



# Management of Obstructive Sleep Apnea in Hospitalized Patients

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**Abstract:** Obstructive sleep apnea (OSA) is highly prevalent in the general population. In addition, patients with comorbid OSA are frequently hospitalized for unrelated conditions. This review focuses on managing patients with comorbid OSA in inpatient and acute care settings for inpatient providers. OSA can impact the length of stay, the risk of intubation, the transfer to the intensive care unit, and mortality. Screening questionnaires such as STOP-BANG can help with screening hospitalized patients at admission. High-risk patients can also undergo additional screening with overnight pulse oximetry, which can be used to guide management. Options for empiric treatment include supplemental oxygen, continuous positive airway pressure therapy (CPAP), auto adjusting-PAP, bilevel positive airway pressure therapy (BPAP), or high-flow nasal cannula. In addition, discharge referral to a board-certified sleep physician may help improve these patients' long-term outcomes and decrease readmission risks.

**Keywords:** OSA; SDB; OSA in-hospital; OSA CHF; auto-CPAP; inpatient sleep medicine



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## 1. Introduction

Obstructive sleep apnea (OSA) is a common comorbidity among hospitalized patients [1]. The presence of OSA can influence the outcomes of the presenting illness, the length of hospital stay (LOS), and discharge planning [2]. This review aims to help physicians and other providers in inpatient settings manage comorbid OSA.

The severity of OSA is defined by the apnea-hypopnea index (AHI) available to inpatient providers from polysomnography (PSG) or home apnea testing reports [3]. The AHI is characterized by either complete cessation of airflow (apnea) or partial cessation of breathing with hypoxemia leading to a 3% or 4% desaturation or arousal from sleep (hypopnea). The number of events per hour of sleep recorded during overnight testing is reported as the AHI and describes the severity of the disease. OSA severity is defined as mild (AHI 5–15), moderate (AHI 15–30), or severe (AHI > 30) based on the number of events per hour [4,5]. OSA has been associated with a higher risk of cardiovascular disease [6–8], including sudden cardiac death [9,10], stroke [11], and hypertension [11,12]. OSA can worsen daytime sleepiness and has been linked to increased motor vehicle accidents [13]. Patients with OSA may be more likely to be readmitted to the hospital and have higher healthcare utilization [14].

## 2. Incidence and Prevalence in Hospitalized Patients

The prevalence of OSA has been described as being between 2 and 4% in multiple population-based studies over the last 3 decades in the United States [15–17] and around the world [18–20]. In a recent analysis, it was estimated that almost 1 billion people around the world are affected by OSA [21]. Still, data on sleep disorders in hospitalized patients

are limited [22–28]. Understanding the management of OSA in inpatient settings is vital for inpatient providers, as a majority of the patients with OSA remain undiagnosed and are likely to present to the hospital without a diagnosis of OSA [29]. Male gender and obesity are the most significant risk factors for OSA [15]. Daytime sleepiness, snoring, and pauses in breathing are notable symptoms of OSA. A comprehensive list of signs and symptoms is described in Table 1 [30–32]. These can frequently be missed in hospitalized patients due to the focus on managing the underlying illness.

**Table 1.** Symptoms and signs of OSA [30–32].

Symptoms of OSA	Clinical Exam Findings in OSA
Snoring	Obesity BMI > 30
Choking, gasping or witnessed apnea	Neck circumference > 17 in
Excessive daytime sleepiness, fatigue	Airway crowding ( tongue size relative to bony oropharynx, Retrognathia)
Non-refreshing overnight sleep	CHF signs ( edema, S3, JVD)
Erectile Dysfunction	PH signs ( P2)
GERD	Nocturnal arrhythmias
Neurocognitive impairment (memory, concentration)	Hypertension

BMI, body mass index; in, inches; CHF, congestive heart failure; JVD, jugular venous distention; PH, pulmonary hypertension; GERD, gastroesophageal reflux disease.

A retrospective chart review of 100 inpatient referrals for hospital-based PSG revealed that sleep-disordered breathing (SDB) was present in 77% of the study population [33]. While this study may have been limited by a referral bias, an increase in body mass index (BMI) was associated with an increase in the presence and severity of SDB. In this study, other common comorbid conditions included congestive heart failure (CHF) and chronic obstructive pulmonary disease (COPD) in addition to OSA [33]. In a study of hospitalized patients, screening with the Berlin questionnaire revealed that 40% of patients over the age of 50 were at high risk for OSA [34].

In a prospective study of 148 patients who had a medical emergency team (MET) activation, 50% of the patients screened positive as high risk for OSA, and hypoxemia was the most common reason for MET activation [35]. In another study, patients who screened as high risk for OSA had higher activation of the rapid response team (RRT). However, the number of RRT activations was lower among patients who were compliant with positive airway pressure (PAP) therapy compared to those who were non-compliant or without PAP therapy [36]. The decrease in RRT activation in those treated for OSA suggests that treatment of OSA in hospitalized settings may improve outcomes.

OSA is frequently identified for the first time during the preoperative assessment of patients undergoing elective surgery [37]. OSA in the postoperative population is associated with almost double the odds of cardiac and non-cardiac complications such as respiratory failure, atrial fibrillation, hospital readmission, LOS, and ICU admission [5,38]. OSA in the cardiac surgery population is associated with a higher incidence of postoperative major adverse cardiac or cerebrovascular events (OR 2.4) and a higher risk of paroxysmal atrial fibrillation (OR 1.94) [38,39].

Multiple commonly seen disorders in inpatient settings are associated with comorbid OSA. Acute exacerbation of CHF is a common reason for hospitalization. SDB has been reported in up to 76% of CHF patients, with OSA present in 36% [40]. In patients admitted to the hospital with hypercarbic respiratory failure, OSA prevalence approaches 80–90% [41]. SDB, including OSA, is highly prevalent in patients with cerebrovascular accidents (CVA) such as stroke or transient ischemic attacks (TIA) (incidence of 70% with AHI  $\geq$  5) and is a risk factor for recurrent strokes [42,43]. Although the severity and symptoms of OSA can improve once the acute phase of stroke has passed (3–6 months), they do not resolve

completely, suggesting that OSA may have been present in patients before the index stroke. In pregnancy, OSA is associated with a higher risk of pre-eclampsia, eclampsia, and gestational diabetes mellitus [44].

### 3. Screening for Sleep Apnea in Hospitalized Patients

#### 3.1. Screening Questionnaires

Screening tools for obstructive apnea include the Berlin questionnaire [45], the Epworth sleepiness scale [46], and the STOP-BANG questionnaire [3,47,48]. Screening questionnaires have low diagnostic accuracy, and the American Academy of Sleep Medicine (AASM) discourages their use for diagnosis in outpatient settings [3]. However, screening questionnaires may be more useful in inpatient settings where sleep studies are challenging to obtain [3]. The Berlin questionnaire comprises 11 questions used to stratify OSA risk, but its length makes it less desirable for routine screening use [45]. The Epworth sleepiness scale assesses primarily daytime sleepiness, and due to its focus on symptoms, its utility is limited in hospitalized patients [46]. The questionnaire with the highest utility in hospitalized patients is the STOP-BANG, which includes eight screening questions (four clinical and four demographic) and was developed for screening pre-op patients for OSA risk [47,48]. The STOP-BANG has been validated in both outpatient and inpatient populations [48,49]. Higher scores correlate with a higher risk of OSA. STOP-BANG questionnaire has a higher sensitivity than other questionnaires based on pooled sensitivity analysis (sensitivity 0.93, specificity 0.36, and diagnostic accuracy 53%) [3].

#### 3.2. Pulse Oximetry

Overnight pulse oximetry (ONO) during sleep has inconsistent sensitivity in the outpatient setting and is not recommended as a primary screening method for OSA [50,51]. ONO can be used to calculate the oxygen desaturation index (ODI), which refers to the number of events per hour with a fall in oxygen saturation from baseline by a predefined percentage (3–4%; ODI 3% and ODI 4%) [52,53]. ODI may be comparable to AHI for the diagnosis of OSA in an inpatient setting [25].

For inpatient settings with limited testing capabilities, ONO is a feasible test to set up and can serve as a screening method to stratify patients for the urgency of a referral for sleep studies and attempting empiric treatment while inpatient [24,25]. A limitation of ONO is that a positive test cannot be used for prescribing CPAP. In addition, using ONO to screen for OSA in the hospital may not be feasible for acutely ill patients with cardiopulmonary pathologies because it may be inappropriate for them to tolerate hypoxemia.

High-resolution pulse oximetry (HRPO) uses oximeters calibrated to have a shorter averaging time and a higher resolution unit for SPO<sub>2</sub> signal detection than standard pulse oximeters. It is also less affected by motion artifacts. HPRO is also feasible to set up in an inpatient setting and may have a sensitivity similar to home apnea screening tests [54,55].

#### 3.3. Polysomnography

PSG in a lab setting is the gold standard test for diagnosing OSA [56]. It is usually performed in outpatient settings, and few centers have inpatient sleep lab facilities that can perform full-scale PSG for hospitalized patients. PSG (Type I or Type II) (Table 2) frequently requires dedicated lab equipment or patients to be moved to the sleep lab overnight for testing, which could be in a remote part of the hospital or an adjacent outpatient clinic facility. This might not be feasible for acutely ill patients. With the advent of home sleep apnea testing devices (Type III devices), there have been several studies that have successfully used home testing devices in hospitalized patients for the diagnosis of OSA [23,57]. These results have been validated with follow-up polysomnography [23,57]. Few hospitals have set up sleep apnea testing in the hospital setting, as these tests are not reimbursed during inpatient admissions. However, inpatient testing with HSAT to prescribe PAP therapy to facilitate discharge in healthcare systems with managed healthcare might be cost-effective as it may reduce LOS while compensating for testing costs [58]. In

smaller centers and community hospitals, overnight pulse oximetry followed by referral to a sleep center might be the only cost-effective method.

**Table 2.** Classification of sleep studies.

Type of Sleep Study	Parameters
Type I—Polysomnogram	Continuous monitoring. Done at a lab 7 Channels or more: Cardiac rhythm rate: EKG Oxygen saturation: SPO2 Stages of sleep/wakefulness: EEG Nasal/oral airflow Chin/limb movement (EMG) Chest/abdomen movement Snoring detection
Type II—Polysomnogram	Same parameters as type I but un-attended Also done at a sleep lab
Type III—Portable home sleep apnea testing devices	HSAT fall under type III Could be used at home or hospital 4–7 channels except EEG Variable based on device technology At least 2 respiratory sensors (flow, effort) EKG, SPO2
Type IV—overnight pulse oximetry, high resolution pulse oximetry	1 or 2 channel usually pulse oximetry and EKG. No respiratory flow information

EKG, electrocardiogram; SPO2 pulse oximetry; EEG, Electroencephalogram; Electromyogram EMG; HSAT, home sleep apnea testing.

#### 4. Treatment Options in Hospitalized Patients

##### 4.1. Overview of PAP Therapy Initiation and Titration

Patients with an established diagnosis of OSA and a prescription for PAP therapy should continue their home therapy unless there are contraindications to PAP, as most patients prefer their home equipment (Figure 1). Most hospitals have policies regarding the use of patient devices after evaluation by respiratory therapy or biomedical engineering. For most patients, other institutions provide auto-adjusting or CPAP devices. Upper body elevation and turning to the side are simple adjuncts to treatment that can reduce AHI events [59]. A summary of treatments is provided in Table 3.

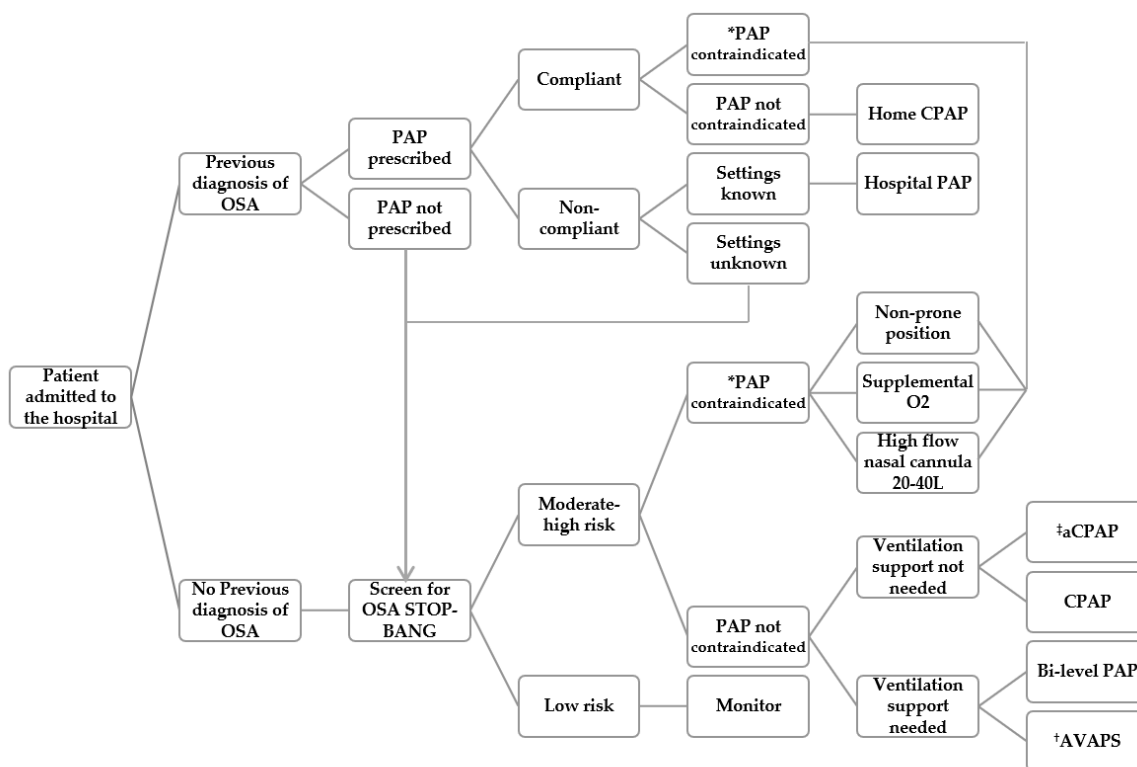
##### 4.2. Auto Titrating CPAP (Auto-CPAP)

Auto-CPAP therapy has been used successfully in the inpatient population [60] (Figure 1). Auto-CPAP devices have built-in feedback mechanisms to assess and titrate pressure to reduce obstructive events or respond to detected snoring [61,62]. After titrating pressure to control events, the devices use feedback to titrate the pressure down until the obstructive events, lower flow, or snoring reappear. They can then titrate the pressure up to eradicate these events [62]. This helps prevent continuous exposure to high pressure. Auto-CPAP is usually set up in a pressure range with upper and lower pressure limits. A pressure range of 4 cm to 15 cm can be a good starting point [63,64]. Auto-CPAP pressure titration has been demonstrated to be similar to CPAP titration in the sleep lab with comparable outcomes [64,65]. Outcome studies in outpatient populations have shown improvements in symptoms of sleepiness, hypercarbia, blood pressure control, and mortality benefits [64–66].

##### 4.3. Bilevel-Positive Airway Pressure Therapy (BPAP)

BPAP devices use two pressures: inspiratory (IPAP) and expiratory (EPAP). BPAP devices are the preferred treatment modality in patients requiring ventilation support, those unable to tolerate high CPAP pressures, and those with conditions such as overlap syndrome (comorbid COPD and OSA) and obesity hypoventilation syndrome (OHS) [67].

Typical starting pressures for BPAP are 12 cm H2O IPAP and 8 cm H2O with adjustments based on patient response.



OSA, obstructive sleep apnea; PAP, positive airway pressure; CPAP, continuous positive airway pressure; O2, Oxygen; aCPAP, Auto CPAP; AVAPS, average volume assured pressure support.  
 \*Contraindications to PAP: GI bleed, Craniofacial trauma, high aspiration risk, altered mental status, vomiting.  
 †Auto-titrating CPAP can be used for empiric treatment.  
 ‡ Effective in neuromuscular disease related hypoventilation and obesity hypoventilation syndrome(OHS).

Figure 1. Algorithm for managing hospitalized patients with OSA.

Table 3. Treatment therapies for OSA in hospitalized patients.

Treatment Options in Hospital	Pros	Cons
Repositioning/using non supine position	Simple intervention. Reduces AHI event frequency.	Limited efficacy. Might not be possible in post op/post procedure patients
Oxygen Therapy	Well tolerated. Improves hypoxemia. Shown to OSA related blood pressure spikes	Does not stop apnea/hypopnea events
CPAP/AutoCPAP	Established treatment Effective for OSA, OHS, CSA except in patients with EF < 45%	Patient tolerance Contraindicated in some patients
Bilevel PAP	Can be used on patients with ventilation problems, OHS, CSA	Same as above
High flow NC	Effective in at least reducing AHI Well tolerated Safer in patients with contraindications for PAP therapy	Less effective than PAP

OSA, obstructive sleep apnea; CPAP, continuous positive airway pressure; OHS, obesity hypoventilation syndrome, CSA, central sleep apnea; EF, ejection fraction; PAP, positive airway pressure; AHI, apnea hypopnea index; NC, nasal cannula.

Compliance with PAP therapy is a significant issue in outpatient and inpatient settings. Early aversion to PAP therapy results in poor compliance. Most of these arise from a feeling



of continuous pressure and/or claustrophobia. Specifically, in the inpatient population, there are contraindications to receiving PAP therapy, such as GI bleeding, altered mental status, risk of aspiration, cranio-facial trauma, or surgery. Moreover, patients who need support above 15 mmHg tolerate bilevel therapy better due to the pressure release effect during expiration. There may be value in starting patients on PAP therapy in the hospital and helping with compliance/troubleshooting issues. In a pilot study of obese patients who underwent in-hospital screening followed by testing in-hospital and being started on PAP therapy (auto PAP or bilevel), Badami et al. showed that patients who were compliant with at least 4 h of PAP were 4 times more likely to continue to stay compliant with PAP post-discharge [68]. In addition to compliance, there are limitations in prescribing PAP therapy at discharge from the hospital, as it is not covered by payers for empiric treatment without a sleep study. This can result in long LOS and delays in discharges.

#### *4.4. Supplemental Oxygen and High-Flow Nasal Cannula*

Supplemental oxygen has been shown to improve hypoxemia in patients with OSA, although it can increase the duration of apnea-hypopnea events [69]. In a trial comparing CPAP to supplemental O<sub>2</sub> for sleep apnea treatment, the two groups had similar reductions in nocturnal hypoxemia; however, patients who received CPAP significantly improved blood pressure [70]. This difference was present even in patients whose blood pressure was well controlled on medications. Similarly, in a later trial comparing CPAP to supplemental O<sub>2</sub>, oxygen reduced intermittent hypoxia and improved blood pressure but did not affect arousal or sleepiness [71]. This data suggest that supplemental oxygen can be used for the short-term treatment of OSA in hospitalized patients.

High-flow nasal cannula (HFNC) using humidified room air (20–70 L) mixed with oxygen (FiO<sub>2</sub> 21–100%) has been successfully used as a treatment of hypoxemic and hypercapnic respiratory failure in place of a positive pressure device such as CPAP or BIPAP [72,73]. HFNC has been proposed as a treatment for OSA, and the use of oxygen at 20 L per minute has been shown to reduce both the AHI and the arousal index [74]. In a few small studies, HFNC has been successfully used to treat OSA in patients unsuitable for CPAP [75,76]. In a small study of stroke patients who could not use CPAP, HFNC was successfully used to reduce AHI [75]. Similarly, in a small study of surgical patients at high risk of OSA, HFNC between 20 and 40 L was better tolerated by the patient than CPAP. It also resulted in a reduced number of desaturation events [76]. In summary, based on current evidence, patients who cannot tolerate a PAP device due to claustrophobia, delirium, or the risk of aspiration during acute illness can be managed with HFNC while inpatient [59,76].

### **5. Management of the Perioperative Patient**

OSA is an established risk factor for a difficult airway during intubation [77]. OSA patients have a higher risk of respiratory complications, including respiratory failure, hypoxemic episodes, and reintubation. In one study, OSA patients were 61% more likely to develop postoperative pneumonia [38]. From an inpatient physician's perspective, the post-operative management of these patients is important as they are transferred from recovery rooms to hospital units under the care of house staff and hospitalists.

High-risk patients should be screened for OSA using screening tools, and consideration should be given to empirically treating positive airway pressure therapy in high-risk patients in the perioperative setting [37]. In a meta-analysis of 26 studies with 50,592 patients, a higher STOP-BANG score was associated with postoperative respiratory and neurologic complications and in-hospital mortality [78]. Although CPAP use has not been shown to reduce cardiovascular complications other than improving blood pressure directly, there was a reduced risk of respiratory and cardiac complications in the postoperative setting in patients using CPAP [38]. Patients with undiagnosed SDB have a higher risk of transfer to the ICU with opioid use [79]. In a study of patients undergoing joint replacement, the risk of postoperative complications was higher in patients with OSA [80]. A second study showed

that using CPAP reduced respiratory and cardiac complications' risk in the postoperative setting [38].

We recommend that inpatient providers consider the empiric use of auto-titrating CPAP in patients with hypoxia or apneic events at high risk for OSA in perioperative settings. Other options include HFNC, which has been shown to improve the AHI [74–76]. In a randomized control trial of post-operative obese patients, CPAP vs. HFNC at 20–40 L/min was equal in keeping desaturation events <4%, with HFNC being tolerated better than CPAP [81]. Another small study reported improvement in AHI when HFNC was coupled with a 30° upper body position [59].

## 6. Considerations in Specific Inpatient Populations

### 6.1. OSA and CHF

SDB, including central sleep apnea (CSA), is common in hospitalized patients with CHF exacerbation, and in one study, 76% had an AHI of more than 15 [82]. In another study of hospitalized CHF patients who underwent a Type III sleep study, 75% had SDB, of which 57% had OSA. A PSG at 8 weeks validated the predictive value of in-hospital testing [23]. Treating OSA has benefits and limitations in CHF patients. In a randomized control trial of patients—with acute CHF exacerbation who underwent in-hospital sleep studies and were randomized to receive either goal-directed medical therapy (GDMT) with PAP or just GDMT—left ventricular ejection fraction (LVEF) increased by 4.5% in the treatment group versus 0.3% in the nontreatment group at 3 days post randomization [57]. Early intervention with PAP therapy for 48 h and GDMT resulted in a significantly higher reduction in pulmonary artery pressures and improvement in acute CHF symptoms compared to GDMT alone in one study of CHF patients with OSA [60]. It also resulted in improvements in the right and left ventricular functions. In another study, patients admitted to the hospital for treatment of CHF exacerbation underwent a hospital Type III sleep study and were prescribed and discharged on auto-PAP [83]. Patients with PAP adherence had a lower readmission rate at 30 days as compared to those who were noncompliant [83]. In another study, early intervention with PAP therapy by screening patients within four weeks of discharge from the hospital also reduced the six-month readmission risk in patients who were compliant with treatment [84]. In a single-center study, patients admitted to the hospital were screened; high-risk patients underwent HRPO testing, and patients with high ODI were referred for PSG and PAP therapy [25]. Patients who were compliant with treatment in the first 3 months had improved survival over a mean follow-up of almost 2 years [25]. In this study, the HRPO ODI showed an AUC of 0.83 when compared to the AHI from PSG, suggesting that HPRO is a valid tool that can be easily implemented in the hospital. CPAP effectively treats acute cardiogenic pulmonary edema and lowers the risk of mechanical ventilation [85,86]. Possible benefits of CPAP included a reduction in sympathetic activity as recurrent arousals are stopped and hypoxemia improved.

Although treatment of OSA with PAP improves outcomes, the treatment of CSA has been linked to an increased risk of death in patients with an ejection fraction (EF) of 45% or less [87]. In this study, patients with an EF of 45% or less and an AHI  $\geq$  15, with most of the patients having CSA, were treated with adaptive servo ventilation (ASV). The treatment group had a higher incidence of all-cause and cardiovascular mortality. While the mechanism underlying this increased mortality is unknown, there are indications that Cheyne–Stokes breathing may be protective in CHF patients, with attenuation resulting in a poor outcome [87]. A recent computer model studying the effects of ASV on CHF patients with OSA shows a complex mechanism of adverse outcomes in patients with extremely low EF, such as increased sympathetic tone, dampened vagal tone, and reduced coronary flow, possibly leading to arrhythmias. As the increased mortality in the original study was unrelated to recurrent admissions or remodeling, sudden cardiac death remains the most plausible cause [88]. Overall, current practice guidelines do not recommend using ASV to treat SDB in patients with EF < 45%. PAP therapy can still be used to treat OSA. CPAP at a fixed pressure is safe and should still be considered for treating CSA in this population [89].

### 6.2. OSA and Pulmonary Disorders and Infections

The presence of OSA and COPD together is termed overlap syndrome [90]. These patients have more profound nocturnal hypoxemia than patients with either disease alone [91]. The management of overlap syndrome is focused on the concurrent treatment of both underlying diseases [92]. CPAP is still an accepted treatment for overlap syndrome and has been shown to improve PaO<sub>2</sub>, PCO<sub>2</sub>, pulmonary artery pressures, maximum inspiratory force, and exercise capacity in small studies [93–95]. Since CPAP does not significantly affect ventilation, the mechanism of action is thought to be related to the off-loading of respiratory muscles as they work against upper airway resistance [92]. For patients who do not improve with CPAP, bilevel ventilation (BPAP) effectively improves hypercarbia and provides ventilation support [90]. If hypoxemia does not resolve with PAP therapy alone, oxygen supplementation may be added to PAP therapy [90].

Patients with OSA have a higher risk of community-acquired pneumonia [96]. Possible mechanisms include swallowing dysfunction, recurrent aspirations, and an increased risk of GERD [97]. In a retrospective cohort study of 250,907 patients, the incidence of OSA was 6.2% (15,569) [98]. Patients with pneumonia and comorbid OSA tend to be men with a higher BMI compared to the control population, with a higher incidence of CHF and COPD. In addition, patients with OSA and pneumonia were at higher risk of being transferred to the ICU, requiring invasive mechanical ventilation, tracheostomies, and extended hospital stays [98,99]. Interestingly, mortality in two large retrospective cohorts of patients with OSA and pneumonia was lower than in patients without OSA [98,99]. This may be related to the chronic intermittent hypoxia associated with OSA and the morbidity–mortality paradox of obesity [27,100]. Providers caring for pneumonia patients should continue screening and watching for SDB.

Patients with OSA have been found to have an increased risk of hospitalization with respiratory viral illnesses, such as coronavirus disease 2019 (COVID-19) [101] and influenza [102]. Patients with OSA are at a higher risk of developing an influenza infection based on a population-based cohort study [103]. Patients with comorbid OSA and COVID-19 were found to be at a higher risk for risk-adjusted mortality [104,105]. In another study, OSA was not only associated with a higher risk of hospitalization but also with a progression to the development of respiratory failure, although the prevalence of OSA was much lower in the control group compared to the reported OSA prevalence in the literature [106]. Hospitalized COVID-19 patients with OSA have been shown to have increased LOS in the hospital as compared to patients without OSA [105].

### 6.3. Obesity Hypoventilation Syndrome

Obesity is a significant risk factor for OSA and is also associated with obesity hypoventilation syndrome (OHS). OHS is defined by the combination of obesity (BMI  $\geq 30$  kg/m<sup>2</sup>), SDB, and awake daytime hypercapnia (awake resting PaCO<sub>2</sub>  $\geq 45$  mm Hg at sea level), after excluding other causes for hypoventilation [107]. CPAP can be offered as a first-line treatment in ambulatorily patients with OHS and coexistent OSA. However, patients hospitalized with respiratory failure and suspected of having OHS may need to be discharged with BPAP until they undergo PSG titration and start PAP through an accredited sleep center [107]. These patients will benefit from early referral to a sleep center on discharge.

### 6.4. OSA and Stroke

Patients with CVA/TIA are at high risk for OSA. In a meta-analysis, 72% of patients had SDB (primarily OSA, 7% CSA) [42]. There was a higher prevalence in men, patients with recurrent strokes, and strokes of unclear etiology. Younger stroke patients had a higher incidence of SDB compared to the general population of similar ages [42]. Since these patients have a high incidence of OSA, early screening with a Type III device has been shown to be as reliable as a PSG [108,109]. In one study, patients admitted with acute CVA underwent portable sleep studies in the hospital and were successfully treated with nasal auto-CPAP, which was well tolerated [110]. Due to the high prevalence of SDB in



CVA patients, referral at discharge should be done for a sleep study [42]. For patients with significant inpatient hypoxemia and desaturations, in-hospital testing and starting on auto-CPAP may be an option with an early referral to a sleep center.

### 6.5. OSA and Neuromuscular Disease

SDB is also common in neuromuscular diseases [111], with various mechanisms at play. Respiratory muscle weakness can lead to mixed events, reduced lung volumes resulting in hypoventilation, oropharyngeal weakness, or tongue hypertrophy resulting in obstructive events [112,113]. CSA can result from associated cardiomyopathy in late stages [112]. Most neuromuscular disease-related SDBs are treatable with a BPAP or average volume assured pressure support (AVAPS) [113]. Patients admitted to the hospital with comorbid OSA and NMD should continue using home respiratory support devices if prescribed. Patients may require additional titration of PAP at discharge and should be referred for follow-up with a sleep physician.

## 7. Outcomes of OSA Patients

A large retrospective case-control study (85,912 matched pairs) by the Veterans Administration compared patient outcomes between those with OSA and controls [14]. Patients with OSA showed a high incidence of readmission to the hospital and an increased risk of admission to the ICU and emergency department, and outpatient visits when adjusted for other comorbidities. However, the length of stay in the hospitalization index was not different between the two groups [14].

Paradoxically, studies have shown an inpatient survival benefit for patients with OSA [27,114]. This has been hypothesized to be related to the protective effects of intermittent hypoxia [100].

### 7.1. Discharge Planning

Current guidelines set by Medicare note that a CPAP device cannot be prescribed without a sleep study. Patients admitted to the hospital who are found to be at high risk for OSA should be referred to a board-certified sleep physician for further evaluation and a PSG. For high-risk and symptomatic patients, a Type III study can be done in the hospital, and patients could be discharged on an auto-CPAP and follow up with a board-certified sleep specialist. Patients with OHS and OSA, though not eligible for CPAP without a sleep study, could be prescribed a BPAP device for treatment of hypercarbia if it is deemed clinically that the hypercarbia is not primarily due to OSA. Patients with OSA-COPD overlap syndrome could also be discharged on a BPAP device for the treatment of hypercarbia. COPD and CHF readmission are quality metrics established by Medicare. Hospitalists across the country are struggling to improve this number. Interventions such as home health, early PCP appointments, CHF clinics, and DME setup can help decrease readmissions. Referral for OSA is a slow process, with most patients requiring a primary care physician referral. Setting up an inpatient service with early referrals to a board-certified sleep physician could be an additional resource to reduce the readmission burden and improve outcomes.

### 7.2. Inpatient Sleep Medicine

Recently, Sharma et al. shared their experience setting up an inpatient sleep service line in the hospital with a comprehensive sleep evaluation. They have proposed a protocol that includes a health record-based screening questionnaire followed by testing with HRPO or a portable sleep study and treatment with auto-CPAP [28]. The protocol also recommends communication with multidisciplinary team members to help with discharge planning. An inpatient service with established screening can also result in more patients being identified with OSA [24]. Inpatient sleep consults are not readily available in most healthcare systems, as most hospitals do not have an inpatient consultative sleep medicine service line. Arrangements could be made for a virtual consult and remote sleep study

interpretation in hospitals with no sleep physicians available. This is a process commonly used by hospitalists for consultants who are not readily available in community settings.

## 8. Conclusions

OSA is a common comorbid condition in hospitalized patients and is frequently undiagnosed and untreated. The management of comorbid OSA can improve outcomes in acute care and perioperative populations. Patients can be treated with oxygen supplementation, HFNC, CPAP, BPAP, or AVAPS based on their underlying condition. Diagnosis and management of patients can help decrease complications, LOS, and healthcare utilization. Patients will benefit from being screened at hospital admission and managed using established protocols, and high-risk patients will be referred to a sleep center at discharge.

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