Sleep and Sleep Disordered Breathing in Hospitalized Patients

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Abstract

Sleep is a fundamental physiological process necessary for recovery from acute illness. Unfortunately for hospitalized patients, sleep is often short, fragmented, and poor in quality, and may be associated with adverse outcomes including inpatient delirium. Many factors contribute to poor sleep in the hospital setting, including preexisting sleep deprivation, sleep disordered breathing, environmental noise and light, patient care activities, and medications. Sleep disordered breathing increases the risk of potentially life-threatening cardiovascular, respiratory, and metabolic consequences, and therefore should be diagnosed and treated in hospitalized patients. Mitigating the sequelae associated with poor sleep quality and sleep disordered breathing requires early identification of modifiable factors impacting a patient's sleep, including engagement of a multidisciplinary team. In this article, we review the current knowledge of sleep in hospitalized patients with a detailed focus on patients with sleep disordered breathing.

Keywords
- sleep deprivation
- hospitalization
- intensive care unit
- critical illness
- obstructive sleep apnea

Sleep is a fundamental physiologic process vital for cognition, cardiovascular function, immune function, glucose metabolism, hormone regulation, and recovery from illness. Acute hospitalization, whether in an inpatient ward or intensive care unit (ICU), contributes to sleep loss and decrements in restorative N3 and rapid eye movement (REM) sleep.[1,2] This loss of restorative sleep is believed to precipitate delirium,[3] a risk factor for adverse in-hospital[4–6] and postdischarge outcomes,[7–10] along with long-term consequences such as posttraumatic stress disorder and post-hospital syndrome.[11]

Several factors contribute to poor inpatient sleep quality (see ►**Fig. 1**). Preexisting sleep disorders, including sleep disordered breathing (SDB), contribute to chronic and acute sleep deprivation in patients admitted to the hospital. Moreover, after hospital admission, environmental factors such as noise and light, patient care activities, and medications contribute to acute sleep deprivation and circadian disruption.[12] Despite this knowledge, significant strides have not been made to improve sleep in hospitalized patients. Early recognition and management of undiagnosed SDB and modifiable disruptors of sleep in the hospital setting require multidisciplinary coordination of care, and may improve patient outcomes. In this article, we discuss sleep in the hospital setting, as it pertains to critically ill and noncritically ill inpatients, with an additional focus on patients with existing or undiagnosed SDB.

Basic Sleep Concepts

This review of sleep physiology, measurement, and deprivation is intended to provide a context for practitioners to better identify and understand sleep issues afflicting their hospitalized patients.

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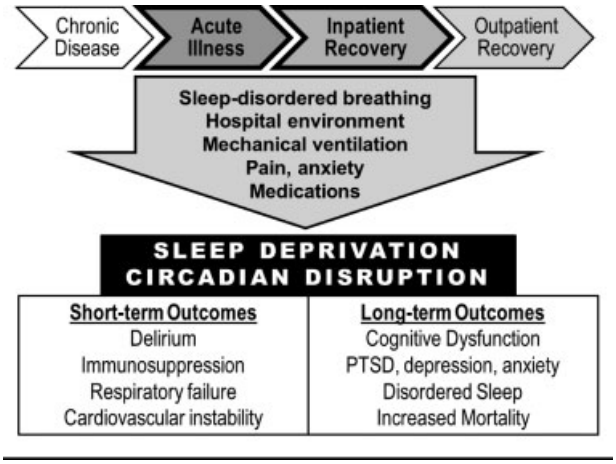

Fig. 1 Proposed model of short- and long-term effects of sleep and circadian disruption associated with acute illness and hospitalization. PTSD, posttraumatic stress disorder.

Normal Human Sleep

Sleep is a periodic, reversible state of cognitive and sensory disengagement from the external environment. Normal overnight sleep in adults typically lasts 7 to 9 hours and consists of four to six 90-to 100-minute periods during which nonrapid eye movement (NREM) and REM sleep alternate in a cyclical fashion (►**Fig. 2**, "normal adult").[13] Wake after sleep onset is minimal, comprising <5% of the overnight sleep period.[14] NREM sleep is comprised of stages N1, N2, and N3, which grossly correlate to depth of sleep. N1 comprises 2 to 5% of a total night's sleep and is characterized by low amplitude, mixed frequency electroencephalogram (EEG) waves. Given a low arousal threshold during N1, this stage is sometimes referred to as "light sleep" and is believed by some experts to represent the transition from wake to sleep, with true sleep not occurring until N2.[14] Following N1 is N2 sleep, which comprises 45 to 55% of a total night's sleep and is characterized by stereotypic EEG findings of K-complexes and sleep spindles.[15] N3 follows N2, comprises 20% of a night's sleep, and is characterized by low frequency, high amplitude delta waves on EEG; N3 is otherwise known as slow wave sleep (SWS). Typically, N3 transitions back to N2 before proceeding to REM sleep, which occupies 20 to 25% of the total sleep period and is made up of tonic and phasic REM. Tonic REM is characterized by skeletal muscle atonia and low voltage, high amplitude, mixed frequency-α, and "saw-tooth" theta waves on EEG. Phasic REM occurs in short bursts intruding intermittently on tonic REM periods, and is characterized by autonomic variability and somatic muscle twitches.[14,15]

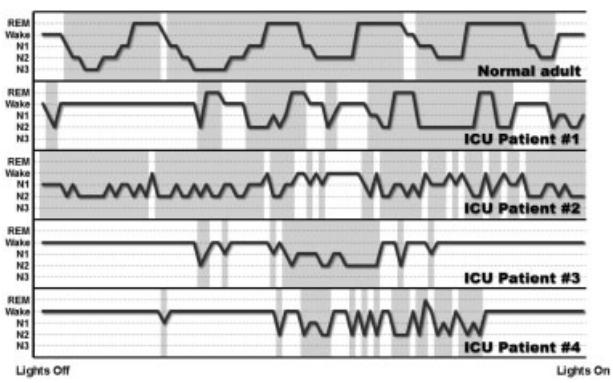

Fig. 2 Hypnograms of overnight sleep in a normal adult and polysomnography recordings from four different ICU patients. Shaded areas represent sleep, and the unshaded areas represent wake. Notably, these four ICU patients demonstrate fragmented sleep with a paucity of REM and an absence of restorative N3 sleep.

Sleep Measurement

The gold standard for sleep measurement is polysomnography (PSG), consisting of EEG, electrooculography, electromyelography, electrocardiography, respiratory flow and effort measurement, pulse oximetry, and a microphone; this allows for the analysis of brain activity, eye movement, muscle, cardiac, and respiratory activity, oxygen saturation, and noise (i.e., snoring), respectively. Because PSG is cumbersome and expensive, especially in the inpatient setting, alternative strategies are available to measure sleep. Actigraphy, which involves a wrist watch type device to detect patient motion, provides a practical and noninvasive tool for sleep measurement but has limited use in the inpatient setting, as it can overestimate sleep in inactive, mostly motionless awake hospitalized patients.[16,17] Alternatively, Bispectral Index (Covidien, Boulder, CO) uses a single integrated EEG forehead electrode to measure brain activity, thus posing a simpler alternative to PSG, but it is limited by artifact, electrode detachment, and a paucity of literature supporting its use for sleep measurement.[18] Given these limitations, subjective instruments such as caregiver observation, the Richards-Campbell Sleep Questionnaire,[19] and the Sleep in the ICU Questionnaire[20] are more commonly used for sleep investigations in the hospital setting. These modalities are inexpensive and can be used on a large scale, but have not been validated in heterogeneous hospital populations. Because of these challenges, sleep measurement remains a major barrier to advancing our understanding and improving poor sleep in the hospital setting.[21–23]

Physiology during Normal Sleep and Sleep Deprivation

Several physiologic changes occur during sleep; those changes most relevant to critical care will be discussed here. During sleep, voluntary control of respiration diminishes, tidal volumes and respiratory rates fluctuate, upper respiratory muscles relax, and hypoxic and hypercapnic ventilatory drives diminish.[24] While these respiratory changes are particularly pronounced during REM sleep,[25,26] NREM sleep is associated with progressive decrements in minute ventilation and a 3 to 7 mm Hg increase in $paCO_2$. In addition, increased parasympathetic tone during NREM results in decreased blood pressure, heart rate, and systemic vascular resistance.[27] Autonomic variability during REM leads to bursts of vagal activity with bradyarrhythmias and sinus pauses during tonic REM, and increased sympathetic activity with increased heart rate and blood pressure during phasic REM.[28]

Growth hormone (GH) and prolactin, anabolic hormones necessary for cell differentiation and proliferation, follow the

sleep–wake cycle and are suppressed during sleep restriction (►**Fig. 3**).[29] GH peaks during early N3, and prolactin peaks during the second half of the overnight sleep period. Similarly, cortisol rises in the early morning and peaks in the late morning, and thyroid-stimulating hormone peaks before sleep onset and declines during sleep. Cortisol loses its periodicity during sleep loss and reentrains during sleep recovery.[30] Thyroid-stimulating hormone is inhibited during N3 and increases with sleep deprivation.[31] As with GH, these hormone changes are theorized to impede patient recovery.

Acute and chronic sleep deprivation can precipitate severe physiologic perturbations and lead to adverse short- and long-term outcomes.[32] The effects of acute sleep loss may be pronounced by preexisting sleep disorders and sleep deprivation. Cognitively, acute sleep deprivation diminishes psychomotor performance, short-term memory, and impairs executive functioning.[33] Mood disturbances also occur, including fatigue, irritability, difficulty concentrating, disorientation, anxiety, depression, and paranoia.[34] From a respiratory standpoint, sleep deprivation may diminish the respiratory response to hypoxemia and hypercapnea.[35] In addition, forced expiratory volume in one second (FEV_1) and forced vital capacity (FVC) have been shown to decrease in sleep-deprived patients with preexisting pulmonary disease.[36] In acute hypercarbic respiratory failure, poor sleep quality may also predict late failure of noninvasive ventilation.[37] Regarding immunity, some animal models suggest diminished immune function after total sleep loss,[38] but this finding is not supported by all studies.[39,40] Sleep loss may also precipitate inflammation and chronic immune dysfunction, leading to end-organ damage which is a proposed etiology of increased cardiovascular disease and cancer in shift worker populations.[41–43]

Sleep Disruption Caused by Hospitalization

Sleep Patterns in the ICU and Inpatient Wards

Sleep in critically ill patients is severely disrupted with increased arousals, decreased REM and N3, difficulty in falling asleep, and scattered distribution of sleep over the 24-hour day, rather than consolidation at night (►**Fig. 2**, "ICU Patients").[44–47] Studies involving 24-hour PSG in mechanically and nonmechanically ventilated medical ICU patients have demonstrated severely reduced overall sleep quantity, elevated N1 and N2 sleep, limited REM sleep, frequent arousals, and increased sleep during the daytime.[1,2,22,48] Similar findings have been seen in surgical ICU patients.[49] Also, approximately 25 to 35% of ICU patients exhibit atypical sleep patterns of unknown clinical significance, characterized by EEG tracings consistent with sleep but lacking sleep spindles, K complexes, and other specific markers of NREM.[22,23,48] These atypical sleep patterns may increase risk of respiratory failure in critically ill patients;[37] however, more research is needed to understand the implications of atypical sleep.

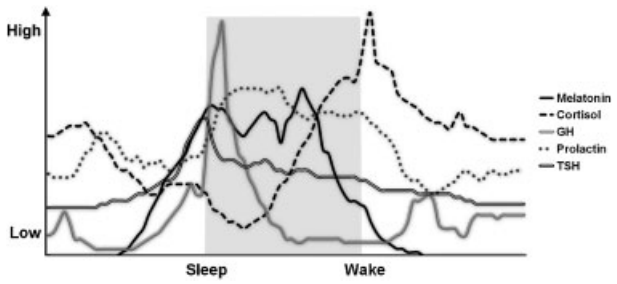

Fig. 3 Levels of melatonin, cortisol, growth hormone (GH), prolactin, and thyroid-stimulating hormone (TSH) across the sleep (shaded rectangular box) and wake cycle in healthy adults. Patients maintained normal habitual sleep–wake cycles, involving activities and meals during the daytime and fasting and sleep at night.

Similar to patients in the ICU, patients on general medical floors experience short sleep duration, poor sleep efficiency, frequent sleep interruptions, and increased daytime sleep. One prospective cohort study involving actigraphy in 118 general medicine inpatients demonstrated a mean sleep duration of 5.5 hours, with 42% of patients reporting noise as major disruptor of sleep.[50] In a similar study also using actigraphy, general medical inpatients aged $\geq$70 years experienced mean sleep times of 3.75 hours and 13 awakenings per night.[51] In addition, 34% of 255 general oncology inpatients reported sleep problems and noted that this complaint was frequently overlooked by care providers.[52]

Environmental Causes of Sleep Disruption in the ICU and General Wards

Noise

Multiple studies demonstrate high noise levels in ICUs,[53,54] with average and peak levels of 43 to 80 A-weighted decibels (dBA) and 80 to 90 dBA,[54–62] respectively, despite Environmental Protection Agency and World Health Organization recommendations for hospital noise maximums of 35 to 45 dBA.[57,63] From the earliest days of critical care, excessive noise has been considered a significant source of stress, anxiety, and negative psychological outcomes in hospitalized patients.[64–77] More recently, noise has been implicated as a common disruptor of sleep in the ICU and general wards.[71,78] Studies involving PSG in critically ill patients have demonstrated a correlation between sound peaks >80 dBA and arousals from sleep,[79] with as many as 17% of nighttime arousals occurring due to noise.[2] Moreover, healthy volunteers exposed to simulated ICU sounds experienced decreased REM sleep[80] and reported prolonged sleep latency, increased arousals, and poorer sleep quality.[81] In the general wards, postoperative orthopedic patients reported that pain (45%) and noise (23%) were the most common factors affecting sleep.[82] Some experts suggest that compliance with overnight sound guidelines may not be possible in modern hospitals due to unavoidable machine noise inside patient rooms. One study demonstrated that recommended noise levels could only be achieved in unoccupied side rooms with all equipment switched off.[54] Although we may need to reconsider our ability to eliminate background machine noise, a reasonable first step toward patient sleep improvement may be to address sound peaks and avoidable disturbing sounds such as staff conversations and unnecessary equipment alarms.

Light

Data on ambient light levels and sleep in the hospital are limited. There are, however, strong theoretical reasons to consider light improvement in any intervention to promote inpatient sleep. Day–night light cycles are the most important determinant in the entrainment of circadian rhythms with the natural decrease of light during evening hours causing a release of melatonin inhibition; the subsequent increase in melatonin promotes sleep. The geriatrics literature suggests that mimicry of normal light cycles improves sleep in institutionalized patients.[83–86] Studies demonstrating poor sleep in general medicine inpatients also revealed that overnight light levels were elevated.[51] Interventions aimed at ICU sleep improvement have demonstrated that light levels can be effectively lowered[87]; however, these findings were linked to sleep measurement via caregiver observation, which overestimates sleep.[88,89] As low light during the day may be as problematic as bright light at night, interventions to promote sleep should also include provision of bright light during the day in conjunction with dimming of lights at night.[90]

Patient Care Interactions

Sleep disturbances in the ICU and general wards may also occur as a result of in–room patient care activities such as medication administration, wound care, bathing, and phlebotomy. Retrospective chart reviews of 147 patient-nights across four ICUs and 180 patient-nights in a surgical ICU demonstrated averages of 42.6 and 51 nocturnal care interactions per room per night shift, respectively.[91,92] Moreover, the surgical ICU investigation noted that bathing and mouth, eye, and wound care occurred most frequently between midnight and 5:00 AM.[92] Another observational study of 1,831 nighttime patient interactions across 200 patients in five ICUs estimated that 13.9% of nocturnal interactions were not time critical.[93] Similarly, on the general wards, an examination of 160 patient-nights in a hematopoietic stem cell transplant unit reported an average of 41 nocturnal care interactions per patient per night.[94] Given that patient care activities occur frequently and are often not time critical, efforts to reschedule and/or cluster nocturnal patient care activities should be considered as part of any sleep promoting intervention.

Patient-Specific Causes of Sleep Disruption in the ICU and General Wards

Mechanical Ventilation

Mechanical ventilation (MV) is a well-known cause of severe sleep disruption. MV is believed to disrupt sleep via patient-ventilator asynchrony, overventilation leading to apneas and subsequent arousals, and inadequate support leading to increased respiratory effort and subsequent arousals.[95] Though extensively studied, there is no strong evidence to support improved patient sleep with one ventilator mode over another.[96] A PSG study of 11 patients comparing assist control (AC) and pressure support ventilation (PSV) demonstrated less sleep fragmentation during PSV; this difference was attributed to diminished patient control of $paCO_2$ in the AC mode.[97] However, a similar comparison of AC, PSV, and clinician-adjusted PSV revealed poor sleep across all modes.[98] A crossover study comparing proportional assist ventilation (PAV) and PSV in 13 patients weaning from MV demonstrated that patient-ventilator asynchrony and sleep arousals were decreased in PAV, resulting in fewer awakenings, and increased REM and SWS.[99] Interestingly, PAV produced lower volume and minute ventilation, resulting in higher overnight $paCO_2$ levels. In contrast, 14 patients with patient-ventilator asynchrony on PSV underwent 24-hour PSG while being exposed to alternating 4-hour periods of PSV and PAV with load adjustable gain (PAV+). During sleep, while patient-ventilator asynchrony events per hour were reduced with PAV+, sleep was more fragmented.[100] Finally, a crossover study of 14 nonsedated patients suggested that neural-adjusted ventilator assist mode could prolong REM, decrease sleep fragmentation, and decrease ineffective respiratory efforts.[101] In the chronically ventilated patient population, a recent study of tracheostomized patients able to tolerate at least 5 hours of separation from MV demonstrated a longer total sleep time but no difference in duration of SWS or REM sleep or fragmentation index with low level PSV, as compared with tracheostomy mask alone.[102] Despite continued research in this emerging area, lack of clinician familiarity and comfort with less conventional modes of ventilation will likely pose a substantial barrier to future ventilator-sleep coordination efforts.

Medications

Many inpatient medications can impact sleep quality (►**Table 1**) and pharmacologic sleep aids are commonly prescribed and requested in the inpatient setting.[103] One of the most common sleep aids is zolpidem, a non-benzodiazepine $GABA_A$–benzodiazepine receptor complex agonist. Because zolpidem does not suppress SWS (as benzodiazepines do), it is considered beneficial for promoting restorative sleep.[104] Other medications perceived to promote sleep are prescribed off-label, may inhibit sleep, and carry untoward side effects.[105,106] One such medication is trazodone, a tetracyclic antidepressant that inhibits serotonin reuptake and antagonizes the H_1 histamine receptor. The number one prescribed medication for insomnia in the United States, trazodone has been shown to subjectively improve sleep and improve sleep efficiency; however, this medication is profoundly sedating and can precipitate life-threatening arrhythmias and drug–drug interactions.[107] In addition, traditional benzodiazepines, also $GABA_A$–benzodiazepine receptor complex agonists with neuroinhibitory properties, can shorten sleep latency and wake time after sleep onset, but profoundly suppress REM and N3.[108,109] Furthermore, antihistamines such as diphenhydramine reversibly antagonize histamine H_1 receptors and inhibit histamine-induced wakefulness, but are sedating and do not improve sleep quality on PSG.[110] Narcotics, though not considered traditional hypnotics, are frequently prescribed in the inpatient setting and are known to provoke nocturnal awakenings, suppress N3 and REM sleep, and cause central apneas.[111–114] The provision of pharmacologic sleep aids to inpatients should

Table 1 Effects of common inpatient medications on sleep

Drug or drug class	Effect on sleep	Mechanism of action
Sedation		
Benzodiazepines	↓W, ↑TST, ↓N3, ↓REM	GABA receptor agonist
Dexmedetomidine	↑N3, ↓SL, ↓REM	α_2-Agonist
Propofol	↑TST, ↓SL, ↓W	GABA receptor agonist
Analgesia		
Opioids	↓TST, ↓N3, ↓REM, ↑W	Mu-receptor agonist
Delirium prophylaxis		
Haloperidol	↑TST, ↑N3, ↑SE, ↓SL, ↓W	Dopamine-receptor antagonist
Olanzapine	↑TST, ↑N3, ↑SE, ↓SL, ↓W	5-HT_2-, D_2-receptor antagonist
Cardiovascular		
β-Blockers	↑W, ↓REM, nightmares	CNS β-receptor antagonist
Dopamine	↓N3, ↓REM	D_2-, β_1-, α_1-receptor agonist
Norepinephrine	↓N3, ↓REM	α- and β-receptor agonist
Epinephrine	↓N3, ↓REM	α- and β-receptor agonist
Phenylephrine	↓N3, ↓REM	α_1-receptor agonist
Sleep aids, other		
Trazodone	↓SL, ± ↑SE, ↑N3, ↑↓REM	Serotonin reuptake inhibitor 5-$HT_{1A,1C,2}$, H_1-receptor antagonist
Zolpidem	↓W, ↑TST, ↓REM	GABA receptor agonist
Antidepressants		
SSRI	↑W, ↓TST, ↓REM	5-HT reuptake inhibition
TCA	↑↓W, ↑↓TST, ↓REM, ↑PLM	5-HT reuptake inhibition
Other		
Antihistamines	↓SL, ± ↑SE, ± ↑N3, ↓REM	H_1-receptor antagonist
Corticosteroids	↑W, ↓N3, ↓REM	Decreases melatonin levels

Abbreviations: CNS, central nervous system; GABA, gamma-aminobutyric acid; N3, deep or slow wave sleep stage; PLM, periodic limb movements; REM, rapid eye movement sleep; SE, sleep efficiency; SL, sleep latency; SSRI, selective serotonin reuptake inhibitors; TCA, tricyclic antidepressants; TST, total sleep time; W, wakefulness after sleep onset; ± ↑, same or improved; ↑, improved; ↓, worsened; ↑↓ variable.

be done vigilantly, given their association with increased falls (zolpidem),[115,116] cognitive side effects,[116] and increased unintended carryover to discharge medication lists.[117]

Sedative use associated with MV also impacts sleep in critically ill patients. A study of 21 sedated mechanically ventilated patients undergoing continuous PSG demonstrated sleep architecture abnormalities and loss of day–night sleep periodicity; only 2 of 21 patients experienced REM.[118] An observational study of 18 patients comparing intermittent sedation, continuous sedation, and continuous sedation with paralytic use showed abnormal sleep architecture, increased SWS, and decreased or no REM sleep on PSG in all three study arms.[119] Patients experiencing daily interruption of sedating benzodiazepine infusions demonstrated, during 24-hour PSG, increased SWS and REM as compared with patients on continuous sedation. However, the continuously sedated group had longer total sleep time and fewer arousals.[120] Newer sedating agents have been investigated for their influence on sleep in the ICU. Neither dexmedetomidine nor propofol demonstrated improvements in REM or SWS in critically ill patients.[121,122] Given these findings, until further data surface, a strategy of minimal sedation with daily interruption of sedation should be promoted in ICU patients; this is consistent with published guidelines.[123]

Sleep Disordered Breathing in the Hospitalized Patient

SDB includes a spectrum of diseases related to disruption of normal respiration during sleep. Grossly, SDB can be divided into diseases of increased airway resistance or closure and/or diminished central drive to breathe. Increased airway resistance resulting in apneas or hypopneas[15] is termed obstructive sleep apnea (OSA). OSA is the most common SDB syndrome and is classified as mild, moderate, and severe based on apnea–hypopnea indices of 5 to 14, 15 to 29, and $\geq$30 events per hour, respectively. Current U.S. prevalence estimates for moderate-to-severe OSA for men and women are 10 and 3% among 30- to 49-year olds, respectively, and 17 and 9% among 50- to 70-year olds.[124] This prevalence is

notably higher in the setting of coronary artery disease (CAD),[125] diabetes,[126] and stroke.[127,128] Central sleep apnea (CSA) and mixed apnea syndromes also occur and are particularly common in patients with systolic and diastolic heart failure (HF). Treatment of SDB is often hampered by underrecognition; approximately 40% of patients remain undiagnosed.[129] In addition, despite well-documented improvement in a variety of important health outcomes, compliance with positive airway pressure (PAP) therapy of patients with OSA remains poor, at approximately 50%.[130,131]

The presence of OSA in an inpatient carries increased risk for complications and poor outcomes during the hospitalization and beyond. For example, in a retrospective cohort study of >250,000 patients at 347 U.S. hospitals, 6.2% of patients hospitalized with pneumonia had OSA and were twice as likely to need invasive ventilation, four times more likely to receive noninvasive ventilation, and used more healthcare resources.[132] Therefore, suspicion for underlying OSA in a hospitalized patient presents a pivotal opportunity for diagnosis and treatment, and prevention of adverse outcomes. Below we discuss specific disease populations at elevated risk.

Cardiovascular Disease and Sleep Disordered Breathing

Untreated OSA is believed to promote cardiovascular disease by various mechanisms, including sympathetic activation,[133] cardiovascular variability,[134] endothelial dysfunction associated with inflammation and oxidative stress,[135] and ventricular dysfunction resulting from intrathoracic pressure fluctuations.[136] OSA is common in patients with CAD and afflicts up to 55% of patients with CAD.[125,137–139] Incident CAD occurs at increased rates in patients with OSA[140] and severe untreated OSA nearly doubles the cardiovascular mortality risk compared with untreated mild-to-moderate OSA, and more than triples the risk compared with those without OSA.[141] In patients with severe OSA and CAD, continuous positive airway pressure (CPAP) therapy reduces the risk for recurrent cardiovascular events.[141–143]

SDB has been observed in 50 to 80% of HF patients with and without preserved ejection fraction.[144–148] CSA including Cheyne–Stokes respiration is also common in patients with HF, and is associated with increased age, male gender, hypocapnia, coexisting atrial fibrillation, and use of diuretics.[149] A randomized controlled trial involving patients with systolic HF suggested that, as compared with medical management alone, 1 month of CPAP improved oxygenation, decreased daytime systolic blood pressure, heart rate, and afterload, and improved left ventricular ejection fraction from 25 to 34%.[150] Recognition of CSA in HF warrants consideration of more advanced noninvasive modes of ventilation such as bilevel PAP and adaptive servo–ventilation.[151,152] Patients with HF and suspected OSA or CSA should be referred for a sleep study and PAP titration.

Cerebrovascular Disease and Sleep Disordered Breathing

As with CAD, cerebrovascular accidents (CVA) and OSA are highly associated. OSA is an independent risk factor for initial and recurrent stroke and is associated with increased length of hospital stay and 6-month mortality following a stroke.[153–155] In addition, development of SDB is common following CVA, with rates ranging from 60 to 96%.[127,128] Factors influencing SDB severity post-CVA include the site and severity of brain ischemia, post-CVA neurological function, and medications.[156] Early recognition and treatment of SDB may prevent further brain damage by attenuating hypoxia-induced vasoconstriction and hypercapnia-induced diversion of cerebral blood flow away from areas of brain supplied by unaffected cerebral vessels.[157] Eighteen months post-CVA, patients unable to tolerate PAP therapy had a fivefold higher risk of recurrent CVA compared with patients utilizing PAP.[158]

Chronic Obstructive Pulmonary Disease and Sleep Disordered Breathing

Patients with chronic obstructive pulmonary disease (COPD) frequently experience poor sleep quality, including increased sleep latency, decreased total sleep time, and increased arousals,[159] and are therefore at risk for profound sleep deprivation while hospitalized. In addition, coexistent COPD and OSA, also known as "overlap syndrome," is common, with approximately 10 to 20% of COPD patients having concurrent OSA.[160] Compared with patients with OSA alone, patients with overlap syndrome have decreased paO_2 and central respiratory drive, and higher $paCO_2$.[161,162] Management of overlap syndrome includes treatment of COPD with inhaled bronchodilators and steroids, which can improve nocturnal oxygen desaturation, subjective sleep quality, total sleep time, and REM duration.[163] In this population, PAP for OSA may specifically attenuate COPD-specific physiological derangements by decreasing upper airway irritation, airway resistance, and auto-PEEP, resulting in improved work of breathing.[164–166]

Improving Sleep in Hospitalized Patients

Current guidelines emphasize the use of a multifaceted, bundled approach aimed at addressing modifiable disruptors of nighttime sleep in the inpatient setting. These interventions include environmental noise and light reduction via "quiet time" protocols[87,88,167–171] and clustering of patient care activities.[123] Earplugs and/or eye masks should also be considered, and have been demonstrated to improve subjective sleep in ICU settings,[172–175] and sleep recorded via PSG in simulated ICU settings.[176,177] Additional small studies support the use of nonpharmacologic therapies such as music therapy,[178] back massage,[179] acupressure,[180] and aromatherapy[181] as safe options for sleep promotion. One ICU study demonstrated the potential benefit of a pharmacologic sleep aid guideline, primarily as a way to discourage medications known to impair sleep, as part of a multifaceted sleep promotion bundle.[3] Finally, as noted earlier, early recognition and treatment of SDB may also be beneficial but, to our knowledge, has not yet been investigated as part of an inpatient sleep promotion bundle.

Several barriers must be considered for any hospital-based sleep promoting intervention, including challenges in

measuring sleep,[3,88,168–171,182,183] mixed buy-in from staff,[87,169,171,182,184] patient acceptance of interventions (i.e., earplugs and eye masks),[3] and lack of proven strategies for intervention sustainability. Nevertheless, given recent investigations demonstrating decreased rates of delirium following interventions to improve sleep in both ICU[3,185] and general medical settings,[186] sleep promotion continues to gain attention as part of efforts to understand and improve patients' recovery from illness and posthospital outcomes. Paramount to the success and sustainability these hospital-based efforts is the involvement of a multidisciplinary team of stakeholders, employment of established quality improvement methods, and consideration of local barriers to implementation.[3] Future hospital-based sleep promotion research should focus on the added benefit of interventions to assess and treat SDB, important associations of sleep improvement and short- and long-term cognitive and physical outcomes, and strategies for long-term intervention sustainability.

Conclusion

Sleep deprivation and circadian disruption is common in inpatients recovering from acute medical illness, especially to the ICU setting. Poor inpatient sleep quality is the result of myriad factors including preexisting sleep disorders, SDB, environmental noise and light, patient care activities, MV, and medications. Patients with SDB represent a particularly vulnerable population for whom hospital admission should serve as an opportunity to aggressively diagnose and treat SDB. Recognition and mitigation of SDB and other modifiable factors affecting sleep quality in the hospital setting must involve a multidisciplinary approach, and may profoundly impact acute and chronic patient outcomes.

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