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Sleep Is Not Disrupted by Exercise in Patients with Chronic Fatigue Syndrome

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Abstract

Purpose—Patients with CFS report that exertion produces dramatic symptom worsening. We hypothesized this might be due to exacerbation of an underlying sleep disorder which we have previously demonstrated to exist.

Methods—Female patients with CFS and matched healthy controls with no evidence of major depressive disorder were studied with overnight polysomnography on a baseline night and on a night following their performing a maximal exercise test.

Results—CFS patients as a group had evidence for disturbed sleep compared to controls. While exercise improved sleep for healthy subjects, it did not do this for the group as a whole. When we stratified the sample based on self-reported sleepiness after a night's sleep, the patient group with reduced AM sleepiness showed improvement in sleep structure while those with increased AM sleepiness continued to show evidence for sleep disruption. Conclusion: Sleep is disturbed in CFS patients as a group, but exercise does not further exacerbate this sleep disturbance. Approximately half the patients studied actually sleep better after exercise. Therefore, activity-related symptom worsening is not caused by worsened sleep.

Keywords

exertion; unexplained illness; sleep disruption; clinical

INTRODUCTION

Chronic Fatigue Syndrome (CFS) is a medically unexplained condition characterized by persistent or relapsing fatigue lasting at least 6 months, which substantially interferes with normal activities. CFS is primarily a problem in women's health in that it occurs in women

nearly twice as often as in men (5). A disabling and characteristic feature of CFS is that even minimal exertion produces a dramatic worsening of symptoms (6). In earlier work, we showed that activity levels fell several days after patients performed a standard cardiac-type stress test (13). We recently replicated and extended this finding using real-time assessment techniques and demonstrated that CFS symptoms do worsen several days following maximal exercise, but that neither mood nor cognitive function was effected (17). Thus, it is unclear to what extent exercise influences the symptom complex of CFS. Because exercise training is recognized as an important and efficacious treatment option for many patients (1), it is important to understand the degree to which acute exercise influences other debilitating aspects of CFS.

One aspect of CFS and acute exercise that has not been systematically examined is its influence on sleep. We have reported that for several days following exercise, the circadian activity rhythm of the patients but not of the controls lengthened significantly suggesting that the exercise had weakened entrainment to the usual 24 hr zeitgeber (10). We hypothesized that alterations in sleep duration and/or quality might have produced these exercise-related changes. We have recently shown that CFS patients as a group have abnormalities in sleep morphology indicative of sleep disruption (14). Because CFS is identified using a clinical case definition, we reasoned that patients' sleep data would be heterogeneous with some patients sleeping normally and others having disrupted sleep. In our earlier work, we found that we could substantially reduce this heterogeneity by stratifying the patients based on whether they reported increased or decreased sleepiness following a night in the sleep lab. Those that reported being sleepier after a night of sleep showed evidence for disrupted sleep while those that reported some reduction in sleepiness after a night's sleep had relatively normal sleep morphology.

In healthy people sleep duration increases following exercise (18). We hypothesized that the reverse would occur in CFS patients – especially in those reporting increased sleepiness after a night of sleep. To our knowledge, no one has evaluated sleep morphology in CFS patients before and after exercise. The purpose of this study was to fill this gap using a relatively homogeneous CFS patient sample – women without comorbid major depressive disorder and studied while they were in the same menstrual phase – to determine the influence of an acute bout of exercise on polysomnographic and self-reported measures of sleep.

METHODS

Subjects

The subjects were women – 17 with CFS and 16 healthy controls – ranging in age from 25 to 50 years old. Subjects with CFS were either physician referred or self referred in response to media reports about our research. Healthy controls were acquaintances of patients or responded to recruitment flyers. All of these subjects had been screened for sleep disorders by a previous night of diagnostic polysomnography (14), and all were negative. Patients fulfilled the 1994 case definition for CFS (4) and thus had no medical explanation for their symptoms based on history, physical examination or lab tests and no serious psychiatric diagnoses including schizophrenia, eating disorders, substance abuse. Psychiatric diagnosis according to DSM-IV criteria was made using the computerized version of the Diagnostic Interview Schedule (DIS-IV) (Compton et al., St. Louis, MO). This diagnostic interview allowed us to identify subjects with major depressive disorder (n=5) who were then not further studied because depression itself is known to relate with sleep morphology (3). The existence of co-morbid fibromyalgia was diagnosed based on the American College of Rheumatology's 1990 criteria (16). All subjects provided informed consent, approved by the medical school's Institutional Review Board to participate in this research.

Experimental procedures

Following instructions to refrain from alcohol and caffeine ingestion and avoid engaging in prolonged and/or strenuous exercise in the daytime of study nights, subjects underwent polysomnographic recording (PSG) in a quiet, shaded hospital room for an initial habituation night and were excluded if any sleep pathology was identified. Patients enrolled in the study subsequently underwent two additional nighttime polysomnographic studies for the results reported here. Subjects went to bed at their usual bedtime and slept until 7:15 to 8 am the next morning. Exercise was performed in the afternoon before the last study night. Subjects were all studied during the follicular phase of their menstrual cycles.

Measurements

Within 6 months of their habituation session in the sleep lab, subjects returned to the sleep lab for two more nights of study during which they were instrumented to allow recording of electroencephalogram (EEG; C3/A2, O1/A2 and FZ/A2), electrooculogram (EOG), submental electromyogram (EMG), and a lead II electrocardiogram (ECG). The first of these nights was used as a control for the final night prior to which subjects performed the maximal exercise test detailed below. On these two nights, subjects had indwelling venous catheters from which blood was sampled remotely without disturbing the subject three times over the course of the night. Sleep was scored by a single scorer according to standard criteria of Rechtschaffen and Kales (12) every 30 s. Sleep onset was defined as the first three consecutive epochs of sleep Stage 1 or the first epoch of other stages of sleep. An arousal was defined according to standard AASM criteria (2) as a return to alpha or fast frequency EEG activity, well differentiated from the background, lasting at least 3 s but no more than 15 s.

Exercise Procedures

A maximal exercise test was performed on an electronically braked cycle ergometer (Lode Corival, Groningen, The Netherlands) during the late afternoon. The seat height and toe clips were adjusted to the desired fit of the subject. The exercise test began with a 3 minute unloaded warm-up. Following warm-up, exercise began at 20 watts. Exercise intensity then was increased by 5 watts every twenty seconds until volitional exhaustion or the point where the subject could no longer maintain the prescribed minimum pedal rate (60 rpm). During the exercise test, measurements of oxygen consumption (VO_2), carbon dioxide production (VCO_2), and expired ventilatory volume (V_E) were obtained breath-by-breath using a MedGraphics Gas Exchange and Pulmonary Function system (Medical Graphics Corporation, St. Paul, MN). Heart rate was monitored during exercise by ECG using a Quinton Q4000 (Quinton Instruments, Seattle, WA). Due to technical difficulties heart rate data were not acquired on 3 CFS participants and 2 control participants. Participants were encouraged during the test to continue as long as possible and to give their best possible effort. Acceptable effort was determined as achieving 80% of age-predicted maximum heart rate and/or a respiratory exchange ratio (RER) of ≥ 1.1 . All subjects were able to achieve at least one of the two criteria and the majority (13/17 CFS & 13/16 controls) met both criteria. CFS and control groups did not differ significantly in terms of percent peak heart rate (CFS = 0.87 ± 0.06 ; Controls = 0.91 ± 0.07), peak respiratory exchange ratio (RER; CFS = 1.26 ± 0.17 ; Controls = 1.30 ± 0.09), or peak VO_2 (CFS = 20.1 ± 5.4 ml/kg/min; Controls = 24.5 ± 5.1 ml/kg/min).

Sleep continuity

Sleep continuity was evaluated by generating a nonparametric survival curve calculated from the combined data within each group (11,¹⁵) of the varying durations of sequential sleep runs (i.e., continuous epochs of sleep separated from one another by epochs of wakefulness) and was expressed as the median duration of all continuous epochs scored as sleep in each subject. A run of sleep was defined using the sequence of epoch-based sleep stages represented in the

hypnogram. A run began with a change from wake to any stage of sleep. A sleep run continued until there was a change from any stages of sleep to wakefulness. In order to compare sleep continuity between groups, all data from all subjects in each group were pooled and a group survival curve was generated using standard statistical techniques which take into account the multiple runs of sleep in each subject (11, 15); this method was derived from an earlier one (9). We expected to replicate our earlier finding of sleep disruption in the subgroup of CFS patients reporting increased sleepiness following a night's sleep.

Subjective Test

A visual analog scale (0–15.5 cm) was used to estimate perceived sleepiness, fatigue, pain, and feeling blue before and after each PSG recording. Visual analogue scales have consistently been shown to provide valid measures of subjective feelings (8, 14).

Depressed mood

The Centers for Epidemiological Study – Depression (CES-D) scale was used as an indicator of depressed mood. This 20-item scale required respondents to rate how often certain symptoms occurred during the past week on a scale from rarely or none (0) to most all the time (3). Items were summed to yield a total score. High values indicated more depressed mood.

Statistical Analyses

We dichotomized data based on subjects' self-reported sleepiness before and after the baseline PSG night. We labeled those with more sleepiness in the morning than on the night before as “AM sleeper” and those with less sleepiness in the morning than on the night before as “AM less sleepy”. Using this criterion, we were able to divide healthy controls and CFS patients into four groups with almost equal numbers of subjects. Changes of sleepiness before and after sleep as well as changes in the other variables captured via visual analog scale were assessed using paired *t*-test. Differences in measured variables between groups were assessed using non-paired *t*-test or ANOVA. Post hoc analyses used Tukey student range tests. Changes of duration of median sleep run between baseline and post exercise nights for patients with CFS in the AM sleeper and less sleepy groups were assessed using paired *t*-test. Statistical significance was accepted when $P < 0.05$.

RESULTS

Table 1 depicts sleep structures for healthy controls and CFS patients on their baseline and post exercise nights in the sleep laboratory. Baseline total sleep time was similar for controls and patients, but patients had poorer quality sleep than controls as manifested by their having longer durations of Awake plus Stage 1 ($p = 0.049$) as well as reduced REM sleep ($p = 0.013$). Exercise improved sleep compared to the baseline night for both patients and controls: The CFS group showed decreased number of arousals ($p = 0.02$) and shorter duration of Stage 1 sleep ($p = 0.005$), but sleep efficiency remained low and duration of Awake plus Stage 1 sleep remained longer than in controls on the post exercise night. Healthy subjects showed a shortened latency to fall asleep after exercise ($p = 0.025$), while the durations of their individual sleep stages did not change; nonetheless their sleep efficiency did improve enough to be significantly higher than that of patients ($p = 0.016$).

Subjective sleepiness, fatigue, and pain before and after the baseline and post exercise nights were significantly ($p < 0.01$) higher in patients than healthy controls as would be expected (see bottom of Table 1). On the morning after exercise, healthy controls reported significantly less sleepiness ($p = 0.008$) and fatigue ($p = 0.048$) than following the post exercise night. In contrast, on that morning, patients reported no change in their sleepiness and an actual small but significant increase in fatigue than on their baseline night ($p = 0.014$).

Because we had found substantial differences in sleep morphology after stratifying the data based on changes of sleepiness before and after the baseline night in our earlier work (8,¹⁴), we repeated the analysis using similarly stratified data. Number of subjects and range in changes of sleepiness for healthy subjects in the AM less sleepy and AM sleeper groups and CFS patients in the AM less sleepy and AM sleeper groups were 9 and $-9.2 - -2.3$, 7 and $0.0 - 11.3$, 8 and $-11.3 - -0.7$, and 9 and $0.0 - 14.1$. Probably because of our having a much smaller sample size here, we found little difference between the sleep of either patient group and that of either control group (see Table 2). Stratifying by changes of sleepiness did provide further information as to the effects of exercise on sleep, however.

Healthy subjects in the AM sleeper group showed the most dramatic change: Their sleep efficiency increased significantly ($p = 0.033$) from that determined on the baseline night and averaged even higher than for controls in the AM less sleepy group. Commensurate with this improvement, Awake time (with and without Stage 1) decreased ($p = 0.49$ and $p = 0.48$, respectively) and REM sleep increased ($p = 0.49$) relative to values on the baseline night.

While healthy subjects in the AM less sleepy group showed only a significant decrease in sleep latency ($p = 0.032$) from their baseline night, patients in the AM less sleepy group showed further improvement in their sleep structure on the night following exercise. Specifically, total sleep time ($p = 0.011$) and REM sleep increased ($p = 0.048$) relative to the baseline night; in addition, sleep efficiency went up. Duration of the median sleep run (14 ± 7 min) increased ($p = 0.018$) relative to the baseline night (8 ± 5 min). In contrast, although patients in the AM sleeper group did show fewer arousals ($p = 0.007$) and less time in Stage 1 sleep ($p = 0.006$), their sleep efficiency remained low and was in fact significantly lower than any of the other groups studied ($p < 0.05$). Duration of the median sleep run (6 ± 4 min) after exercise did not change from that on the baseline night (6 ± 2 min).

The survival curve of all sleep runs depicted in Figure 1 shows that patients in the AM less sleepy group had a higher percentage of long runs of sleep on the post exercise night compared to their baseline night (i.e., less continuous sleep). For example, the proportion of runs lasting more than 10 min was 45.2% and 54.5% on the baseline and post exercise nights, respectively. This directional change was not seen in AM sleeper patients.

When the entire data set was dichotomized based on the CFS group's median sleep efficiency on the baseline night (i.e., 80%), 4 healthy subjects had poor sleep efficiency too and this group showed improved sleep efficiencies ($p = 0.049$) after exercise while CFS patients in the group with the poor sleep efficiency ($< 80\%$) did not.

Both groups of CFS patients had higher depression scores than both groups of controls on both PSG nights, but means did not exceed the cutoff for mild depression in otherwise well people. As expected, fatigue and pain and scores before and after both study nights were significantly higher in both groups of patients than in both groups of healthy controls ($p < 0.01$). Patients in the AM less sleepy group had increased fatigue ($p = 0.039$) on the morning after exercise relative to their baseline morning. Healthy women in the AM sleeper group reported more sleepiness ($p = 0.043$) on the night after exercise than on the baseline night; no other groups showed this increase in sleepiness. In contrast, patients in the AM sleeper group showed the converse: reduced sleepiness on the night following exercise compared to those in the AM less sleepy group but the highest sleepiness scores ($p < 0.05$) of all the groups on the morning after exercise. Both groups of controls as well as patients in the AM less sleepy group reported reduced sleepiness and reduced fatigue ($p < 0.05$) on the morning after exercise compared with the evening before, but this was not the case for those in the AM sleeper group.

DISCUSSION

As we showed in our previous paper using data from the initial habituation and diagnostic night these subjects spent in the sleep laboratory (14), CFS patients as a group have disrupted sleep characterized by significantly poorer quality sleep than controls. However, in contrast to our expectation, the patients as a group showed evidence of improved sleep after exercise.

The results were clearer after we used the same stratification strategy that we had employed in our earlier work – namely splitting subjects into those who were either sleepier or less sleepy after a night's sleep. Here, as expected, exercise improved the sleep quality of healthy controls who had reported increased AM sleepiness after the baseline sleep night. Contrary to expectation, it had the same result in CFS patients with decreased AM sleepiness. However, patients who reported increased AM sleepiness showed no improvement in sleep disruption, but exercise did not further exacerbate their sleep pathology. These patients also had the lowest average sleep efficiency of any of the groups studied.

Since exercise did not produce a significant worsening of sleep morphology in CFS, the complaints of symptom worsening which are reported to occur the next day after exertion cannot be explained by disruption in sleep. Following exercise, approximately half the patients actually sleep better than on their baseline study night while the rest simply did not improve.

The data from this report and our previous report indicate that a subset of patients with CFS have an underlying sleep disorder characterized by sleep disruption leading to the patients' feeling sleepier after sleep than before it. Our success in being able to stratify patients based on self-report data suggests that collecting data on sleepiness before and after a night of sleep is a useful way of stratifying patients without the need for formal polysomnography. The fact patients showed no worsening in sleep architecture following their performing a maximal exercise test is useful information for patient education. Patients are often afraid to exert themselves because of the fear of symptom worsening following such exertion. While symptom exacerbation may in fact occur, it cannot be due to further disturbances in sleep in that at least half the patients studied here showed actual improvement in their sleep following a rather strenuous exercise. And importantly, the remaining patients showed no further deterioration in their sleep patterns. Although the healthy controls demonstrated greater improvements in sleep, CFS patients demonstrated improvements in time spent awake and arousals during Stage 1 sleep that are similar to that generally seen in healthy good sleepers (18). These data strongly suggest that gentle physical conditioning which has been suggested as a treatment for CFS will also not disrupt sleep and may in fact lead to symptom improvement. This suggestion is consistent with meta-analytic data demonstrating that exercise does not need to be strenuous to improve sleep. Youngstedt et al. (18) reported similar effect sizes on positive sleep outcomes for light, moderate and vigorous exercise in healthy good sleepers.

Unfortunately, there is a paucity of research aimed at determining the influence of exercise on sleep in populations with disturbed sleep. Limited evidence suggests that the sleep-promoting influences of exercise are more pronounced when sleep is disrupted such as in elderly populations, patients with depression or patients with restless leg syndrome (18). Further, exercise has been shown to reduce feelings of depression and anxiety in patients with major depressive and anxiety disorders (7), as well as, in CFS patients (1). However, these studies have relied almost exclusively on self-report measures of sleep quality. The present investigation extends this research by objectively assessing sleep morphology in CFS and demonstrating both the lack of deleterious effects of exercise in patients with the most disturbed sleep and the benefit of sleep promoting effects of exercise in patients with decreased AM sleepiness.

In conclusion, exercise improved sleep structure for healthy volunteers as well as for CFS patients reporting less sleepiness after a night's sleep than before it. In contrast, sleep structure did not change for patients reporting more sleepiness after a night's sleep than before it. In general, these patients were the ones with the lowest sleep efficiencies on baseline sleep polysomnography. Finding that exercise did not worsen sleep leads to two conclusions: first, that any ill effects of exercise are not due to altered sleep and second, that exercise does not have a deleterious effect on sleep and in fact, helps it in some CFS patients. This latter finding should prove important in helping patients deal with increasing their activity without worrying about negative health consequences.

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Fig. 1.

Survival curves of every episode of sleep (i.e., a bout of sleep preceded and followed by periods of wakefulness) for patients in the AM less sleepy and in the AM sleepier groups for whole-night hypnograms on the baseline and post exercise nights stratified by the duration of the sleep episode. In order to compare sleep continuity between groups, all data from all subjects in each group were pooled and a group survival curve was generated using standard statistical techniques (9). Patients in the AM less sleepy group showed a significant shift toward longer bouts of sleep on the post exercise night compared to the baseline night. CFS, chronic fatigue syndrome.

Table 1

Sleep structure and subjective scales of healthy controls and CFS patients during the baseline (B) and post exercise (E) polysomnographic (PSG) night

				Healthy	CFS
n				16	17
age				38 ± 9	41 ± 8
BMI				25 ± 5	25 ± 5
CES-D			B	5 ± 6	14 ± 8 *
			E	4 ± 5	14 ± 8 *
Time in bed	(min)		B	420 ± 30	434 ± 48
			E	425 ± 46	443 ± 55
Total sleep time	(min)		B	368 ± 39	364 ± 46
			E	382 ± 45	376 ± 64
Sleep efficiency ^a	(%)		B	85 ± 9	81 ± 8
			E	89 ± 6	82 ± 11 *
# of arousal	(/hr)		B	10.5	14.7
			E	9.5	10.5 [†]
Awake	(min)		B	52 ± 34	70 ± 34
			E	41 ± 28	67 ± 49 *
Stage 1	(min)		B	52 ± 20	61 ± 33
			E	45 ± 19	47 ± 23 [†]
Awake plus Stage 1	(min)		B	104 ± 40	131 ± 52 *
			E	86 ± 38	114 ± 54 *
Stage 2	(min)		B	212 ± 32	209 ± 31
			E	219 ± 36	215 ± 35
Stage 3	(min)		B	29 ± 24	35 ± 25
			E	38 ± 33	42 ± 26
Stage 4	(min)		B	3 ± 9	4 ± 6
			E	3 ± 10	10 ± 25
SWS (Stage 3+4)	(min)		B	32 ± 28	39 ± 29
			E	41 ± 35	50 ± 33
Stage REM	(min)		B	72 ± 24	55 ± 18 *
			E	76 ± 20	65 ± 27
Sleep latency ^b	(min)		B	22 ± 31	18 ± 13
			E	7 ± 5 [†]	16 ± 24
REM latency ^c	(min)		B	104 ± 91	114 ± 58
			E	76 ± 31	85 ± 26
Sleepiness	@evening		B	4 ± 4	9 ± 4 *
			E	4 ± 3	10 ± 4 *
	@morning		B	3 ± 3	8 ± 4 *
			E	2 ± 2 [#]	9 ± 4 *
Fatigue	@evening		B	2 ± 3	10 ± 3 *
			E	3 ± 3	11 ± 3 *

			Healthy	CFS
Pain	@morning	B	1 ± 2	8 ± 3 *
		E	1 ± 2 [#]	9 ± 3 [†] *
	@evening	B	0 ± 0	7 ± 4 *
		E	0 ± 1	7 ± 4 *
Feeling blue	@morning	B	0 ± 1	7 ± 3 *
		E	0 ± 2	7 ± 4 *
	@evening	B	0 ± 1	1 ± 2
		E	0 ± 0	2 ± 3 *
	@morning	B	0 ± 1	1 ± 2
		E	0 ± 1	1 ± 1

Values are means ± SD. BMI, body mass index; CES-D, Centers for Epidemiological Study -Depression; SWS, slow wave sleep; REM, rapid eye movement; SWS, slow wave sleep; REM, rapid eye movement;

* Significantly different from healthy controls ($p < 0.05$, ANOVA);

[†] Significantly different from baseline night ($p < 0.05$, ANOVA);

[#] Significantly different from evening ($p < 0.05$, paired t-test);

^a total sleep time/time in bed × 100%;

^b time from lights out to sleep onset;

^c time from lights out to first epoch of Stage REM; underline indicates $p < 0.01$.

Table 2

Sleep structure and subjective scales of healthy controls and CFS patients during the baseline (B) and post exercise (E) polysomnographic (PSG) night

		Healthy _{AM,less sleep} [§]	Healthy _{AM,sleeper} [§]	CFS _{AM,less sleep} [§]	CFS _{AM,sleeper} [§]
n(FM)		9 (0)	7 (0)	8 (2)	9 (5)
age		41 ± 8	35 ± 9	39 ± 10	43 ± 6
BMI		25 ± 5	25 ± 5	23 ± 4	27 ± 6
CES-D	B	5 ± 6	5 ± 5	14 ± 9 ^a	14 ± 7 ^a
	E	4 ± 5	5 ± 4	16 ± 8 ^{a,b}	13 ± 7 ^a
<i>Sleep structure</i>					
Time in bed	(min)	421 ± 32	419 ± 30	417 ± 59	448 ± 31
	E	431 ± 34	417 ± 61	457 ± 16 [†]	432 ± 74
Total sleep time	(min)	372 ± 35	363 ± 46	362 ± 57	365 ± 38
	E	382 ± 28	381 ± 64	408 ± 44 [†]	348 ± 68
Sleep efficiency	(%)	86 ± 6	84 ± 11	84 ± 8	78 ± 7
	E	87 ± 5	91 ± 8 [†]	88 ± 11	76 ± 9 ^{a,b,c}
# of arousal	(/hr)	10 ± 5	10 ± 5	12 ± 7	15 ± 7
	E	11 ± 5	8 ± 5	11 ± 7	10 ± 4 [†]
Awake	(min)	48 ± 32	56 ± 39	55 ± 34	83 ± 30
	E	48 ± 24	32 ± 31 [†]	49 ± 49	83 ± 45 ^b
Stage 1	(min)	52 ± 16	51 ± 25	55 ± 35	67 ± 32
	E	49 ± 13	40 ± 24	47 ± 28	47 ± 20 [†]
Awake plus Stage 1	(min)	101 ± 35	108 ± 47	110 ± 59	150 ± 39
	E	97 ± 35	72 ± 39 [†]	95 ± 59	130 ± 48 ^b
Stage 2	(min)	218 ± 27	203 ± 37	200 ± 32	216 ± 30
	E	229 ± 29	207 ± 42	228 ± 20 [†]	204 ± 42
Stage 3	(min)	26 ± 15	33 ± 33	44 ± 24	27 ± 24
	E	36 ± 24	40 ± 43	53 ± 22	33 ± 26
Stage 4	(min)	1 ± 3	5 ± 14	6 ± 7	1 ± 3
	E	1 ± 1	7 ± 15	9 ± 12	11 ± 34
SWS (Stage 3+4)	(min)	27 ± 17	39 ± 39	50 ± 28	29 ± 27
	E	37 ± 25	47 ± 47	62 ± 29	38 ± 34
Stage REM	(min)	75 ± 20	70 ± 30	57 ± 22	53 ± 16

		Healthy _{AM,less sleep} [§]	Healthy _{AM,sleepier} [§]	CFS _{AM,less sleep} [§]	CFS _{AM,sleepier} [§]
	E	68 ± 12	87 ± 24 [†]	72 ± 28 [†]	59 ± 25 ^b
Sleep latency	(min)				
	B	26 ± 38	15 ± 21	15 ± 11	20 ± 15
	E	8 ± 5 [†]	6 ± 5	10 ± 9	22 ± 31
REM latency	(min)				
	B	98 ± 85	112 ± 103	89 ± 44	137 ± 61
	E	83 ± 31	67 ± 29 [†]	68 ± 16	101 ± 24 ^{b,c}
		<i>Visual analog scale (0–15.5)</i>			
Sleepiness	@evening				
	B	6 ± 4	1 ± 1 ^a	11 ± 2 ^{a,b}	7 ± 4 ^{b,c}
	E	4 ± 4	4 ± 3 [†]	13 ± 3 ^{a,b}	8 ± 4 ^c
	@morning				
	B	1 ± 1 [#]	5 ± 4 [#]	6 ± 4 [#] ^a	10 ± 3 [#] ^{a,b,c}
	E	2 ± 2 [#]	1 ± 2 [†] [#]	8 ± 4 [†] [#] ^{a,b}	9 ± 3 ^{a,b}
Fatigue	@evening				
	B	3 ± 3	0 ± 0	10 ± 2 ^{a,b}	9 ± 3 ^{a,b}
	E	4 ± 4	1 ± 2	12 ± 3 ^{a,b}	9 ± 4 ^{a,b}
	@morning				
	B	1 ± 2	1 ± 1	7 ± 3 [#] ^{a,b}	9 ± 3 ^{a,b}
	E	1 ± 3 [#]	1 ± 1	9 ± 3 [†] [#] ^{a,b}	10 ± 3 ^{a,b}
Pain	@evening				
	B	0 ± 0	0 ± 0	8 ± 4 ^{a,b}	6 ± 3 ^{a,b}
	E	0 ± 1	0 ± 0	8 ± 5 ^{a,b}	7 ± 3 ^{a,b}
	@morning				
	B	0 ± 1	0 ± 0	7 ± 4 [#] ^{a,b}	7 ± 2 ^{a,b}
	E	1 ± 3	0 ± 0	7 ± 5 ^{a,b}	8 ± 4 ^{a,b}
Feeling blue	@evening				
	B	0 ± 1	0 ± 0	1 ± 2	1 ± 2
	E	0 ± 0	0 ± 0	2 ± 4	1 ± 2
	@morning				
	B	0 ± 1	0 ± 1	1 ± 2	1 ± 2
	E	0 ± 1	0 ± 0	1 ± 1	1 ± 2

Values are means ± SD.

[§]Data dichotomized based on difference between daytime and nighttime self-reported rating of sleepiness.

^aSignificantly different from healthy in the AM less sleepy group ($p < 0.05$, ANOVA);

^bSignificantly different from healthy in the AM sleeper group ($p < 0.05$, ANOVA);

^cSignificantly different from CFS in the AM less sleepy group ($p < 0.05$, ANOVA);

[†]Significantly different from baseline night ($p < 0.05$, ANOVA).;

[#]Significantly different from evening ($p < 0.05$, paired t-test); underline indicates $p < 0.01$.