG sleep. Both chronic insomnia and poor EEG sleep are associated independently with dysphoria, hyperarousal, diminished waking function, and negative subjective sleep quality. Separate arousal and sleep systems are posited to account for these results. **Key words:** insomnia, EEG sleep, sleepiness, mood, performance.

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ANOVA = analysis of variance; EEG sleep = sleep measured by electroencephalography; IN = insomnia complaint group; MAST = Memory and Search Test; MMPI = Minnesota Multiphasic Personality Inventory; MSLT = Multiple Sleep Latency Test; NC = no complaint group; POMS = Profile of Mood States; SaO_2 = oxygen saturation; STAI = Spielberger State-Trait Anxiety Inventory; VO_2 = oxygen consumption.

INTRODUCTION

Patients who report chronic insomnia complain not only of poor sleep but also of other symptoms, such as fatigue, stress, anxiety, and depression (1, 2). Laboratory evaluations of insomniacs have documented long daytime sleep latencies or inability to fall asleep using the MSLT (1, 3–5), consistently longer subjective estimates of sleep latency and time spent awake relative to electroencephalographic measures of sleep (6, 7), and greater physiological activation as indexed by body temperature (8–10), whole-body metabolic rate (1), and heart rate (8, 11–14).

Observations of increased physiological activation accompanied by fatigue and dysphoria have led to the suggestion that chronic insomnia is primarily a disorder of central nervous system hyperarousal rather than a disorder specifically associated with sleep. Indeed, caffeine-induced physiological activation in normal young adults produces many of the primary and secondary symptoms of patients with insomnia, including poor nocturnal sleep, dysphoric mood, and greater anxiety as measured by the MMPI (15). Disturbing sleep without physiological activation, however, does not produce secondary symptoms of insomnia (16). These results provide evidence that a factor other than the sleep-wake system, as indexed by electroencephalography, is the primary basis for the insomnia symptom complex (16). This is supported by the fact that many patients who report chronic insomnia have normal EEG sleep. Such patients have presented often enough for treatment of their sleep disorder that a diagnostic classification, sleep state misperception, has been assigned to them (17). Some investigators believe that sleep state misperception is an artifact of the variability of insomniac sleep such that a good night of sleep could occur occasionally on laboratory evaluation nights (18). A study addressing that issue, however, reported that patients with sleep state misperception had consistently high sleep efficiencies on 6 laboratory evaluation nights scheduled over a 3-month period (19). Despite high sleep efficiency, these patients continued to report less sleep time than

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that recorded by electroencephalography. Subsequent investigation of patients with sleep state misperception using measures of whole-body metabolism indicated a persistent state of hyperarousal and dysphoria similar to, but not as severe as, that observed in a group of patients with chronic insomnia and poor EEG sleep (20).

Together, the results of studies of patients reporting chronic insomnia reinforce the notion of a persistent state of hyperarousal that could produce longer sleep latency (as measured by the MSLT), higher metabolic rate, greater body temperature, increased tension, decreased vigor, and more personality disturbance (15). By itself, however, chronic hyperarousal is not enough to determine who will experience disturbed EEG sleep. Indeed, observations of disturbed EEG sleep in patients with insomnia are so infrequent that insurance providers are reluctant to reimburse for polysomnographic evaluation. Thus, in the present study, we examined a wide range of behavioral, physiological, and subjective factors in large groups of subjects reporting chronic insomnia or normal sleep with two purposes in mind, to determine the extent to which self-reported sleep parameters were related to electroencephalographic measures and to determine whether any of the wide range of subjective or behavioral factors were predictive of poor EEG sleep. The ultimate goal was development of a predictive equation that could discriminate between groups and be used to guide treatment.

METHODS

Subjects

Participants were required to be healthy and 18 to 50 years old; both men and women were included. Potential subjects were solicited mostly through ads placed in local newspapers seeking participants for a sleep study. A few subjects were referred from sleep disorders treatment centers, but the specific referral source (newspaper or sleep center) was not recorded. Each potential subject completed an initial questionnaire, which included a brief sleep history, a medical history, and drug and alcohol abuse screening tests.

Insomnia Complaint Group

Individuals were considered for the IN group if they indicated on the initial questionnaire that they had a sleep problem, that it took them at least 45 minutes to fall asleep at least 4 nights each week, or that they were awake for at least 60 minutes after falling asleep each night for at least 4 nights each week. These conditions had to exist for at least 1 year.

No Complaint Group

Individuals were considered for the NC group if they indicated normal sleep on the initial questionnaire. These individuals also were required to report less than 30 minutes of sleep latency and less than 30 minutes of time awake during the night.

Exclusion Criteria

Potential subjects who indicated excessive caffeine consumption (>250 mg of caffeine per day), who were using psychoactive medication or drugs, or who had completed a drug or alcohol abuse program within the previous year were excluded. Potential subjects with a history of depression or psychiatric hospitalization and those who had a history strongly suggestive of circadian dyssynchrony (eg, shift workers), sleep apnea, or periodic leg movements also were excluded. Individuals meeting the study criteria were invited to participate after they completed an informed consent form and 2 hours of laboratory acclimatization that involved practice on computer tests and questionnaires to be used in the study.

Procedure

Shortly after the introductory/practice day, each subject spent 2 nights and the intervening day in the laboratory. On both nights, a standard clinical polysomnogram, including two eye channels, central and occipital electroencephalographic channels, chin and leg electromyographic channels, electrocardiographic channels, airflow, and chest movements, was recorded. On the first night, blood SaO_2 also was recorded. On the second night, VO_2 was recorded instead of SaO2, using a Sensorimedics Deltatrac high-flow metabolic monitor. The leg electromyogram, electrocardiogram, airflow, chest movements, and SaO_2 were used only for screening purposes and were not scored for additional analysis.

During the day, subjects performed computer tests, completed the MMPI and a sleep history, and were fed the same menu of food prepared at the laboratory. Caffeinated beverages were not available. Subjects usually did not leave the laboratory during the day and did not engage in any activity more vigorous than walking to the bathroom. After the second night in the laboratory, subjects completed brief computer tests and then were free to leave.

All subjects were assigned their own room for the course of the study. Each room contained a standard hospital bed and furniture, including a desk with an Apple IIGS computer. Subjects participated in the study in groups of one to two individuals. Subjects completed all tests and questionnaires in their own rooms while being observed by a technician by means of video monitors. Subjects were not permitted to sleep during the day and were closely monitored to prevent sleep during computer tests (video monitoring) and during daytime metabolic observations (EEG monitoring). Meals and breaks were scheduled in another area of the laboratory, which was also observed by a technician.

Measurements

Polysomnographic sleep. Nighttime sleep recordings were scored using standard clinical and EEG criteria (21). In addition, brief EEG arousals during sleep were scored according to criteria established by the American Sleep Disorders Association (22). Subjects were excluded from further analysis if polysomnographic recordings indicated an apnea/hypopnea index greater than 10, a periodic leg movement arousal index greater than 10, or EEG evidence of psychoactive drug use.

Performance and self-reports. Estimates of time taken to fall asleep, number of awakenings, time awake after sleep onset, and a rating of sleep quality on a five-point scale were given by each subject within 15 minutes of awakening from each night of sleep in

the laboratory. Performance and mood were assessed with a battery of tests administered at 2-hour intervals from 8 AM to 8 PM during the intervening laboratory day. The battery included the MAST (percentage of letters recognized in 2 minutes at each of three memory loads) (23), proofreading speed (number of lines read in 10 minutes), hand tremor (2-minute insertion of a stylus into a 4-mm opening with the percentage of side-touching time measured), a computer modification of the Williams Word Memory Test of immediate free recall (number of words remembered) (24), visual vigilance (percentage of hits in 30 minutes) (25), subjective sleepiness (rating on 10-point visual analog scale), the POMS, the state section of the STAI, and oral temperature.

Multiple Sleep Latency Test. Four-channel sleep recordings (LE-A2, RE-A2, C3-A2, and Oz-A1) were made at 10 AM, 12 PM, 2 PM, 4 PM, 6 PM, and 8 PM of the day spent in the laboratory. For each MSLT, subjects were in bed for 20 minutes or until the first scorable epoch of non–stage 1 sleep occurred. All reported MSLT scores are latencies to stage 1 sleep.

Metabolic rate. Complete details of the measurement of the VO_2 index of metabolic rate were reported by Bonnet and Arand (1). Waking VO_2 measurements were recorded for 20 minutes immediately after awakening after the first night in the laboratory, for 20 minutes before lights out on the second night, and throughout the entire second night of sleep. Waking metabolic rate was defined as the mean of the postsleep and presleep VO_2 measurements, excluding any measurements made during body movements. Sleeping metabolic rate was defined as the mean of all VO_2 data recorded during sleep, excluding measurements taken within 3 minutes of awakenings or other EEG arousals. VO_2 between lights out and sleep onset was not scored because of movement artifact and because sleep latencies varied widely.

Design and Analysis

The initial screening resulted in 134 subjects assigned to the IN group and 57 subjects assigned to the NC group. Thirteen IN subjects and one NC subject were excluded from further analysis because sleep apnea or periodic leg movement syndrome was observed during polysomnographic recordings. These exclusions resulted in 121 subjects in the final IN group (72 men and 49 women) and 56 subjects in the final NC group (35 men and 21 women). Subjects in each group were then classified according to the number of nights of poor EEG sleep experienced in the laboratory. A poor night of sleep was defined as an EEG sleep latency of at least 45 minutes or a sleep efficiency of less than 85%. Sleep efficiency was defined as the percentage of time spent in any sleep stage (excluding time awake) during the period beginning at initial sleep onset and ending at the final morning awakening.

Analysis of variance. Differences between groups (IN vs. NC) and differences in number of nights of poor EEG sleep (0, 1, or 2) were examined by using 2×3 ANOVAs. Repeated-measures factors for nights in the laboratory or time of testing during the day were added to the ANOVA design as appropriate for a particular dependent measure. Because the goal of the study was to determine which measures discriminate reliably between groups on the basis of EEG sleep quality, the interaction of group with nights of poor EEG sleep was of particular interest in these analyses. Because of unequal numbers of subjects in each treatment cell, least-squares solutions to the ANOVAs were calculated using the SAS general linear models procedure (26). Consequently, all reported averages are weighted means adjusted for unequal cell frequencies by the least-squares procedure.

Regression. Measures exhibiting significant or nearly significant mean differences were entered stepwise into multivariate regression equations to devise a set of variables that predicts poor EEG sleep while simultaneously controlling for the correlation among those variables. Regression equations were calculated to predict the number of nights of poor EEG sleep and to predict sleep efficiency and sleep latency on the second night in the laboratory.

RESULTS

Differences Between Complaint Groups

Table 1 presents mean responses on the initial questionnaire for the IN and NC groups and *F* values for the main effect of difference between groups. Compared with the NC group, the IN group reported longer sleep latencies, more frequent awakenings, and more time awake after sleep onset. The IN group also reported smoking more cigarettes, but they did not report drinking more alcoholic or caffeinated beverages. In addition, the IN group had significantly higher scores on all subscales of the MMPI except the lie (L), masculine-feminine (MF), and hypomania (MA) subscales.

Daytime averages of results of repeated laboratory tests of subjective state and performance are shown in Table 1 for each complaint group. The IN group reported less sleepiness, higher state anxiety on the STAI, and more negative mood on five of six subscales of the POMS. They also performed more poorly on the Williams Word Memory Test. No interaction of complaint group with time of day was observed for any measure of subjective state or performance.

Means for selected physiological measures are shown in Table 1 for each complaint group. Overall, the IN and NC groups did not differ in body temperature, waking or sleeping metabolic rate, or the ratio of waking to sleeping metabolic rate. On the MSLT (not shown), the IN group took longer to fall asleep at 4 PM (13.0 vs. 10.8 minutes). This difference was supported by a significant interaction of group with test time ($F = 2.86$, $p < .02$) and by results of post hoc Newman-Keuls tests of differences between means. Post hoc tests also indicated that the groups did not differ at any other MSLT time.

Differences Among EEG Sleep Groups

Means for groups experiencing 0, 1, or 2 nights of poor laboratory EEG sleep are presented in Table 2 along with *F* values for the main effect of differences between groups. As shown, subjects experiencing 1 or 2 nights of poor EEG sleep were significantly older and more likely to be male, and they reported greater time awake after sleep onset on the initial questionnaire. Poor EEG sleep was not associated with a history of frequent problematic nights of sleep, longer sleep la-

TABLE 1. Initial Questionnaire and Laboratory Self-Reported, Performance, and Physiological Responses for Complaint Groups

	Complaint Group					
	NC		IN		*F*	*p*
	Mean	(SD)	Mean	(SD)		
Initial questionnaire						
Age (y)	36	(8)	35	(9)	<1	
Sex (% male)	68		62		<1	
Duration of sleep problems (y)			7.96	(9.19)		
Frequency of sleep problems (nights/wk)	0.20	(0.68)	4.67	(1.99)	227	.001
Sleep latency (min)	16	(8)	83	(64)	49.45	.001
Awakenings (*N*)	1.03	(0.97)	4.15	(5.52)	15.49	.001
Time awake after sleep onset (min)	25	(22)	66	(55)	25.53	.001
Alcohol consumption (drinks/wk)	4.01	(6.75)	6.60	(10.48)	2.45	
Caffeine consumption (drinks/d)	1.47	(1.98)	1.43	(1.83)	<1	
Tobacco consumption (cigarettes/d)	3.52	(6.88)	8.46	(10.53)	9.07	.004
Laboratory self-reported parameters[a]						
Sleepiness	6.64	(1.73)	5.86	(1.45)	8.72	.004
State anxiety (STAI)	29.98	(6.13)	37.02	(8.68)	26.64	.001
POMS						
Tension	4.46	(3.63)	7.68	(5.04)	17.71	.001
Depression	3.91	(5.63)	8.93	(9.82)	11.76	.001
Anger	3.50	(4.85)	6.50	(7.82)	6.57	.02
Vigor	18.89	(7.07)	16.54	(6.71)	4.00	.05
Confusion	4.38	(3.23)	6.26	(4.19)	8.09	.006
Fatigue	16.83	(4.73)	15.41	(5.19)	2.73	
Laboratory performance						
Words remembered	7.08	(2.75)	5.86	(2.59)	7.58	.007
Vigilance hit rate (%)	77	(22)	72	(22)	1.27	
Proofread lines (*N*)	290	(102)	292	(140)	<1	
MAST hit rate (%)	85	(13)	86	(11)	<1	
Hand steadiness (% time off target)	13	(17)	14	(15)	<1	
Physiological parameters						
Metabolic rate (VO_2) sleep/wake ratio	0.847	(0.046)	0.836	(0.061)	1.22	
Body temperature (°F)	98.9	(5.9)	98.8	(4.8)	2.33	

[a] Higher values indicate more of each parameter.

tencies, or use of alcohol, caffeine, or tobacco. There were no significant differences in MMPI scores between EEG sleep groups.

Daytime averages of results of repeated laboratory tests of subjective state and performance are shown in Table 2 for each EEG sleep group. Scores on the POMS subscales of tension, depression, anger, and confusion increased with increasing nights of poor EEG sleep, whereas immediate-recall memory performance decreased. No interaction of EEG sleep group with time of day was observed for any measure of subjective state or performance.

Means for selected physiological measures are shown in Table 2 for each EEG sleep group. The ratio of sleeping to waking metabolic rate increased with nights of poor EEG sleep, as did daily averages on the MSLT. Body temperature did not differ among EEG sleep groups.

History of Insomnia and EEG Sleep

Equal percentages of subjects in each complaint group experienced 0, 1, or 2 nights of poor EEG sleep, as shown in Table 3 ($\chi^2 = 0.004$, $p = .998$). These results indicate that the IN group was not more likely to have impaired laboratory sleep as defined by EEG sleep efficiency or sleep latency. Furthermore, there were no interactions between complaint group and nights of poor EEG sleep for any daytime measure of subjective state, performance, or physiological state even though main effects for complaint group and poor nights of EEG sleep were associated with those measures. The lack of interaction is illustrated in Figure 1 for POMS tension and immediate-recall memory performance, where differences between complaint groups are plotted as a function of number of nights of poor EEG sleep.

EEG sleep parameters and subjective ratings of sleep

TABLE 2. Initial Questionnaire and Laboratory Self-Reported, Performance, and Physiological Responses for EEG Sleep Groups

	Nights of Poor EEG Sleep							
	0		1		2		*F*	*p*
	Mean	(SD)	Mean	(SD)	Mean	(SD)		
Initial questionnaire								
Age (y)	33	(8)	37	(9)	37	(7)	5.45	.006
Sex (% male)	48		72		76		5.34	.006
Duration of sleep problems (y)	2.28	(2.48)	2.54	(2.73)	2.46	(2.93)	<1	
Frequency of sleep problems (nights/wk)	3.30	(7.57)	5.63	(9.65)	5.16	(9.85)	1.27	
Sleep latency (min)	45	(5)	45	(6)	57	(8)	<1	
Awakenings (*N*)	1.95	(1.85)	2.07	(2.11)	3.76	(9.52)	1.92	
Time awake after sleep onset (min)	35	(37)	41	(42)	61	(78)	3.65	.03
Alcohol consumption (drinks/wk)	4.92	(8.68)	4.80	(8.62)	6.19	(12.25)	<1	
Caffeine consumption (drinks/d)	1.69	(1.77)	1.30	(2.29)	1.36	(1.50)	<1	
Tobacco consumption (cigarettes/d)	5.69	(8.46)	4.37	(9.72)	7.91	(11.85)	1.30	
Laboratory self-reported parameters[a]								
Sleepiness	6.04	(1.56)	6.34	(1.61)	6.38	(1.55)	<1	
State anxiety (STAI)	32.20	(7.84)	32.69	(9.23)	35.63	(8.90)	2.22	
POMS								
Tension	4.57	(4.06)	5.54	(4.59)	8.12	(5.85)	7.30	.001
Depression	4.17	(7.00)	5.70	(8.84)	9.40	(11.85)	4.34	.02
Anger	3.30	(6.03)	4.06	(6.28)	7.64	(9.33)	4.78	.01
Vigor	16.52	(7.31)	18.14	(7.07)	18.50	(5.22)	1.32	
Confusion	4.36	(3.51)	5.07	(4.20)	6.54	(4.47)	3.67	.03
Fatigue	15.22	(5.20)	17.56	(4.38)	15.59	(5.22)	3.17	.05
Laboratory performance								
Words remembered	6.80	(2.65)	7.20	(2.34)	5.41	(1.25)	4.94	.01
Vigilance hit rate (%)	73	(22)	78	(21)	72	(18)	<1	
Proofread lines (*N*)	286	(79)	277	(98)	309	(85)	<1	
MAST hit rate (%)	87	(10)	87	(12)	83	(15)	1.73	
Hand steadiness (% time off target)	12	(14)	11	(13)	18	(20)	1.98	
Physiological parameters								
Metabolic rate (VO_2) sleep/wake ratio	0.825	(0.061)	0.839	(0.467)	0.860	(0.051)	4.32	.02
MSLT sleep latency (min)	10.37	(4.33)	13.09	(4.19)	15.03	(3.31)	16.08	.001
Body temperature (°F)	98.8	(5.6)	98.8	(4.5)	98.8	(4.86)	<1	

[a] Higher values indicate more of each parameter.

averaged over 2 nights are presented in Table 4 for each complaint group. ANOVAs on EEG sleep parameters revealed a small but significant difference between complaint groups in latency to stage 2 sleep. Analysis of differences between complaint groups in ratings of the perception of laboratory sleep revealed that the IN group reported longer sleep latencies, more time awake after sleep onset, more frequent awakenings, and poorer sleep quality.

Frequency of brief EEG arousals are shown in Figure 2 for each complaint group as a function of nights of poor EEG sleep. It is apparent in this figure that EEG arousals were disproportionately higher in the IN group experiencing 2 nights of poor EEG sleep, which was supported by a significant interaction of complaint group with nights of poor EEG sleep ($F = 3.46$, $p < .04$). Similar patterns of interaction are presented in Table 5 for both EEG and subjective measures of laboratory sleep latency. The same tendencies, although not statistically significant, are apparent in the measures of subjective time awake after sleep onset and subjective sleep quality, which also are shown Table 5.

Predicting Poor EEG Sleep

Stepwise multiple regression analysis indicated that 20% of the variance associated with number of nights of poor EEG sleep could be explained by the following variables (partial correlation in parenthesis): POMS tension (0.25), age (0.22), time awake after sleep onset reported on the initial questionnaire (0.18), and sex (0.15, more male). Sleep efficiency on the second laboratory night was associated with age (−0.27), POMS tension (−0.22), time awake after sleep onset reported on the initial questionnaire (−0.21), and POMS vigor (−0.14) with a total of 21% of variance explained. Sleep latency on the second laboratory night was associated with POMS tension (0.26) with 7% of variance explained.

TABLE 3. Number and Percentage of Subjects in Each Complaint Group Experiencing 0, 1, or 2 Nights of Poor EEG Sleep

Complaint Group	Nights of Poor EEG Sleep			Total
	0	1	2	
NC				
N	28	16	12	56
%	50.0	28.6	21.4	
IN				
N	61	34	26	121
%	50.4	28.1	21.5	
Total				
N	89	50	38	177
%	50.3	28.3	21.5	

DISCUSSION

Extensive screening procedures resulted in clear identification of a group complaining of frequent, longstanding insomnia that was not associated with other clinical sleep disorders, psychiatric problems, or substance abuse. This selection procedure differs from those used in some earlier studies of daytime functioning in insomniacs that combined patients with a variety of psychiatric or sleep disorders diagnoses (5, 27). Thus, the potential confounding influences of other problems or disorders were avoided in the present study. Comparison of this group to a similarly screened group reporting no history of insomnia revealed a consistent functional pattern, including higher MMPI scores and more frequent tobacco use in the IN group and, in the laboratory, less subjective sleepiness, dysphoric moods, poorer immediate-recall memory performance, and equal or longer daytime sleep latencies in the same group. This pattern of results reinforces those of earlier studies reporting functional deficits in chronic insomniacs (1–5). As in previous studies, the deficits seem to reflect a state of tense/anxious hyperarousal rather than a sleep debt because the IN group had latencies on the MSLT that were equal to or longer than those observed in the NC group.

Functional state also was examined using a "dose-response" approach with respect to sleep measured physiologically by the EEG. A dose in this case was defined as the degree of persistent sleep disruption indexed by the number of poor nights of EEG sleep in the laboratory. The definition of a poor night of sleep as one with an extended sleep latency or low sleep efficiency is consistent with that typically used in clinical settings. Subjects divided along those lines exhibited consistent functional differences that, in most cases, were greatest between the groups experiencing consistently good or poor EEG sleep. Scores for

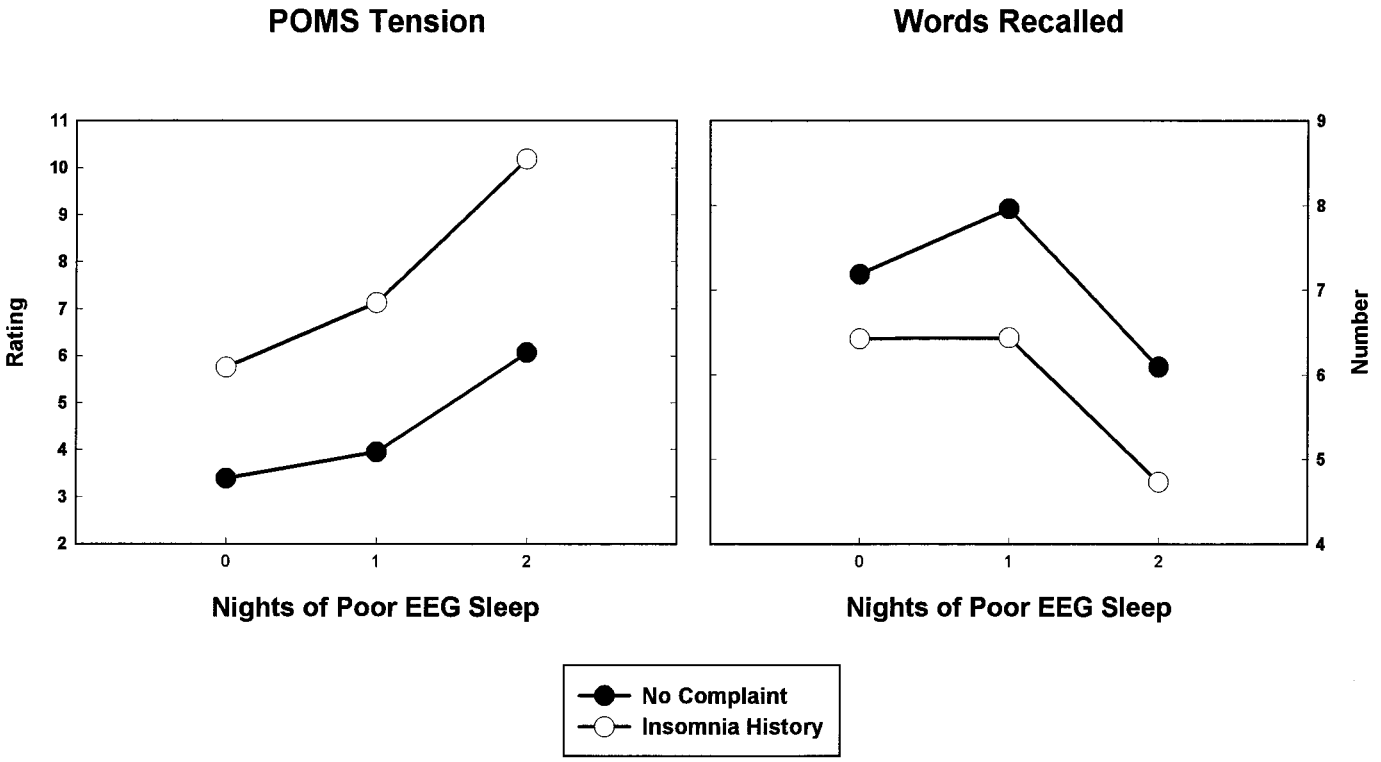

Fig. 1. Differences between complaint groups in POMS tension and immediate-recall memory performance as a function of number of nights of poor EEG sleep.

TABLE 4. EEG Sleep Parameters and Subjective Ratings of Sleep Averaged Over 2 Nights for Complaint Groups

	NC		IH		*F*	*p*
	Mean	(SD)	Mean	(SD)		
EEG sleep parameters						
Total Sleep (min)	386	(61)	379	(71)	1.55	
Sleep efficiency	84	(11)	83	(14)	<1	
Latency to stage 1 (min)	18	(23)	22	(26)	2.82	.10
Latency to stage 2 (min)	29	(28)	37	(34)	5.90	.02
Stage 1 sleep (%)	15	(7)	16	(10)	<1	
Stage 2 sleep (%)	38	(11)	38	(12)	<1	
Stage 3 sleep (%)	8	(4)	6	(6)	2.95	.10
Stage 4 sleep (%)	6	(6)	6	(7)	<1	
REM[a] sleep (%)	17	(6)	17	(6)	<1	
Time awake after sleep onset (min)	73	(51)	74	(50)	<1	
REM[a] latency (min)	96	(66)	106	(65)	1.64	
Awakenings (*N*)	2.95	(14)	2.91	(18)	<1	
EEG Arousals (*N*/h)	7.69	(1.70)	8.99	(1.63)	3.13	.10
Subjective sleep ratings						
Sleep latency (min)	26	(17)	61	(51)	25.59	.001
Time awake after sleep onset (min)	39	(38)	84	(84)	15.98	.001
Awakenings (*N*)	2.86	(2.85)	4.51	(3.71)	7.74	.007
Sleep quality[b]	2.32	(0.80)	3.24	(0.90)	40.26	.001

[a] REM = rapid eye movement.
[b] Lower values indicate better quality of sleep.

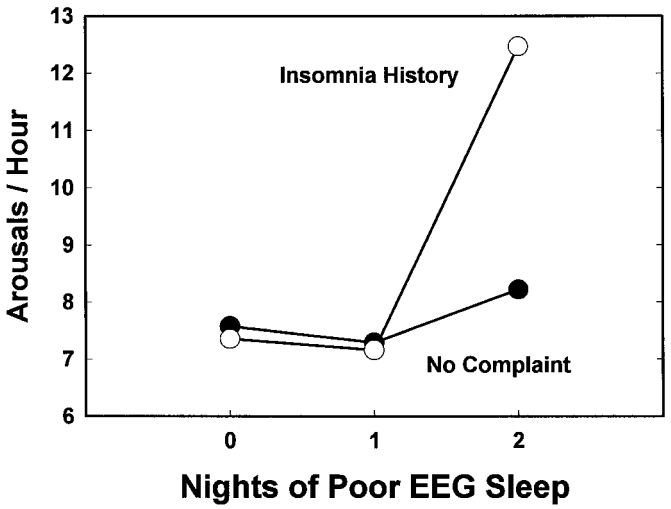

Fig. 2. EEG arousals per hour of sleep for each complaint group as a function of nights of poor EEG sleep.

the group experiencing 1 night of poor EEG sleep usually fell between the extreme groups or were similar to those of the consistently poor EEG sleep group. Poor nights of EEG sleep were associated with being male, older age, and reports on the initial questionnaire of more time awake after sleep onset and, in the laboratory, greater fatigue, more dysphoric moods, and poorer immediate-recall memory performance. Physiologically, poor EEG sleep was associated with longer daytime sleep latencies at all MSLT test times and with higher metabolic rates during nighttime sleep (using waking metabolic rate as a denominator to control for individual differences). Together, these results suggest a state of tense/anxious hyperarousal and daytime hypervigilance associated with EEG sleep disturbance in a progressive or dose-response manner.

Poor EEG sleep was not associated with a reported history of chronic insomnia. The most striking finding in the present study was that a strictly defined group reporting chronic insomnia was no more or less likely than a group with no sleep complaints to have a poor night of EEG sleep as defined by sleep latency or sleep efficiency criteria. Analysis of EEG sleep stage differences also revealed little difference between complaint groups even though the IN group reported their laboratory sleep as consistently and robustly worse than the NC group on all rating scales. These results reinforce the notion that a factor independent of the EEG sleep-wake system exerts a major influence on the experience of sleep in individuals reporting chronic insomnia. Results from daytime laboratory tests showing reliable group main effects but no interactions of complaint group with poor EEG sleep group also are consistent with the notion of an independent factor. Specifically, the differences between complaint groups experiencing poor nights of EEG sleep were no greater than the differences between the complaint groups experiencing good nights of EEG sleep. This parallelism is readily apparent in Figure 1, which illustrates tense mood ratings and immediate-recall memory performance.

TABLE 5. Interactions of Complaint Group With Nights of Poor EEG Sleep

	Nights of Poor EEG Sleep			Mean	(RMSE)[a]	Interaction	
	0	1	2			*F*	*p*
Latency to stage 1 sleep							
NC	07	32	15	18	(23)	3.96	.02
IN	11	20	37	22			
Mean	09	26	26				
Subjective sleep latency							
NC	20	31	28	26	(40)	4.38	.02
IN	39	50	94	61			
Mean	29	40	61				
Subjective time awake after sleep onset							
NC	18	40	58	40	(65)	2.51	.10
IN	40	71	140	84			
Mean	29	56	99				
Subjective sleep quality							
NC	2.30	2.34	2.33	2.33	(0.85)	2.35	.10
IN	2.84	3.32	3.58	3.25			
Mean	2.57	2.83	2.96				

[a] RMSE = root mean square error.

Consistent with those results, efforts to determine the best combination of predictors of poor EEG sleep using multiple regression techniques did not include a reported history of trouble sleeping as part of the equation. Regardless of sleep history, the best predictors of poor EEG sleep were tense mood, age, reported time awake after sleep onset, and sex with just 20% of variance explained. Only reported time awake after sleep onset is associated specifically with sleep. Results for this variable might suggest some association between reported insomnia and poor EEG sleep. It must be noted, however, that the association of reported time awake with poor nights EEG sleep did not depend on a *history* of chronic insomnia as defined by a long-term complaint of problems with sleep, which included trouble falling asleep and at least 1 hour of wakefulness after retiring.

The results of the present study suggest the possibility of a two-dimensional organization to sleep and arousal. One dimension distinguishes the complaint groups and is defined by whether there is a subjective sleep problem. On the problematic side of that dimension, patients may or may not have poor EEG sleep, but they certainly have measurable daytime effects associated with their subjective sleep problem. They also have MMPI scores indicating greater pathological profiles. The other axis in the two-dimensional organization is related to EEG sleep. The disturbance side of that dimension is associated with substantial fatigue and negative affect, a performance decrement, and decreased ability to fall asleep during the day. Such disturbance, however, is not associated with perception of sleep as a problem, despite the fact that those with poor EEG sleep did report more time awake during the sleep period on the initial questionnaire and more time awake and longer sleep latencies during their laboratory sleep. The existence of such a two-dimensional system is hypothetical because we cannot yet characterize it according to known physiological mechanisms.

Individuals with both increased non-EEG arousal and poor EEG sleep (the problematic sides of both dimensions) are likely to feel the worst because of the summation of disruptive effects along two independent axes of the hypothesized sleep-arousal system. Those individuals had a distinctly higher rate of brief EEG arousals during sleep that was reflected in one of only two statistically robust interactions in the data. The other robust interaction, the estimate of nighttime sleep latency, also was disproportionately higher in the IN group with two poor EEG nights. That group also had the longest nighttime EEG sleep latencies, although those latencies were not extreme compared with some of those in other groups. Estimates of time awake after sleep onset and overall sleep quality had interaction patterns similar to the others, but they were not statistically robust. The interactions point to some unique features in the IN group with the most EEG sleep disturbance. Those features seem to be restricted to the sleep period, however, because they were not reflected in daytime behavioral, physiological, or subjective measures. Whether this was a result of not having chosen the proper daytime measures is open to question, of course, but the set of measures used was quite comprehensive and has been shown to have high

sensitivity to arousal and sleep-wake manipulations in several previous studies.

The present results suggest that treatment for insomnia should be focused on tension/anxiety and hyperarousal rather than on sleep per se or at least on sleep as indexed by the EEG. Polysomnographic screening for other sleep disorders may still be indicated, however, when sleep apnea or periodic limb movement syndrome is suspected.

Deemphasizing use of electroencephalography in studies of insomnia could be reassuring because it shifts the focus away from (perhaps misguided) attempts to "verify" an insomnia problem by electroencephalographic methods, especially when persistent EEG sleep disturbance will be observed less than 22% of the time (Table 3), and a substantial percentage of those with poor EEG sleep do not report a sleep problem. If polysomnography is considered the "gold standard" for the verification of sleep disorders, the current results make it difficult to define insomnia as a sleep disorder. Just as Kales et al. (28) have reported that limb movements occur during sleep in a large group of asymptomatic individuals, the present study indicates that poor EEG sleep occurs in a large group of individuals who do not report insomnia. Kales et al. (28) questioned whether there is a causal link between limb movements and a sleep complaint. In a similar manner, the current data suggest the possibility that poor EEG sleep is not a causal factor in reported insomnia. Consequently, we may need to reconsider our ideas about what constitutes insomnia and what the appropriate treatment end points might be.

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