

Title: Factors affecting sleep in the critically ill: an observational study

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< first level heading > Abstract

Key words: intensive care, sleep disruption, sound levels, illuminance levels

<second level heading> Purpose

The aim of the current study was to describe the extrinsic and intrinsic factors affecting sleep in critically ill patients and examine potential relationships with sleep quality.

<second level heading> Materials and Methods

Sleep was recorded using polysomnography and self-reports collected in adult patients in intensive care. Sound and illuminance levels were recorded during sleep recording. Objective sleep quality was quantified using total sleep time divided by the number of sleep periods (PSG sleep period time ratio). A regression model was specified using the 'PSG sleep period time ratio' as the dependent variable.

<second level heading> Results

Sleep was highly fragmented. Patients rated noise and light as most sleep disruptive. Continuous equivalent sound levels were 56 dB(A). Median daytime illuminance level was 74 lux and night-time levels were 1 lux. The regression model explained 25% of the variance in sleep quality ($p=0.027$); the presence of an artificial airway was the only statistically significant predictor in the model ($p=0.007$).

<second level heading> Conclusions

The presence of an artificial airway during sleep monitoring was the only significant predictor in the regression model and may suggest that although potentially uncomfortable, an artificial airway may actually promote sleep. This requires further investigation.

Trial registration: Australian New Zealand clinical trial registry (<http://www.anzctr.org.au/>):

ACTRN12610000688088.

< first level heading > Introduction

Patients treated in intensive care units (ICU) frequently experience poor quality sleep (1, 2). The quantity of sleep may be acceptable but it is highly fragmented, thus stage 1 and 2 sleep is prolonged and slow wave and rapid eye movement (REM) sleep is short (1, 2). A multitude of extrinsic and intrinsic sleep disruptive factors many of which are interrelated may be responsible for sleep disruption in ICU patients (3). Intrinsic factors are patient related and include prehospital sleep quality, the inflammatory response, pain and circadian rhythm disruption.

Polysomnographic (PSG) sleep data and data for variables potentially associated with sleep disruption (for example environmental sound and illuminance levels) were collected from 53 ICU patients (1). In order to devise and test future interventions to improve sleep in ICU patients we planned to analyse the data to explore the relative effect of these factors on sleep arousals but as sleep was highly fragmented (median sleep period without waking: 3 m) other methods of analysis were necessary to explore factors that disrupt sleep in ICU patients. We sought to model sleep disruption in this cohort of ICU patients. Thus the aim of the current study was to describe the extrinsic and intrinsic factors affecting sleep in critically ill patients and specifically to:

- a. Examine the relationships between the extrinsic and intrinsic factors affecting sleep and sleep quality
- b. Develop a regression model to explain the variance in sleep quality in ICU patients

< first level heading > Methods

< second level heading > Participants and setting

Adult ICU patients older than 17 years with an anticipated ICU length of stay greater than 24 hours were invited to participate. Exclusion criteria were: history or evidence of sleep disorder (e.g. obstructive sleep apnoea), history or evidence of psychiatric illness, known diagnosis of dementia, drug or alcohol withdrawal at time of screening and central neurological impairment (e.g. brain trauma confirmed by scan, hypoxic brain injury, suspected encephalopathy, seizure disorder or drug overdose).

The study was conducted in a 36 bed general and cardiothoracic adult ICU at a 600 bed metropolitan hospital in Sydney, Australia. This hospital was a tertiary referral facility for specialty services such as cardiac, spinal, renal, neuroscience and burns. Twenty-four hour sleep recording (PSG) took place in five of the eight patient rooms in the ICU. Approval to conduct the study was provided by the Human Research Ethics Committees for the Hospital (protocol number: 0809-201M(SP)) and the University. Patients gave informed consent to participate which was confirmed by their closest proxy who also provided written informed consent. The sleep data collected from participants and some environmental sound and illuminance level data collected in this study have been previously published (1). This paper contains additional information about the data concerning sleep disruptive factors and their relationship(s) to sleep quality.

< second level heading > Instrumentation

< third level heading > *Sleep measurement*

Sleep was measured objectively using polysomnography (PSG) for one 24 hour period (this protocol is described elsewhere (1)) and subjectively at the conclusion of PSG monitoring and in the hospital ward. Patients self reported on the quality their sleep before hospitalisation (Insomnia Severity Index (ISI) (4) and responded to a question in the Sleep in Intensive Care Questionnaire (SICQ) (5)), in ICU (using the Richards Campbell Sleep Questionnaire (RSCQ) (6)) and on the Hospital ward (RCSQ and SICQ)).

Insomnia Severity Index: The ISI was developed to identify clinically significant insomnia (based on Diagnostic and Statistical Manual of Mental Disorders 4th Edition (DSM-IV) (7) diagnostic criteria for the condition). Concurrent validity ($r = 0.65$) was investigated in unpublished work by Morin and reported by Bastien, Vallières et al. (4). Lower scores on this instrument indicate better sleep (a cut off score of 15 is indicative of significant sleep difficulty).

Sleep in Intensive Care Questionnaire: The SICQ was administered to assess the patients' perception of sleep disturbances and their sleep in the ICU. The SICQ (5) contains seven questions, some with more than one item. Responders are requested to rate their overall sleep quality at home and in the ICU (and at three different times during their stay) using the SICQ. In addition, ratings on daytime sleepiness are included, along with sources of perceived sleep disruption and noise. Items are rated

on a scale one to ten. Ten is the most desirable score for items contained in questions one to five and one is the most desirable score for items in questions six to seven. The SICQ was developed in the 1990s in North America to assess ICU patients' sleep quality and the factors which contributed to sleep disruption while they were in ICU (5). There are no reports of formal validation or psychometric tests of the SICQ. However the authors performed pilot testing on 43 patients and as a consequence added question seven regarding noise disruption.

Richards Campbell Sleep Questionnaire: The RCSQ (6) comprises five 100mm visual analogue scales: sleep depth, latency, awakenings, time awake and quality of sleep. Responses are scored by measuring the distance from the low end of the scale to the mark made by the patient. The total score for the RCSQ is calculated by adding the score for each VAS and dividing by five. High scores indicate good quality sleep.

The RCSQ was pilot tested in a medical ICU (n=9, 100% male, 14 nights, (8)) and validated in a more extensive investigation involving 70 male patients (6). The correlation between total RCSQ score and PSG sleep efficiency index (SEI) was moderate, $r = 0.58$ ($p < 0.001$); the total RCSQ score was able to predict 33% of the variance in the SEI (6). There are no published RCSQ data for healthy individuals on which to base a comparison or provide cut off scores for poor, moderate or good sleep.

< third level heading > *Sound level measurement*

A portable sound level meter (SLM) and analyzer (Model 2250) (meeting international standard IEC 61672-1), microphone (Model 4189) attached to a 3.0 metre extension lead and calibrator (Model 4231) (Brüel and Kjaer™, Denmark) were used. Sound level meter software BZ7222 ver 1.5, Frequency analysis software BZ7223 ver 1.5 and logging software BZ7224 ver 1.4.1 were used (Brüel and Kjaer™, Denmark). The SLM was programmed to record sound pressure broadband parameters along with LZ spectra at a sampling and logging frequency of one sample per second for 24 hours during PSG recording. Maximum input level was 141.07 dB and 1/3 octave bandwidth was used for the sound spectra. The 'Logging' mode was used. Calibration was performed prior to each study at 1,000Hz, 94dB as a reference output. The microphone was placed approximately one metre above the patient's head in order to record the sound level the patient was exposed to.

< third level heading > *Illuminance level measurement*

The T-10 illuminance meter (Minolta™) was used to record light levels throughout the 24 hour PSG data collection period. The T-10 is an illuminance meter used by light engineers. It was attached to the laptop computer via a serial port to USB port converter. Automatic calibration occurred when the meter was switched on. A one minute sampling and recording period was used. The illuminance meter sensor was placed on the pillow beside the patient's head in order to record the illuminance level the patient was exposed to as accurately as possible.

< Second level heading > *Data management and analysis*

Clinical and demographic data analysed using SPSS™. Sound level reports were generated using the Utility software for Handheld Analyzers BZ 5503 (Brüel and Kjaer™). Sound and illuminance level data were analysed using Excel™ (Microsoft™). Summary statistics (median sound level for the entire recording, 21.00 - 06.00hrs and 06.00 – 21.00hrs) were calculated.

Normally distributed continuous data were described using means and standard deviations and non-normally distributed continuous data were described using medians and interquartile ranges.

Categorical data were described using frequencies and percentages. Correlation coefficients (Pearson (r) for normally distributed continuous data and Spearman (r_s) for non-normally distributed and point biserial correlation (r_{pb}) for dichotomous data) were used to examine the relationships between environmental factors and sleep quality as measured by PSG and patient self-report (RCSQ).

In order to 'quantify' PSG sleep quality we used the TST divided by the number of sleep periods (PSG sleep period time ratio). We chose this in order to accommodate for the lack of independence of each sleep period (even small amounts of slow wave sleep suppress the propensity to sleep afterwards) and the slight variation in recording times between patients. We also selected this in preference to using the arousal index because in most patients sleep periods were short (a few minutes) and therefore the arousal index was (artificially) low. We theorised arousal index would not be a good indication of sleep quality. There were also insufficient amounts (numbers of patients who experienced) of slow wave and REM sleep to use these variables in multivariate analysis. Patient self-report of sleep quality was defined as the total score for the RCSQ which was administered after the PSG equipment was removed in ICU.

A linear regression model was specified to assess the influence of intrinsic and extrinsic factors on sleep quality as measured by the PSG sleep period time ratio; the model included predictors most highly rated by patients as sleep disruptive (that is sound and illuminance levels). The outcome was PSG sleep period time ratio and the predictors were: number of sounds peaks >100 dB(C), presence of an artificial airway during sleep monitoring, administration of benzodiazepine medications and median daytime (06.00-21.00hrs) illuminance level during PSG sleep monitoring.

< first level heading > Results

< Second level heading > *Patient demographic characteristics and PSG summary statistics*

The patients were predominately male and the majority were admitted to ICU with a nonoperative diagnosis (see Table E1. supplementary material).

Results of the ISI indicate that few patients were troubled with significant symptoms of insomnia prior to hospitalisation. However sleep monitoring with PSG indicated that sleep when they were in the ICU was highly fragmented and the quality was poor (little slow wave and REM sleep). Self-reported sleep quality was significantly poorer in the ICU than prior to hospitalisation (SICQ: 7.06 ± 2.52 versus 4.50 ± 2.14 , $p = <0.05$). The patients also reported poor sleep on the RCSQ (see Table E2. Supplementary material).

< Second level heading > *Extrinsic sleep disruptive factors*

Patients rated noise and light as the most sleep disruptive in ICU (Table 1). Continuous equivalent (56.60 ± 2.16 dB(A)) and background sound levels exceeded WHO standards on sound levels in hospital (47.20 ± 3.41 dB(A)). Illuminance levels were appropriate at night (median: 1 lux) but too dim for normal circadian rhythm during the daytime (median: 74 lux) (Table 1). The minimum median lux level was 23.35 lux and the maximum was 351.00 lux. (Sixteen patients were exposed to a median daytime lux level of 100 or above).

< Second level heading > *Intrinsic sleep disruptive factors*

There were a limited number of potentially sleep disruptive intrinsic factors that were clinically significant, for example mean pain score was low at <2/10 and mean anxiety level was low. However approximately half of the patients had an artificial airway in situ during sleep recording and a third of

the sample were administered benzodiazepine medication during sleep recording (see Table E3. supplementary material). Both of these intrinsic factors were included in the regression model.

< Second level heading > Associations between sleep disruptive factors and quantitative and subjective sleep quality outcomes

The presence of an artificial airway during sleep monitoring was positively associated with the PSG sleep period time ratio ($r_{pb} = 0.40$, $p = 0.004$) (that is the presence of an artificial airway was associated with less sleep disruption); all other potential sleep disruptive factors were poorly associated with the PSG sleep period time ratio (for example the administration of benzodiazepine medications: $r_{pb} = 0.10$, $p = 0.502$, the number of sound peaks $>100\text{dB(C)}$ during PSG recording: $r = -0.18$, $p = 0.228$ and median daytime (06.00-21.00hrs) illuminance level: $r_s = 0.02$, $p = 0.876$).

There were no strong associations between subjective sleep quality in ICU (total RCSQ score) and potential sleep disruptive factors. The correlation coefficients with the total RCSQ score were low, for example for the number of sound peaks $>100\text{dB(C)}$: $r = -0.19$, $p = 0.279$, administration of benzodiazepine medications: $r_{pb} = -0.21$, $p = 0.184$ and presence of an artificial airway: $r_{pb} = 0.13$, $p = 0.425$.

A multiple regression model was developed to assess the effects of each of these variables on sleep quality. The predictor variables used in the model were the number of sound peaks $>100\text{dB(C)}$, the presence of an artificial airway, administration of benzodiazepine medications and the median daytime (06.00-21.00hrs) illuminance level during PSG sleep recording; the dependent variable was PSG sleep period time ratio. The model explained 25% of the variance in sleep quality ($p = 0.027$); the presence of an artificial airway was the only statistically significant predictor in the model ($p = 0.007$) (Table 2).

<first level heading> Discussion

There was evidence of considerable sleep disruption in this cohort of ICU patients. Both qualitative and quantitative measures of sleep indicated that sleep quality was poor; it was highly fragmented, there was little slow wave and REM sleep and patients' self-reports indicated sleep was poor. These findings are comparable to the results of studies over the past three decades examining sleep in ICU patients (2, 9-11).

One of the aims of the current study was to examine the relationships between the extrinsic and intrinsic factors affecting sleep quality in order to elucidate the main source of sleep disruption. The patients rated noise and light levels as the most sleep disruptive on the SICQ and sound pressure levels were higher than the WHO standards for hospitals (12). In addition illuminance levels were not conducive to the encouragement of normal circadian rhythm; a median 74 lux (sixteen patients in the sample were exposed to a median lux level of 100 and above; the maximum was 351 lux) is not sufficiently bright during daytime hours. A daytime lux level of 100 or higher is considered sufficient to suppress melatonin secretion and encourage normal circadian rhythm in most individuals (13). The use of mechanical ventilation is a known impediment to sleep in ICU patients (14). Medications, especially benzodiazepines, are commonly used to induce sleep but are widely understood to suppress slow wave and REM sleep (15). However the only significant correlation between sleep quality and potentially sleep disruptive factor was the 'presence of an artificial airway during sleep monitoring'. This relationship was positive indicating that sleep quality may be better in the presence of an artificial airway. This is surprising because even though the patients in the current study did not specify the presence of an artificial airway as sleep disruptive there are frequent reports by patients in the international literature of the discomfort associated with artificial airways particularly the endotracheal (ET) tube (16-18). For example in a study in which former ICU patients were interviewed while still in hospital 68% remembered the discomfort associated with the ET tube and one third of the patients remembered that ET associated discomfort interfered with their sleep (16).

The other aim of the study was to develop a regression model to explain the variance in sleep quality in ICU patients. A regression model was fitted; a quantitative measure of sleep quality (the ratio between the number of sleep periods without waking and the total sleep time) was the dependent variable and the predictor variables were two intrinsic factors and factors that the patients had rated as most sleep disruptive that is, noise and light. Interestingly we found that the presence of an artificial airway during sleep monitoring was the only variable with a large regression coefficient which was statistically significant but sleep was not found to be related to sedation level measured by the VICS ($r_s = -0.21$, $p=0.13$) or receipt of benzodiazepine medication ($r_{pb} = 0.10$, $p = 0.502$). We are unsure whether this is an anomalous result that cannot be explained or there is some potential explanation that needs further exploration.

This study has several limitations, the primary being the sample size. Polysomnography is a challenging and labour intensive technique when employed to assess sleep in the critically ill. Our study comprised one of the larger sample sizes but there was still considerable variation in demographic and clinical characteristics between patients. Sleep was measured once during the ICU patients' stay in ICU (in one ICU). Serial (or continuous) measurements may have provided a more complete picture of sleep disruptive factors. Intrinsic and extrinsic factors vary greatly during the illness trajectory for example plasma inflammatory mediator levels may be higher, the use of mechanical ventilation is more frequent and activity levels and (concomitant) sound levels tend to be higher during the acute phase. (The ability to measure PSG continuously throughout the patient's ICU stay was limited by the availability of human and equipment resources). Absolutely precise synchronisation of the environmental illuminance and sound level monitoring equipment with the PSG recorder would have provided the opportunity to explore the relationship of awakenings with changes in these parameters. However the resources were not available to do this either.

It is likely for many reasons that our model was not well specified. There are many other factors in ICU likely to affect sleep (the model explained 25% of the variation in the sleep quality) and our sample size was small, though larger than many in other ICU sleep studies. It is also likely that there is a non-linear relationship between sleep quality and quantity and the many factors affecting sleep in ICU. For example the effect of mechanical ventilation and sedative medications on sleep may be associated during procedures or during the acute phase of illness but not at other times. This type of relationship would be difficult to explore for one 24 hour episode of sleep monitoring during the patients' treatment in ICU. It is possible that the sleep disruptive factors are interrelated and therefore associations are difficult to measure and assess. There is considerable difficulty measuring sleep in ICU patients and exploring factors affecting sleep quality in this population. Interrater reliability for sleep was moderate for sleep technicians (1) so this unlikely to be the whole explanation (that is classifying sleep when the patient was just behaviourally 'still'). Although the interrater reliability for the analysis of sleep data was moderate (1), there remains the possibility that some data were misinterpreted; EEG anomalies were present in our data and there are known problems with conventional sleep scoring in this population (19).

There is current comment from ICU sleep researchers concerning the definition of sleep (or restorative state) and the most appropriate method of measuring it in ICU patients (19, 20). Arguably until this is clearly elucidated it will be difficult to be fully confident of the significant causes (contributing factors) for sleep disruption in ICU patients. While this important work is undertaken clinicians may look to evidence from epidemiological studies for suggestions on how the ICU environment may be made more conducive to rest and sleep, for example the sleep disruptive effects of aeroplane noise (21). In addition, the discipline of sleep medicine has much to offer such as findings from research into the effects of hypnotics and sedatives (15, 22).

<first level heading> **Conclusion**

Our study reconfirms and extends the findings of researchers investigating sleep in ICU patients. We developed a regression model to explore the effect of intrinsic and extrinsic factors on sleep in ICU patients. The results support findings from previous publications exploring this phenomenon, that is the assessment of sleep and factors affecting it is problematic in the ICU setting.

The specified model explained 25% of the variation in sleep quality (defined as the TST divided by the number of sleep periods). Surprisingly, the presence of an artificial airway during sleep monitoring had the largest positive effect after controlling for the other variables. This may suggest that although often a source of discomfort, an artificial airway may actually promote sleep. However, this requires further investigation to elucidate a possible underlying mechanism.

<first level heading> **Competing interests**

The authors declare that they have no competing interests.

<first level heading> **Authors contributions**

RE conceived and developed the protocol and design, acquired and analysed the data, wrote the manuscript, SM supervised RE, assisted with the development of the design, interpretation of the data and writing the manuscript. TR advised on data analysis, interpretation of the analysis and assisted with writing the manuscript. All the authors have read and approved the manuscript for publication.

<first level heading> **References**

1. Elliott R, McKinley S, Cistulli P, et al. Characterisation of sleep in intensive care using 24 hour polysomnography: an observational study. *Crit Care*. 2013;17(2):R46.
2. Gehlbach BK, Chapotot F, Leproult R, et al. Temporal disorganization of circadian rhythmicity and sleep-wake regulation in mechanically ventilated patients receiving continuous intravenous sedation. *Sleep*. 2012;35(8):1105-14.
3. Elliott R, McKinley S, Cistulli P. The quality and duration of sleep in the intensive care setting: An integrative review. *Int J Nurs Stud*. 2011;48(3):384-400.
4. Bastien CH, Vallieres A, Morin CM. Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Med*. 2001;2(4):297-307.
5. Freedman NS, Kotzer N, Schwab RJ. Patient perception of sleep quality and etiology of sleep disruption in the intensive care unit. *Am J Respir Crit Care Med*. 1999;159(4 Pt 1):1155-62.
6. Richards KC, O'Sullivan PS, Phillips RL. Measurement of sleep in critically ill patients. *J Nurs Meas*. 2000;8(2):131-44.
7. American Psychiatric Association. Diagnostic and statistical manual of mental disorders : DSM-IV. 4th ed. First MB, editor. Washington, DC: American Psychiatric Association; 1994. 886 p.
8. Richards KC, Bairnsfather L. A description of night sleep patterns in the critical care unit. *Heart Lung*. 1988;17(1):35-42.
9. Fontaine DK. Measurement of nocturnal sleep patterns in trauma patients. *Heart Lung*. 1989;18(4):402-10.
10. Freedman NS, Gazendam J, Levan L, et al. Abnormal sleep/wake cycles and the effect of environmental noise on sleep disruption in the intensive care unit. *Am J Respir Crit Care Med*. 2001;163(2):451-7.
11. Little A, Ethier C, Ayas N, et al. A patient survey of sleep quality in the Intensive Care Unit. *Minerva Anesthesiol*. 2012;78(4):406-14.
12. Berglund B, Lindvall T. Guidelines for community noise. Geneva, Switzerland: World Health Organization; 1999.
13. Lack LC, Wright HR. Chronobiology of sleep in humans. *Cellular and Molecular Life Sciences*. 2007;64(10):1205-15.

14. Delisle S, Ouellet P, Bellemare P, et al. Sleep quality in mechanically ventilated patients: comparison between NAVA and PSV modes. *Ann Intensive Care*. 2011;1(1):42.
15. Bastien CH, LeBlanc M, Carrier J, et al. Sleep EEG power spectra, insomnia, and chronic use of benzodiazepines. *Sleep*. 2003;26(3):313-7.
16. Rotondi AJ, Chelluri L, Sirio C, et al. Patients' recollections of stressful experiences while receiving prolonged mechanical ventilation in an intensive care unit. *Crit Care Med*. 2002;30(4):746-52.
17. Hofhuis JG, Spronk PE, van Stel HF, et al. Experiences of critically ill patients in the ICU. *Intensive and Critical Care Nursing*. 2008;24(5):300-13.
18. Karlsson V, Bergbom I, Forsberg A. The lived experiences of adult intensive care patients who were conscious during mechanical ventilation: a phenomenological-hermeneutic study. *Intensive Crit Care Nurs*. 2012;28(1):6-15.
19. Watson PL, Pandharipande P, Gehlbach BK, et al. Atypical Sleep in Ventilated Patients: Empirical Electroencephalography Findings and the Path Toward Revised ICU Sleep Scoring Criteria. *Crit Care Med*. 2013;41(8):1958-67.
20. Kondili E, Alexopoulou C, Xirouchaki N, et al. Effects of propofol on sleep quality in mechanically ventilated critically ill patients: a physiological study. *Intensive Care Med*. 2012.
21. Muzet A. Environmental noise, sleep and health. *Sleep Medicine Reviews*. 2007;11(2):135-42.
22. Gimenez S, Clos S, Romero S, et al. Effects of olanzapine, risperidone and haloperidol on sleep after a single oral morning dose in healthy volunteers. *Psychopharmacology (Berl)*. 2007;190(4):507-16.

Tables

Table 1. Potential sleep disturbing factors

Patient self reports SICQ item	Mean \pm SD, 0-10
Noise	5.70 \pm 2.75
Light	5.15 \pm 2.61
Nursing interventions	5.05 \pm 2.44
Diagnostic testing	4.49 \pm 2.67
Vital signs	4.25 \pm 2.12
Blood samples	4.01 \pm 2.20
Administration of medications	3.84 \pm 2.12
Environmental sound and illuminance levels	
Sound (n=49)	
Leq ^a mean \pm SD, dB(A)	56.60 \pm 2.16
LF ^b mean \pm SD, dB(A)	47.20 \pm 3.41
Lpeak ^c , mean \pm SD, dB(C)	107.33 \pm 10.32
Sound peaks >100dB(C) per recording, mean \pm SD, n	13.20 \pm 10.37
Illuminance level (n = 45)	
Illuminance level during daytime ^d , median [IQR ^e], lux	74.20 [43.54-139.80]
Highest illuminance level during daytime, lux	3230.00
Lowest illuminance level during daytime, lux	0.06
Illuminance level during night-time ^f , median [IQR ^e], lux	1.7 [1.13-2.52]
Highest illuminance level during night-time, lux	285.00
Lowest illuminance level during night-time, lux	0.00

Leq^a = continuous equivalent sound level, LF^b = background sound level, Lpeak^c = peak sound pressure level, Daytime^d = 0600-2100hrs, IQR^e = interquartile range, night-time^f = 2100-060hrs

Table 2. Multivariate analysis: Regression model of the influence of intrinsic and extrinsic factors on the PSG sleep period time ratio

	unstandardized coefficient	<i>p</i> value
number of sound peaks >100dB(C)	-0.09	0.366
presence of an artificial airway during sleep monitoring	6.82	0.007
administration of benzodiazepine medications	1.07	0.678
median daytime (06.00-21.00hrs) illuminance level	0.02	0.137
dependent variable = PSG sleep period time ratio, $R^2 = 0.246$, $p = 0.027$		

Supplementary material

Table E1. Patient demographic characteristics

Characteristic	Statistic
Age, mean \pm SD ^a , years	58.74 \pm 20.67
Severity of illness (APACHE ^b II) score, mean \pm SD	18.70 \pm 8.20
Male gender, n [%]	36 [68]
Nonoperative diagnosis, n [%]	35 [84]

SD^a = standard deviation, APACHE^b = acute physiology and chronic health evaluation

Table E2. Selected statistics for sleep quality

PSG derived results	
TST ^a , median [IQR ^b], hh:mm	05:00 [02:52-07:14]
Stage 1 and 2, median [IQR], %	96.80 [91.70-100.00]
Stage 3 median [IQR], %	0.00 [0.00-1.05]
REM ^c median [IQR], %	0.00 [0.00-6.00]
Sleep periods during recording period, median [IQR], n	38.00 [19.00-56.50]
PSG sleep period time ratio ^d , mean \pm SD, n	6.75 [4.32-12.20]
Subjective reports of sleep quality	
ISI ^e median [IQR]	5.50 [1.00-13.75]
ISI, \geq 15, n [%]	10 [19]
SICQ ^f (at home), mean \pm SD ^g	7.06 \pm 2.52
SICQ (ICU), mean \pm SD	4.50 \pm 2.14
RCSQ ^h (ICU), mean \pm SD, mm	51.36 \pm 24.42

^aTST = total sleep time, IQR^b = interquartile range, REM^c = Rapid eye movement, PSG sleep period time ratio^d = the ratio of TST divided by the number of sleep periods, ISI^e = Insomnia Severity Index (higher scores indicate more insomnia symptoms), SICQ^f = Sleep in Intensive Care Questionnaire (0-10, 10 is excellent), SD^g = standard deviation RCSQ^h = Richard Campbell Sleep Questionnaire (0-100, 100 is better)

Table E3. Patient clinical characteristics

Factor	Statistic
Pain score, mean \pm SD ^a , 1-10	1.87 \pm 2.66
Sedation level VICS ^b Interaction score, mean \pm SD	27.06 \pm 3.80
Sedation level VICS Calmness score, mean \pm SD	29.00 \pm 2.70
Anxiety (FAS ^c) score, mean \pm SD	2.83 \pm 1.30
Patients receiving benzodiazepine medication, n [%]	16 [30]
Patients with an artificial airway in situ during sleep recording, n [%]	28 [54]

SD^a = standard deviation, VICS^b = Vancouver Interaction and Calmness Scale, FAS^c = Faces Anxiety Scale