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High prevalence of sleep-disordered breathing in the intensive care unit — a cross-sectional study

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participants included in the study.

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Abstract

Purpose—Sleep-disordered breathing may be induced by, exacerbate, or complicate recovery from critical illness. Disordered breathing during sleep, which itself is often fragmented, can go unrecognized in the intensive care unit (ICU). The objective of this study was to investigate the prevalence, severity, and risk factors of sleep-disordered breathing in ICU patients using a single respiratory belt and oxygen saturation signals.

Methods—Patients in three ICUs at Massachusetts General Hospital wore a thoracic respiratory effort belt as part of a clinical trial for up to 7 days and nights. Using a previously developed machine learning algorithm, we processed respiratory and oximetry signals to measure the 3% apnea–hypopnea index (AHI) and estimate AH-specific hypoxic burden and periodic breathing. We trained models to predict AHI categories for 12-h segments from risk factors, including admission variables and bio-signals data, available at the start of these segments.

Results—Of 129 patients, 68% had an AHI ≥ 5; 40% an AHI > 15, and 19% had an AHI > 30 while critically ill. Median [interquartile range] hypoxic burden was 2.8 [0.5, 9.8] at night and 4.2 [1.0, 13.7] %min/h during the day. Of patients with AHI ≥ 5, 26% had periodic breathing. Performance of predicting AHI-categories from risk factors was poor.

Conclusions—Sleep-disordered breathing and sleep apnea events while in the ICU are common and are associated with substantial burden of hypoxia and periodic breathing. Detection is feasible using limited bio-signals, such as respiratory effort and SpO_2 signals, while risk factors were insufficient to predict AHI severity.

Keywords

Apnea; Sleep-disordered breathing; Intensive care unit; Critical care; Machine learning

Introduction

Sleep-related breathing disorders are common and almost 1 billion adults across the globe are suspected to suffer from obstructive sleep apnea [1]. Untreated obstructive and central sleep apnea have been shown to be associated with both morbidity and mortality [2–5]. Given that sleep apnea prevalence increases with age, obesity, and several medical conditions [6], there is likely a high prevalence of undiagnosed sleep apnea in the general

population and in hospitalized patients [7–11]. Patients admitted to an ICU typically have conditions or disease complications putting them at high risk for sleep-disordered breathing.

There are limited data available regarding the prevalence of sleep-disordered breathing in the intensive care unit (ICU) [12–17] and no data regarding hypoxia specifically associated with apnea events in the ICU. Existing studies in the literature have reported a prevalence of sleep-disordered breathing, defined by an AHI > 5, between 47 and 74% in patients in the ICU; however, all studies have relatively low sample sizes (between 14 and 127 patients) and collected data for a maximum of one night per patient only. There is conflicting information in the literature about the risks and outcomes associated with sleep apnea in critically ill patients [18, 19]. The ability to better study the prevalence of sleep-disordered breathing in the ICU would allow for improved understanding of these risks and potential poor outcomes.

The typical diagnostic method for identifying sleep apnea is polysomnography (PSG), which includes a variety of equipment including electroencephalography. This type of study is clinically not feasible in an ICU setting due to a variety of factors including the amount of equipment necessary, the condition of the patient, and the patient's mental status. This makes diagnosis and treatment of sleep apnea during an ICU stay challenging. Typically, if sleep apnea is suspected, patients are referred to a sleep laboratory for polysomnography testing after they leave the hospital. Therefore, there is a need for methods beyond polysomnography to further study the prevalence of sleep-disordered breathing and sleep apnea in the ICU setting. An easily applied and sufficiently accurate method for detection of sleep-disordered breathing apnea may increase the likelihood of treatment during the ICU stay, e.g., using positive airway pressure in non-ventilated patients. There are a variety of options to measure sleep-disordered breathing using a subset of the available signals from PSG, including airflow, electrocardiogram, oximetry signals, and respiratory effort belts. These simplified methods are potential alternatives to full PSG testing. Respiratory belts are convenient to use in a variety of settings, including in the ICU, as they require less equipment, are relatively non-invasive, and typically involve simple setup. Additionally, using a thoracic respiratory effort signals alone to measure sleep-disordered breathing has been shown to provide reliable and valid measurements of sleep apnea in outpatient settings [20–23]. However, there are few studies that explore the use of thoracic respiratory belts, or other simplified apnea detection methods, in the ICU setting [15, 17].

In the present study we (a) assessed the prevalence of sleep-disordered breathing events for patients in the ICU with a wearable respiratory effort belt and oxygen saturation signals over multiple days, (b) aimed to measured event-specific hypoxia and periodic breathing, and (c) investigated whether sleep-disordered breathing prediction from risk factors is sufficient, or if severity can only be assessed using monitoring and biosignal analysis. This study investigated sleep-disordered breathing in patients while they were in the ICU only, not beforehand or afterwards. Therefore, all findings and results reported below are true for the ICU only; if and to what extent sleep-disordered breathing is influenced by preexisting disorders or affects post-ICU sleep-disordered breathing was generally not addressed here.

Methods

Study oversight and dataset

Adult patients were enrolled after written consent in a randomized quadruple blinded clinical trial, investigation of sleep in the intensive care unit (NCT03355053, [24]), at the Massachusetts General Hospital (MGH), USA, from June 2018 to November 2019. Patients were enrolled from three different ICUs — medical ICU, surgical ICU, and medical/surgical ICU. Full inclusion and exclusion criteria for the clinical trial are provided in the electronic supplementary material. Briefly, the aim of the clinical trial was to investigate the effects of low-dose dexmedetomidine on sleep quality and delirium; patients were randomized into three groups where they were infused overnight for 11 h, starting usually at 8 pm, with 0.1 or 0.3 mcg/kg/h dexmedetomidine (low dose) or placebo (normal saline). At the time of conducting the present study, the clinical study was not concluded and due to ongoing blinding, the effects of low-dose dexmedetomidine are not analyzed.

Patients were included in this study if ECG and wearable belt data were successfully acquired and available for further analysis. The study was approved by the MGH Institutional Review Board. The results are reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines [25].

Patients agreed to wear a thoracic respiratory belt (*Airgo*, a CE Class IIa certified wearable medical device, Figure E1, [26]) as part of the trial during their ICU stay both during the day and at night for a minimum of one night and a maximum of seven nights. The device measures respiratory effort as well as actigraphy [26]. The device was not worn when mechanically ventilated, and the cohort contains both patients that were intubated earlier or later in their course of hospitalization (see Table 1). We collected demographic data, information regarding medication administration, ICD-10 codes, laboratory testing results, and vital signs from the hospital's electronic medical records and bedside telemetry monitors, and computed Charlson comorbidity index (CCI) [27] and sequential organ failure assessment (Sofa) [28]. The SpO_2 data was collected with a standard ICU pulse oximeter (Masimo, Switzterland).

For data analysis, we divided the data collected from each patient both into 24-h segments (8 am–8 am) and 12-h segments ("day": 8 am–8 pm, "night": 8 pm–8 am). Throughout the study, we used 24-h segments when we aimed to assess average measures (e.g., for assigning an AHI value for a patient), and 12-h segments when we judged a shorter time window to be clinically more meaningful (e.g., for predicting future AHI within the next 12 h instead of 24 h) or if we were interested in the differences between day and night.

Apnea detection and AHI

The wearable respiratory device uses a 10-Hz sampling rate. We used a previously published machine learning model trained on the recommended American Academy of Sleep Medicine (AASM) scoring criteria [29] (i.e., 3% desaturations qualify as hypopnea) that accurately detects individual "apnea events" (apneas and hypopneas) and calculates the apnea hypopnea index (AHI) using this wearable respiratory signal with SpO_2 signals in a non-ICU setting [20]. In a sleep laboratory cohort with expert labels available (AHI

mean: 11, AHI standard deviation: 13), the validation performance was an area under the receiver operating curve of 0.94 and an area under the precision-recall curve of 0.48, an AHI categorization accuracy of 0.80 and a *r*-squared of 0.92 when predicting AHI values [20]. Briefly, the algorithm extracts respiratory and SpO_2 features at each second within a 90-s context window and utilizes a decision-tree-based ensemble classification method, a random forest machine learning model, to make a binary decision if an apnea event is present. Sleep staging to determine wake vs. sleep states was done using published deep machine learning models based on respiratory and actigraphy signals [30, 31]. Apnea–hypopnea detections while patients were awake are not considered for analysis.

The apnea–hypopnea index (AHI) was determined by the number of apneas per hour of sleep. We defined the AHI of patients to be the maximum AHI obtained for their 24-h periods of monitoring. We defined periods of amplified sleep wake transition instability (ASWTI) (see electronic supplementary material) and repeated the analysis as above with this alternative, more relaxed definition of sleep.

For AHI visualization, a swimmer plot was generated. We divided the data into 24-h segments (8 am–8 am) and 12-h segments ("day": 8 am–8 pm, "night": 8 pm–8 am) and computed the corresponding AHI. Histograms were created to show the distribution of AHI per 12-h segments (days and nights), with a bin size of 2.5 AHI, and a cumulative version showing the empirical cumulative distribution of AHI values.

Sleep-disordered breathing characterization

We determined hypoxic burden, a respiratory event-specific hypoxia measure that quantifies the area under the desaturation curve from pre-event baseline, for each 12-h segment [32]. We computed hypoxia measures that do not rely on apnea detections: the number of oxygen desaturations of 3% or more per hour of sleep (ODI) and the fraction of sleep spent under an SpO_2 value of 90.

To detect and assess the prevalence of periodic breathing [33] in the ICU, we computed self-similarity [34] in the respiration signal around detected apneas. See electronic supplementary material for further details.

Subgroup analysis

We computed AHI and the hypoxic burden for subgroups and employed *t*-tests, Mann–Whitney *U* tests, or one-way ANOVA tests to assess differences.

Risk factor analysis

We analyzed the extent to which it is possible to predict the maximum observed AHI using variables available at admission. Briefly, we used a leave-one-subject-out cross validation design and used linear regression, support vector machine, and random forest models. We also analyzed the predictability of the AHI within any given 12-h segment using "admission variables" and lab-, medication-, and vital sign-based variables within the preceding 12 h. The input variables for both tasks as well as more details on the methodology can be found in the electronic supplementary material.

Results

Study oversight and dataset

Out of 189 enrolled patients in the clinical trial, for 129 patients, ECG and wearable belt data were successfully acquired and available for further analysis. Out of these 129 patients, two had unusable respiration data due to poor signal quality. Three patients were excluded due to having less than 1 h of detected sleep while wearing the device. The remaining 124 patients were included in the analysis which yielded 350 12-h data-segments and 265 24-h segments. Across all data that met inclusion criteria, there were 4454.90 h of available respiration data from the wearable respiratory device and 3702.30 total hours of both SpO_2 and respiration data available. For all 24-h segments, the median ([QR] amount of respiration data available was 13.80 [10.90–23.90] hours, and the median [IQR] amount of overlapping respiration and SpO_2 data available was 12.35 [7.30–23.90] hours. The median [IQR] hours of sleep as detected by the sleep staging model were 8.90 [4.47–13.40] hours, and the median [IQR] hours of sleep detected by the amplified sleep wake transition period model was slightly increased at 9.85 [4.88–14.00] hours. Patient demographics can be found in Table 1.

Apnea detection and AHI

There were 84 patients (68%) who had an estimated AHI > 5, 40% had an AHI > 15, and 19% had an AHI > 30 based on the maximum AHI of 24-h segments during their ICU stays. Correspondingly, 55% of all 12-h segments show presence of sleep-disordered breathing(AHI > 5), while 28% and 12% were categorized as moderate and severe AHI, respectively. The mean AHI when including the amplified sleep wake transition instability (ASWTI) periods was slightly higher, see Table 2. Figure 1 shows a swimmer plot for the number of apneas occurring in the ICU and the temporal trends for all patients. The figure shows that the overall prevalence and temporal trends in the medical and surgical ICUs are similar and that the AHI levels per subject over time are relatively stable. Figure 2 shows histograms of the observed AHI in all 12-h segments. We further show 20-min example signals (both respiratory effort and SpO_2) with detected apneas for four patients with varying AHI in Fig. 3.

Sleep-disordered breathing characterization

We calculated the hypoxic burden to determine the severity of the apneas. We found that the median [IQR] hypoxic burden at night was 2.8 [0.5, 9.8] %min/h and during the day was 4.2 [1.0, 13.7] %min/h. The hypoxic burden of 17% of all patients was ≥ 30%min/h, and 8% had a hypoxic burden ≥ 50%min/h. Correlation between apnea-related hypoxic burden and general hypoxia measures was found to be low to moderate ($r = 0.32$, $r^2 = 0.1$ for time below SpO_2 90, $r = 0.62$, $r^2 = 0.39$ for oxygen desaturation index), see Figures E2 and E3. Table 2 shows the results for the hypoxic burden in ICU patients. Additionally, we wanted to determine if there was self-similarity occurring in the signals. These types of breathing patterns are often present in patients with central sleep, periodic breathing, and high loop gain apnea versus obstructive sleep apnea. The percentage of days and nights showing high self-similarity for each category, AHI > 5, AHI > 10, and AHI > 15 was 7.3%,

6.8%, and 7.1% respectively. The percentage of patients with self-similar patterns occurring is presented in Table 2; an example signal is shown in Fig. 4.

Subgroup analysis

AHI and hypoxic burden measures were not statistically different between patients with and without previous OSA diagnosis (median AHI 8.8 and 10.1, p-value = 0.37; hypoxic burden 5.8 and 4.7, p-value = 0.37). There was no significant difference in the amount of maximum oxygen flow rate received between the groups (medians for both groups 2 L/min, p-value = 0.33). The median AHI (9.7 and 6.1) and hypoxic burden (7.2 and 3.6) measures were not significantly larger for 24-h segments when patients received supplemental oxygen supply than when no oxygen was supplied (Mann Whitney U tests: $p = 0.13$ and 0.06 respectively); see Figure E4. Eighty patients had opioids administered at least once while wearing the belt. The percentage of patients who received oxygen therapy was higher in the opioid group (75%, $n = 60$) than in the non-opioid group (48%, $n = 21$, proportion z-test $p = 0.002$). We did not find any significant correlation between the dosage of opioids received and the maximum AHI or hypoxic burden (test results in the electronic supplementary material). Mean cumulative opioid and benzodiazepine doses were not significantly higher in the sleep-disordered breathing group (AHI > 5) than in the non-sleep-disordered breathing group (32 mg and 21 mg fentanyl equivalent respectively, $p = 0.16$; 4 and 2.6 mg midazolam equivalent respectively, $p = 0.62$). Of the patients who were mechanically ventilated before the study period, 32 (25%) showed similar AHI and increased hypoxic burden values compared to non-mechanically ventilated patients: mean (SD) AHIs 16.9 (18.0), 16.7 (16.4), t-statistic − 0.06, $p = 0.95$; mean (SD) hypoxic burden 25.9 (49.0), 14.1 (23.9), t-statistic 1.8, $p = 0.08$). We did not observe statistically significant AHI or hypoxic burden values for patients grouped by diagnoses (ANOVA $p = 0.22$, $p = 0.50$ respectively), see Table E1. Hospital readmission rates within 30 days were similar in the sleep-disordered and non-sleep-disordered breathing patients (26% readmission rates for both groups, chi squared $p = 0.88$; these rates are comparable to those seen in the literature [35].

Risk factor analysis

AHI > 5 was moderately predictable from admission variables using a random forest model (ROC AUC 0.61 [0.504–0.71], PRC AUC 0.78 [0.68–0.87]. For all other AHI thresholds, the ROC value was not significantly different than 0.5. When predicting AHI occurring after a day/night with admission variables and AHI status (AHI > 5, > 10, > 15, > 30) from the preceding 12 h, we found that a logistic regression model was moderately able to predict AHI > 5 within the next 12 h (ROC AUC 0.57 [0.52–0.62], PRC AUC 0.64 [0.57–0.71]). Results were not significant at any other AHI threshold. All prediction results can be found in the electronic supplementary material.

Discussion

The key findings of our study are as follows: (1) sleep-disordered breathing in the ICU is common and undiagnosed, (2) a fifth of all patients showed substantial hypoxic burden (≥ 30%min/h) associated with respiratory events, (3) seven percent of patients with sleep-disordered breathing showed symptoms of high loop gain, (4) opiate use did not

substantially explain sleep-disordered breathing or hypoxia events, (5) patients grouped by diagnoses showed similar mean AHIs; (6) severity of sleep-disordered breathing was only poorly predictable from baseline clinical variables.

Due to the lack of convenient detection methods, little is known about the prevalence of undiagnosed sleep-disordered breathing in the intensive care unit population during their ICU stay. Here, we investigated the prevalence of sleep-disordered breathing in both medical and surgical ICU populations using a single, wearable respiratory belt and SpO_2 with a machine learning model trained to detect apnea events. We found six studies in the literature, see Table 3 (see search parameters in the electronic supplementary material) that similarly aimed to detect sleep-disordered breathing using/from biosignals (respiratory signals, PSGs) while patients were in the ICU. Our study followed each patient the longest (maximum 7 nights) and included 124 subjects, whereas in the literature, there were 14–127 patients studied for a maximum of one night. Prior studies found that the prevalence of sleep apnea (with an AHI > 5) was between 47 and 74% [12–17]. Our results, 68% of patients with an AHI > 5, are consistent with these previous findings, falling towards the higher end of this range. This indicates a large burden of undiagnosed and untreated sleep-disordered breathing in the ICU population. We also found that ~ 19% of patients had an AHI ≥ 30. Sleep-disordered breathing detected during our study only applies to ICU stay — patients with high AHI in the ICU may or may not have sleep-disordered breathing outside of this setting. Likely, parts of the measured AHI can be attributed to pre-existing sleep-disordered breathing, while there are also many factors in the ICU and in critically ill patients that may cause or exacerbate sleep-disordered breathing. Further studies are needed to determine the underlying cause of the detecting sleep-disordered breathing and to determine whether the sleep-disordered breathing will persist after ICU stay.

The apnea-related hypoxic burden, measuring hypoxia severity specifically related to apnea events, has not been previously studied in the ICU. Previous research has shown that a hypoxic burden above 43%min/h had a significant association with cardiovascular disease–related mortality for a sleep laboratory cohort [32]. We found that 17% of patients in our study had a hypoxic burden ≥ 30%min/h, and 8% had a hypoxic burden ≥ 50%min/h; these patients may benefit from screening oximetry post-discharge. Moreover, the low to moderate correlations between apnea-specific hypoxic burden and more general hypoxia indices demonstrate that apnea-specific hypoxia is not well measured with standard desaturation indices — and consequently, that the hypoxic burden provides additional clinical information.

Accurately identifying patients at risk for sleep-disordered breathing in advance would allow for more efficient resource allocation in clinical care. When investigating the predictability of patients' maximum AHI based on admission variables and daily vital, labs and medication variables, we found that only the AHI > 5 prediction was modestly accurate and statistically significant. Therefore, predicting AHI for ICU patients based on clinical variables alone proved difficult. As this is an observational study, all patients are treated with standard care, including interventions that may relate to sleep apnea treatment. This is evident in our dataset, for example, as patients on opioids were more likely to receive oxygen therapy (nasal cannula or continuous positive airway pressure (CPAP)) to prevent/

treat breathing problems that are typically associated with opioids. Patients with oxygen therapy showed a greater AHI and hypoxic burden, *despite* using oxygen. Two possible explanations are that patients with higher risk of sleep apnea may have been identified by clinicians and measures to mitigate sleep apnea were taken, or that patients with occurring oxygen desaturations were treated with oxygen therapy which did not resolve sleep apnea pathophysiology. Furthermore, given those occurring treatments, we did not find any risk factors that might help to identify this severe undiagnosed sleep-disordered breathing. Finally, we observed most patients in the ICU to be compliant with the simple wearable device and it did not significantly disrupt patient care. Sleep apnea and cyclical hypoxia have well-established undesirable consequences including hemodynamic stress, endothelial dysfunction, dysmetabolism, and inflammation[36–41].

While the sample size of our study is larger than of comparable studies in the literature, 124 patients are still a relatively small cohort. We are planning to do a follow-up study when enrollment of the clinical trial is complete, at which time we will have the full sample size available. Currently, we do not obtain statistical significance in the associations between AHI and outcomes such as CAM-S scores and mortality. We believe it is more appropriate to report this with the full sample size.

The gold-standard, defined by the AASM, to assess sleep-disordered breathing and diagnose sleep apnea, remains full polysomnography. In this study, no full PSGs were performed but the method described uses a single respiratory effort band and standard-ICU pulse oximeter. Standard ICU oximeters typically have a lower sampling frequency and potentially different algorithmic settings, which are usually adjustable by the ICU clinicians, compared to sleep PSG oximeter. Full polysomnography would be more accurate yet would have been unfeasible relative to the recording durations we obtained, especially repeatability. Although we have validated the presented approach in 404 subjects against full PSGs and manual single-expert scoring in the sleep laboratory, it is possible that results in the ICU might deviate somewhat. In the sleep laboratory, the validation performance was an area under the receiver operating curve of 0.94 and an area under the precision-recall curve of 0.48, an AHI categorization accuracy of 0.80 and an r-squared of 0.92 when predicting AHI values. Performance concerns are addressed, at least in part, as the model was specifically designed using readily interpretable and clinically significant features for decision-making, which allow us to verify the results by direct visual inspection. Indeed, we have manually reviewed the results extensively and have seen no evidence of differences in belt signal quality between the sleep laboratory and the ICU. Furthermore, artifacts are unlikely to be classified as apnea events because of the preprocessing steps, and because sleep has to be detected and an apnea event has to be detected by two different models — making it less likely that non-physiological artifacts get classified as apnea events. It is more likely that a performance deviation from the published validation study might result due to different and more complex physiological states, sleep architecture and fragmentation, and breathing patterns in the ICU. According to the AASM manual, apnea and hypopnea events need to show drops in peak signal excursion in breathing signals and/or a drop in SpO_2 signals from pre-event baseline. However, pre-event baseline is not specifically defined, resulting in inter-rater disagreement which might be increased in the ICU due to different types of breathing patterns. Therefore, future studies to characterize sleep analysis performance of reduced

and more patient-friendly equipment in the ICU, need not only to validate against PSG but also against scoring by multiple experts to allow comparison of machine-expert with and expert-expert agreement. Without having these data, we may need to be conservative with conclusive statements about diagnoses based on the measured AHI. While the substantial amount of detected apnea events does show a high prevalence of sleep-disordered breathing in the ICU, it remains to be investigated if measured AHI categories reflect a classical diagnosis of sleep apnea.

A second notable limitation of this study is that the clinical trial that these patients were enrolled involved randomizing patients into three groups, where two of the groups received a low dose of dexmedetomidine (0.1 or 0.3 mcg/kg/h) overnight continuously, and the third group received placebo (normal saline). Although previous studies have shown low-dose continuous dexmedetomidine infusions did not result in respiratory depression in ICU patients [42, 43], the effects of dexmedetomidine on neurological, cardiopulmonary, and sleep regulation in ICU populations need to be investigated further. As this is an ongoing quadruple blind trial, we could not analyze the effect of this randomization and could not estimate a potential influence of the medication on our results.

Further limitations of this study include that the study was done in a single center and lacks diversity in terms of race (87% white) and that the cohort of this study was limited to patients fulfilling the inclusion criteria of the associated clinical trial who agreed to wear the respiratory band. Some patients were unable or unwilling to wear the band due to wounds, rib fractures, or behavioral constraints. Thus, the current study is limited by this selection bias. Different types of ICUs (e.g., neurological, general post-operative, cardiothoracic) will have different risk factors and comorbid conditions, and results could be different.

In summary, undiagnosed sleep-disordered breathing is common in the ICU and is associated with a substantial burden of hypoxia and periodic breathing. Our results demonstrate the potential of detecting sleep-disordered breathing without the need for formal polysomnography. The AHI detected may be different from that estimated by conventional polysomnography, due to recorded parameters (no EEG) and patterns of disordered breathing in the ICU which may not be present in the sleep laboratory. Admission data and clinical variables available at the beginning of the night were not sufficient to predict undiagnosed sleep-disordered breathing which shows that accurate and reliable detection of sleep-disordered breathing in the ICU is difficult to do without the use of bio-signals such as respiration signals and SpO2. This highlights the importance of implementing convenient monitoring devices in the ICU setting.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Conflict of interest

Dr. Westover is a co-founder of Beacon Biosignals and reports grants from NIH, during the conduct of the study; Dr. Cash reports other COI from Neuralink, Paradromics, and Synchron, and Beacon Biosignals, outside the submitted work. Dr. Thomas reports personal fees from GLG Councils, Guidepoint Global, and Jazz Pharmaceutics, outside the submitted work. In addition, Dr. Thomas has a patent ECG-spectrogram with royalties paid by MyCardio, LLC, a patent Auto-CPAP with royalties paid by DeVilbiss-Drive, and an unlicensed patent CO2 device for central/complex sleep apnea issued. Dr. Thompson reports consulting for Bayer, Novartis, and Thetis, outside the submitted work. In addition, Dr. Kuller has a patent: Patent US10123724B2 “Breath volume monitoring system and method” issued. Dr. Kuller reports non-financial support from Myair Inc., during the conduct of the study, and non-financial support from Myair Inc, outside the submitted work.

Data Availability

We provide the main code used in this study, the apnea detection models, and de-identified data on our GitHub page: https://github.com/mghcdac/respiratory_event_detection_wearable.

Abbreviations

AHI	Apnea-hypopnea index
ICU	Intensive care unit
PSG	Polysomnography
CCI	Charlson comorbidity index
SOFA	Sequential organ failure assessment
ASWTI	Amplified sleep wake transition instability
ODI	Oxygen desaturation index
SD	Standard deviation
CPAP	Continuous positive airway pressure

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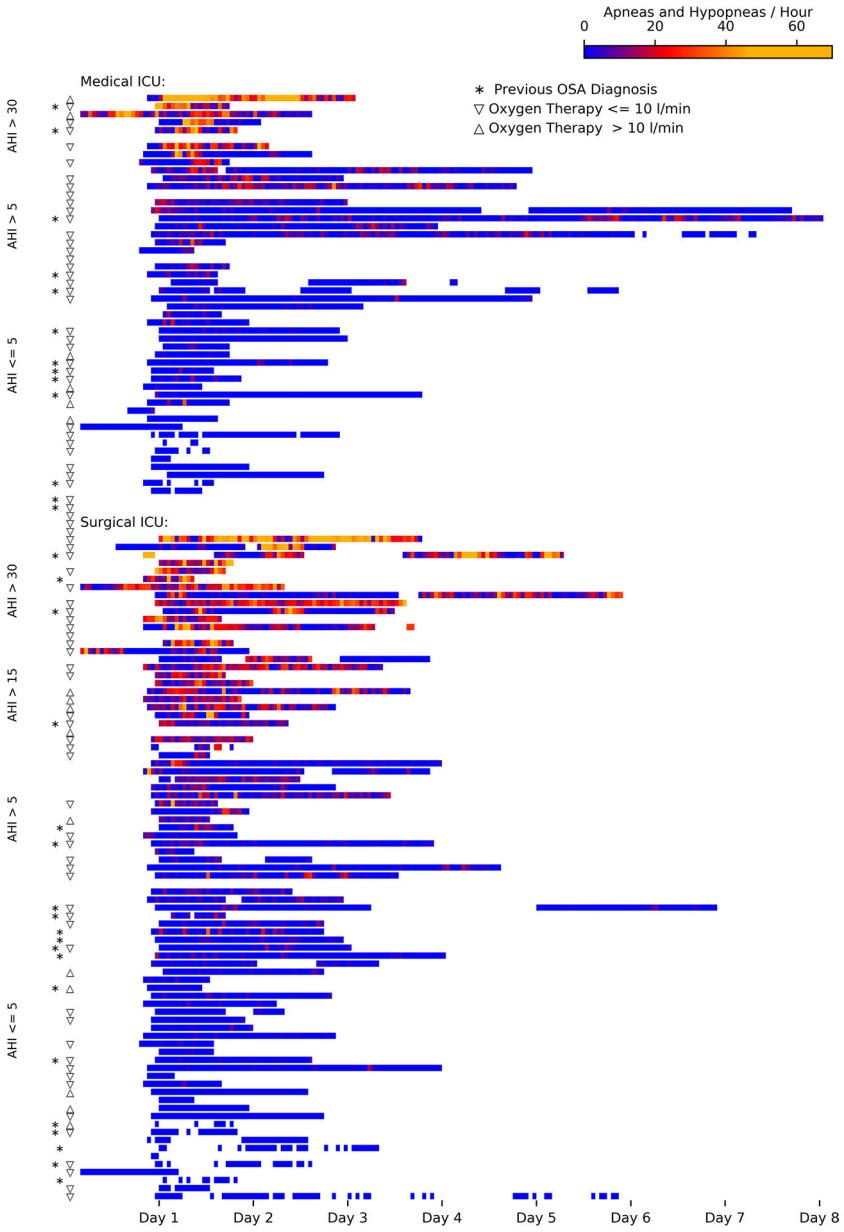

Fig. 1.

Swimmer plot showing the apnea detections for each patient in the study. Each row represents one patient, and each bin represents 1 h colored as the amount of apnea events detected. Apneic events were only detected when patients were asleep. Each patient is aligned to the same day time so that the ticks represent the 20:00 timestamp. Patients have an indication on the plot if they had a previous documented diagnosis of obstructive sleep apnea, and whether or not they were receiving oxygen therapy. The patients are split depending on whether they were admitted to a medical ICU or a surgical ICU

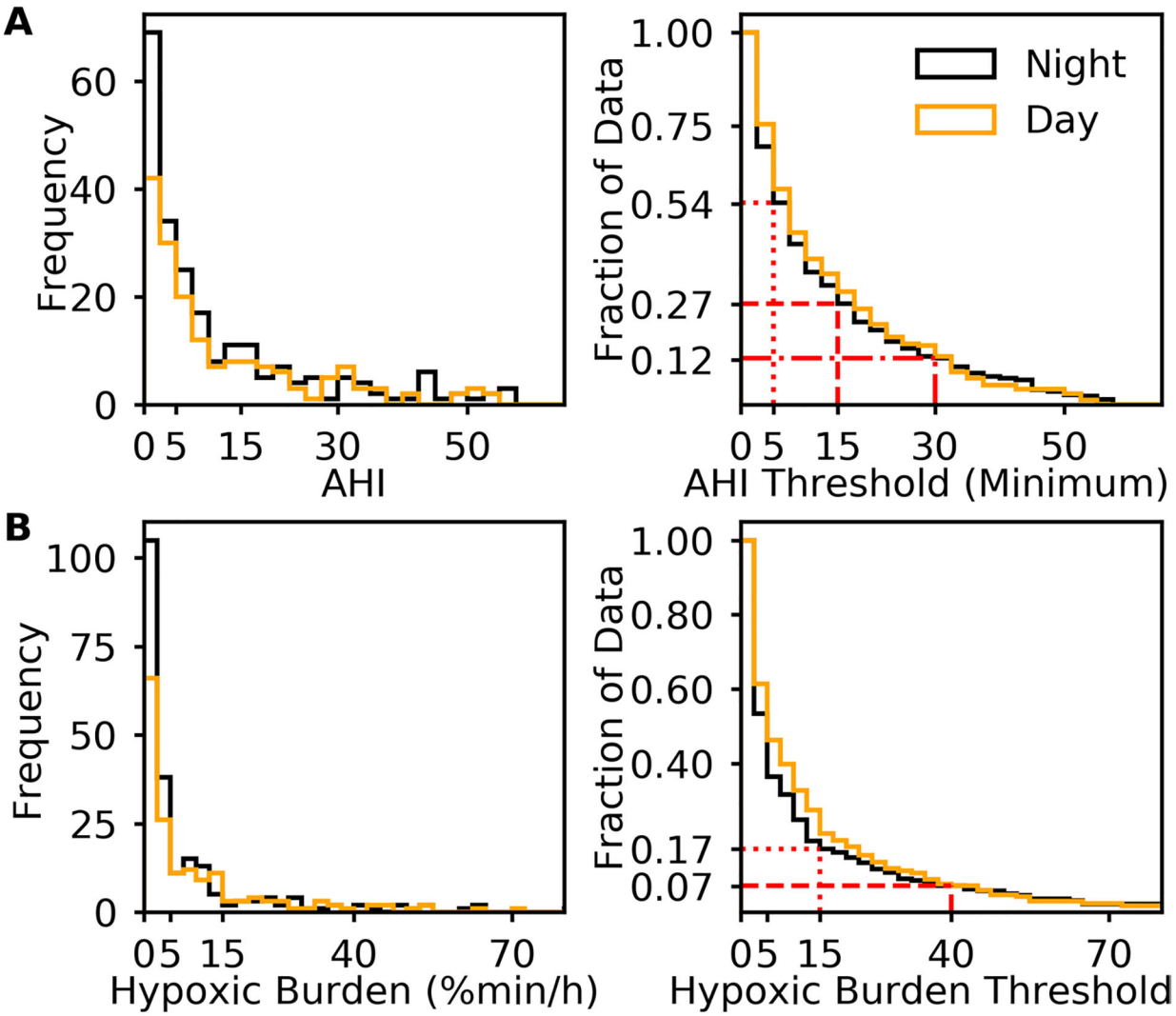

Fig. 2.

AHI and hypoxic burden distribution for 12-h segments. Distributions of apnea–hypopnea index (Panel **A**) and the apnea-specific hypoxic burden (Panel **B**) among all 12-h segments included in the analysis, i.e., at least 90 min of data available and at least 1 h of sleep. 8am–8 pm and 8 pm–8am segments are defined to be “day” and “night” respectively. Left panels show ordinary histograms, and right panels show cumulative histograms. We have found similar amount of AHI levels for day and night periods. Fifty-four percent of all nights show an AHI > 5 and 27% an AHI > 15. Similarly, the hypoxic burden is similar for day and night and 17% of nights show a hypoxic burden larger than 15%desaturation-min/h

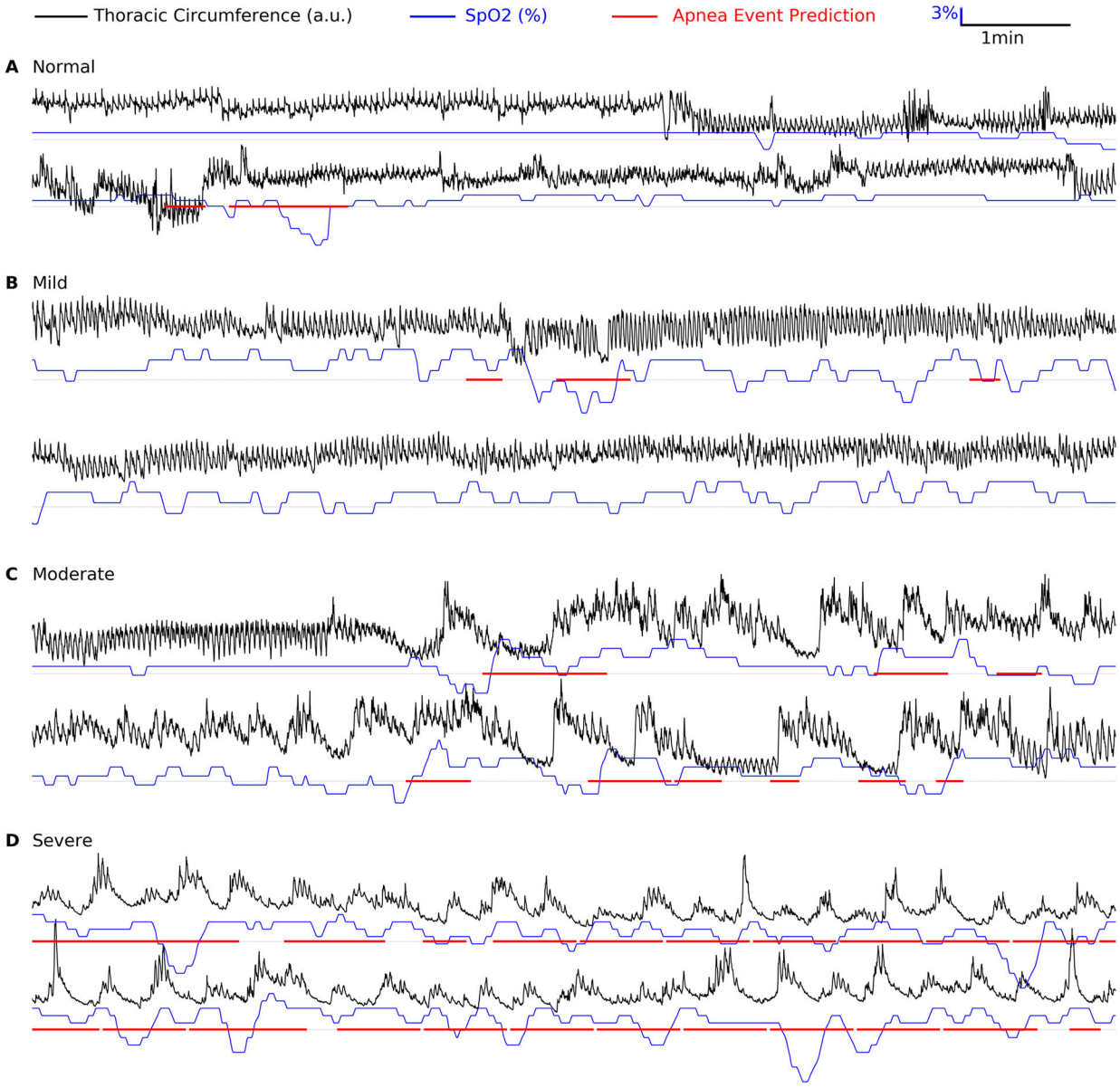

Fig. 3.

Example detection with signal. Twenty-minute example respiratory and SpO_2 signals with detected apnea events for each apnea–hypopnea index category. **A** 70-year-old female with previous OSA diagnosis admitted to medical ICU due to respiratory failure, AHI = 2. **B** 56-year-old male without previous OSA diagnosis admitted to surgical ICU due to myxoid chondrosarcoma, AHI = 12. **C** 75-year-old male without previous OSA diagnosis admitted to surgical ICU after a fall, AHI = 22. **D** 57-year-old male without previous OSA diagnosis admitted to medical ICU due to gastrointestinal bleeding, AHI = 54

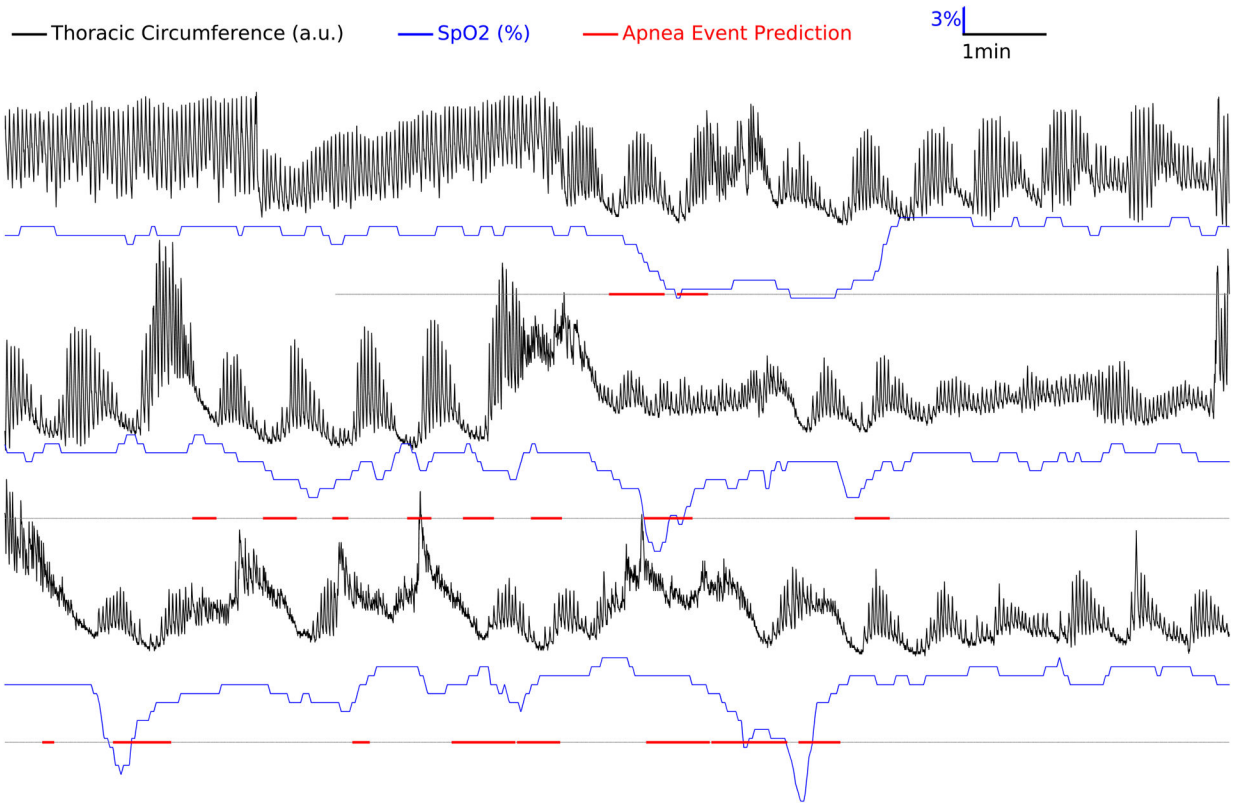

Fig. 4.

Self-similar breathing. Example respiratory and SpO_2 signal with apnea detections and detected high self-similarity (periodic breathing). Patient was female, 88 years old, and admitted to the surgical ICU due to a hip fracture

Table 1

Patient characteristics of the 129 patients included in this study

	n **(%)**
Age (years)[a]	67 (10)
Sex	
Male	72 (58)
Female	52 (42)
Race	
White	108 (87)
Black or African American	4 (3)
Asian	2 (2)
Other	3 (2)
Unknown	7 (6)
Hispanic ethnicity	6 (5)
Unknown hispanic ethnicity	1 (1)
BMI (kg/m^2)[a]	27.4 (6.0)
Wearable belt length (cm)[a]	89 (11)
Charlson comorbidity index	2.3 (2.1)
Weighted CCI	3.6 (3.6)
Previous OSA diagnosis	32 (26)
History of CHF	33 (27)
History of COPD	34 (27)
SOFA score at enrollment	2.30 (2.29)
ICU type	
Medical	45 (36)
Surgical	79 (64)
Primary and/or secondary diagnosis	
Acute kidney injury	33 (27)
Shock	30 (24)
Respiratory failure	27 (22)
Heart failure	23 (19)
Anemia	20 (16)
Sepsis	19 (15)
Pneumonia	15 (12)
Encephalopathy, altered mental status	14 (11)
Pneumothorax, hemothorax,	13 (10)
Pulmonary edema, pleural effusion	
Hemorrhage	10 (8)
GI perforation, incarcerated	
Hernia, SBO, ischemic colitis	8 (6)
Cirrhosis s/p liver transplant	7 (6)

	***n* (%)**
COPD, interstitial lung disease	
ICU length of stay (days)[a]	5 (5)
Hospital length of stay (days)[a]	16 (15)
In-hospital mortality	0 (0)
Three-month mortality	21 (17)
Readmission	
Hospital within 30 days	17 (14)
ICU within 30 days	8 (6)
Emergency department within 30 days	7 (6)
Mechanical Ventilation	
During hospitalization	34 (27)
Before enrollment	26 (21)
During study period	11 (9)
After 14-day inpatient study period	8 (6)
Duration (days) of mechanical ventilation	1 (0.2, 2.8)
Any oxygen therapy during study period	80 (65)
Medications usage within study period	
Opioids used	80 (65)
Fentanyl equivalent (mg)[a]	27.8 (35.0)
Benzodiazepines used	16 (13)
Midazolam equivalent (mg)[a]	3.6 (5.2)
Antipsychotics used	28 (23)
DDD-method equivalent[a]	0.2 (0.3)

[a]Mean (standard deviation),

[b]median (inter-quartile range)

Table 2

Sleep-disordered breathing detection and characterization

	No sleep-disordered breathing AHI ≤ 5	Mild 5 ≤ AHI < 15	Moderate 15 ≤ AHI < 30	Severe 30 ≥ AHI
N(%) patients	42 (34)	32 (26)	26 (21)	24 (19)
N(%) females	20 (38)	13 (25)	9 (17)	10 (19)
N(%) males	22 (31)	19 (26)	17 (24)	14 (19)
With ASWTI-sleep: *N*(%) patients	37 (30)	35 (28)	29 (23)	23 (19)
N(%) BMI < = 25	21 (41)	12 (24)	9 (18)	9 (18)
N(%) BMI > 25	21 (29)	20 (27)	17 (23)	15 (21)
Hypoxic burden (%min/h)	2.1	7.6	17	47.9
Oxygen desaturation Index (3%)	6.3	11.1	14.4	26.6
% Sleep spent with SpO_2 < 90	4	5	3	5
With ASWTI-sleep: Oxygen Desaturation Index (3%)	6.4	11.1	14.3	25.5
With ASWTI-sleep: % sleep spent with SpO_2 < 90	4	5	3	5
AHI mean	1.8	7.0	16.7	31.0
AHI standard deviation	0.9	3.3	6.1	16.5
N(%) patients with self-similarity	9 (21)	11 (26)	5 (19)	5 (21)
N(%) patients with CAM-S < = 3	31 (40)	20 (26)	15 (19)	11 (14)
N(%) patients with 3 < CAM-S < = 6	6 (23)	8 (31)	3 (12)	9 (35)
N(%) patients with CAM-S > 6	5 (25)	3 (15)	8 (40)	4 (20)
N(%) patients with 3-month-mortality	7 (33)	6 (29)	1 (5)	7 (33)
N(%) with ICU LOS < = 3 days	21 (40)	12 (23)	10 (19)	10 (19)
N(%) with ICU LOS > 3 days	21 (30)	20 (28)	16 (23)	14 (20)
N CCI < 2.5	28 (37)	17 (22)	12 (16)	19 (25)
N CCI > 2.5	14 (29)	15 (31)	14 (29)	5 (10)
N without oxygen ever	13 (46)	4 (14)	6 (21)	5 (18)
N with oxygen ever	29 (30)	28 (29)	20 (21)	19 (20)
N without opioids ever	9 (25)	7 (19)	10 (28)	10 (28)
N with opioids ever	33 (38)	25 (28)	16 (18)	14 (16)
N wo/ benzodiazepines	29 (31)	22 (23)	25 (26)	16 (19)
N w/ benzodiazepines	13 (45)	10 (34)	1 (3)	5 (17)
N without CHF	32 (36)	24 (27)	14 (16)	20 (22)
N with CHF	10 (29)	8 (24)	12 (35)	4 (12)
N without COPD	28 (31)	23 (26)	21 (23)	18 (20)
N with COPD	14 (41)	9 (26)	5 (15)	6 (18)

All 129 patients were stratified by baseline or clinical variables (rows), and by their measured apnea–hypopnea index (columns). Abbreviations: *AH*, apnea–hypopnea index; *ASWTI*, amplified sleep wake transition instability; *CCI*, Charlson comorbidity index; *CAM-S*, confusion assessment method score; *ICU LOS*, intensive care unit length of stay; *CHF*, congestive heart failure; *COPD*, chronic obstructive pulmonary disease

Table 3

Literature review

Study	*N*	Method of OSA detection	Number of nights studied	Findings
[12]	64 male critical care unit patients	Polysomnography	1 night	AHI ≥ 5, 47% of patients Oxygen desaturation to ≤ 90%, 61% of sample
[13]	56 ICU patients	Polysomnography	1 night	AHI ≥ 5, 40 patients (71%)
[14]	73 patients admitted to the cardiac ICU	Cardiorespiratory sleep study and/or polysomnography	1 night	Positive sleep study (AHI > 5), 54 patients (74%) Confirmed diagnosis (from outpatient sleep study), 46 patients (63%) Mild OSA (AHI ≥ 5), 14 patients (30%) Moderate OSA (AHI ≥ 15), 11 patients (24%) Severe OSA (AHI ≥ 30), 21 patients (46%)
[15]	127 coronary care unit patients	Respiratory polygraphy	1 night	Non-OSA (AHI < 15), 38 patients OSA (AHI ≥ 15), 89 patients Median [IQR] oxygen desaturation index > 4%/h for non-OSA group, 4.7 [3.5–10.1] Median [IQR] oxygen desaturation index > 4%/h for OSA group, 20.2 [6.6–38.1] Median [IQR] time with SaO_2 < 90% for non-OSA group, 1.6 (0.2–10.0) Median [IQR] time with SaO_2 < 90% for OSA group, 4.20 (0.9–15.6)
[16]	14 patients admitted to an intermediate ICU	Portable polysomnography	1 night	Obstructive sleep apnea syndrome, 10 patients with mean respiratory disorder index h^{-1} (RDI) 60.1 (25.9) Central sleep apnea, 2 patients with mean RDI h^{-1} 45 (28.3) Obesity-hypoventilation syndrome (OHS), 2 patients with mean RDI h^{-1} 12.5 (3.5)
[17]	31 ICU patients	Respiratory polygraph	1 night	Mean obstructive apnea index (OAI) in patients with OAI > 5, 13 ± 6 New diagnosis of OSA in 14 patients (56%)

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