Perioperative Management of the Patient With Obstructive Sleep Apnea: A Narrative Review

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The prevalence of obstructive sleep apnea (OSA) has reached 1 billion people worldwide, implying significant risk for the perioperative setting as patients are vulnerable to cardiopulmonary complications, critical care requirement, and unexpected death. This review summarizes main aspects and considerations for the perioperative management of OSA, a condition of public health concern. Critical determinants of perioperative risk include OSA-related changes in upper airway anatomy with augmented collapsibility, diminished capability of upper airway dilator muscles to respond to airway obstruction, disparities in hypoxemia and hypercarbia arousal thresholds, and instability of ventilatory control. Preoperative OSA screening to identify patients at increased risk has therefore been implemented in many institutions. Experts recommend that in the absence of severe symptoms or additional compounding health risks, patients may nevertheless proceed to surgery, while heightened awareness and the adjustment of postoperative care is required. Perioperative caregivers should anticipate difficult airway management in OSA and be prepared for airway complications. Anesthetic and sedative drug agents worsen upper airway collapsibility and depress central respiratory activity, while the risk for postoperative respiratory compromise is further increased with the utilization of neuromuscular blockade. Consistently, opioid analgesia has proven to be complex in OSA, as patients are particularly prone to opioid-induced respiratory depression. Moreover, basic features of OSA, including intermittent hypoxemia and repetitive sleep fragmentation, gradually precipitate a higher sensitivity to opioid analgesic potency along with an increased perception of pain. Hence, regional anesthesia by blockade of neural pathways directly at the site of surgical trauma as well as multimodal analgesia by facilitating additive and synergistic analgesic effects are both strongly supported in the literature as interventions that may reduce perioperative complication risk. Health care institutions are increasingly allocating resources, including those of postoperative enhanced monitoring, in an effort to increase patient safety. The implementation of evidence-based perioperative management strategies is however burdened by the rising prevalence of OSA, the large heterogeneity in disease severity, and the lack of evidence on the efficacy of costly perioperative measures. Screening and monitoring algorithms, as well as reliable risk predictors, are urgently needed to identify OSA patients that are truly in need of extended postoperative surveillance and care. The perioperative community is therefore challenged to develop feasible pathways and measures that can confer increased patient safety and prevent complications in patients with OSA. (Anesth Analg 2021;132:1231–43)

GLOSSARY

AHI = apnea-hypopnea index; **ASA** = American Society of Anesthesiologists; **CI** = confidence interval; **CPAP** = continuous positive airway pressure; **FDA** = Food and Drug Administration; **ICU** = intensive care unit; **LOS** = length of stay; **NMB** = neuromuscular blocking; **OR** = odds ratio; **OSA** = obstructive sleep apnea; **P-SAP** = Perioperative Sleep Apnea Prediction Score; **PACU** = postanesthesia recovery unit; **PAP** = positive airway pressure; **SASM** = Society of Anesthesia and Sleep Medicine; **Spo_2** = blood oxygen saturation; **STOP-Bang** = Snoring, Tiredness, Observed apnea, blood Pressure, Body mass index, Age, Neck circumference, and Gender

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Accepted for publication January 8, 2021.

Funding: None.

Conflicts of Interest: See Disclosures at the end of the article.

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DOI: 10.1213/ANE.0000000000005444

With a prevalence of 1 billion people worldwide, obstructive sleep apnea (OSA) has become a public health concern.[1] Over the last decade, evidence has consistently confirmed the presence of OSA as a modifier of perioperative outcome, driving postoperative morbidity, and critical complications that may occasionally result in death.[2–4] In this context, OSA patients are at particular risk for cardiopulmonary complications and prone to the postoperative requirement of critical care.[5,6]

The high prevalence of OSA especially in the surgical population has incentivized clinical practices to develop

protocols of risk mitigation. The desire to implement perioperative safety management strategies is particularly strengthened by evidence demonstrating that critical, life-threatening, and fatal postoperative events are often deemed as preventable in retrospect.[7–10]

Despite a plethora of clinical evidence delineating the perioperative risks associated with OSA, scientific data on the efficacy of perioperative safety measures are largely lacking. Moreover, OSA is increasingly acknowledged as a complex condition with significant heterogeneity in terms of disease severity, comorbidity burden, and complication risk.

Perioperative societies and expert groups, including the American Society of Anesthesiologists (ASA),[11] the Society of Anesthesia and Sleep Medicine (SASM),[12] and the American Academy of Sleep Medicine,[13] have provided recommendations for the perioperative care of patients with OSA, attempting to base their guidance on the implementation of scientifically supported management measures.[11,12] A summary of recommendations is provided in Tables 1, 2.[11,12] This narrative review seeks to summarize the main aspects and considerations that are significant for the perioperative management of patients with OSA.

CRITICAL FEATURES OF OSA

The pathophysiology of OSA is marked by an interaction between unfavorable changes in upper airway anatomy and collapsibility and the instability of ventilatory control.[14] While OSA disease severity poses a major driver of perioperative complication risk, it varies significantly within its patient population. Thus, patients with a narrower and more collapsible airway often due to obesity or craniofacial abnormalities may exhibit more severe forms of OSA. Pathophysiological changes relate to the ability of upper airway dilator muscles to respond to pharyngeal collapse and apnea during sleep. As such, a decreased tone of upper airway dilator muscles constitutes a critical element of OSA pathogenesis. Once obstruction has occurred and ventilation is reduced, subsequent blood gas changes stimulate respiratory effort and upper airway dilator muscle activity concurrently.[15] This response typically terminates events of apnea and is initiated when the respiratory arousal threshold is reached, respectively, when a certain neuromechanical drive or pressure triggering arousal from sleep is met.[16] Patients with OSA can be stratified into those exhibiting a high or a low arousal threshold in response to apnea-related hypoxemia and hypercapnia. A low arousal threshold implies that patients wake up more frequently to minimal stimuli before reaching critical, hypoxemic oxygen saturation levels (nadir blood oxygen saturation [Spo_2] >82%). Thus, low arousal thresholds are particularly marked by disruptive sleep, a higher prevalence in mild to moderate OSA, and are mostly associated with a higher frequency of hypopneas rather than apneas.[14] On the other hand, patients exhibiting high arousal thresholds have a lower propensity to arouse from apneic events, with possible breaches of critical oxygen desaturation levels, therefore bearing the risk for severe arousal failure and precipitous hypoxemia to the extent of an arousal arrest.[17] High arousal thresholds are usually observed in moderate to severe OSA.

Table 1. Recommendations for the Intraoperative Management of OSA From the Society of Anesthesia and Sleep Medicine and the American Society of Anesthesiologists Task Force on Perioperative Management of Patients With OSA[a]

Airway management	Known or suspected OSA to be considered an independent risk factor for difficult intubation and mask ventilation. Management according to "The practice guidelines for management of the difficult airway" is recommended.
Hypnotic and sedative medications	Patients at increased perioperative risk from OSA to be considered especially susceptible to the worsening of upper airway collapsibility and depression of central respiratory activity caused by hypnotic medications, including propofol, inhalational agents, and benzodiazepines.
	Risk for postoperative respiratory compromise to be considered in the determination of medication regimens and drug dosing.
	Risk for adverse respiratory events to be considered during procedural sedation with propofol or benzodiazepines.
Opioids	Increased risk for opioid-induced respiratory depression to be considered in OSA.
	Altered pain perception and increased opioid potency to be anticipated in OSA.
Neuromuscular blockade	Increased risk for the impact of postoperative residual neuromuscular blockade, hypoxemia, or respiratory failure to be considered in OSA.
Anesthesia technique	Regional anesthesia is preferable over general anesthesia, whenever applicable.
	Superficial surgical procedures: use of local anesthesia or peripheral nerve blocks with/without moderate sedation to be considered.
	Moderate sedation to be continuously monitored by capnography.
	During sedation, CPAP or oral appliances to be considered in patients with previous use.
	General anesthesia with a secured airway is preferable over deep sedation without a secure airway in OSA.
	Neuraxial anesthesia to be considered for peripheral procedures.
Emergence from anesthesia	Patients at increased perioperative risk from OSA to be extubated while awake, in the absence of contraindications.
	Full reversal of neuromuscular blockade to be verified before extubation.
	Extubation and recovery recommended in the lateral, semiupright, or other nonsupine positions, whenever feasible.

Abbreviations: ASA, American Society of Anesthesia; CPAP, continuous positive airway pressure; OSA, obstructive sleep apnea.
[a]Summary of recommendations on the intraoperative management of OSA. Content was derived from Gross et al[11] and Memtsoudis et al.[12]

Table 2. Recommendations for the Postoperative Management of OSA From the American Society of Anesthesiologists Task Force on Perioperative Management of Patients With OSA and the Society of Anesthesia and Sleep Medicine[a]

Postoperative analgesia	Regional analgesic techniques should be considered for the reduction of systemic opioid requirements.
	In the context of neuraxial analgesia, benefits and risks of opioid use versus sole local anesthetic utilization should be considered.
	Continuous background infusions to be avoided or used with extreme caution in the presence of patient-controlled systemic opioids utilization.
	Increased risk for respiratory depression and airway obstruction to be considered during concurrent administration of sedative agents.
	Multimodal analgesia to be considered for the reduction of systemic opioid requirements.
Supplemental oxygen	After surgery, supplemental oxygen to be administered continuously, until baseline oxygen saturation can be maintained with room air.
Pulse oximetry	Continuous pulse oximetry to be administered as long as patients remain at increased risk.
Patient positioning	Nonsupine positions are recommended during anesthesia recovery, whenever feasible.
CPAP use	CPAP or noninvasive positive pressure ventilation to be continuously administered throughout the hospitalization in patients with preoperative treatment.
	Initiation of nasal CPAP or noninvasive positive pressure ventilation to be considered in the event of frequent or severe airway obstruction or hypoxemia during postoperative monitoring.

Abbreviations: ASA, American Society of Anesthesia; CPAP, continuous positive airway pressure; OSA, obstructive sleep apnea.
[a]Summary of recommendations on the postoperative management of OSA. Content was derived from the Gross et al[11] and Memtsoudis et al.[12]

These patients are at particular risk when utilizing sedatives and narcotics which may additionally delay an already failing arousal to the point of precipitous falls in Spo_2 levels and respiratory arrest.[17] Furthermore, the stability of ventilatory control presents another distinguished driver of OSA, which is defined by the propensity to manifest a cyclical breathing pattern during sleep whereby patients oscillate between obstructive breathing events and arousal.[14] The pathophysiology of repeated sleep arousal sleep cycles with repetitive nocturnal hypoxemic events ultimately precipitates unfavorable consequences, including cardiac arrhythmias, myocardial ischemia, pulmonary and systemic hypertension with biventricular hypertrophy, and sleep fragmentation with a decline in intellectual function and behavioral changes.[18]

PREOPERATIVE RISK ASSESSMENT

The obligatory groundwork for the enforcement of safety measures is laid by the preoperative identification of patients at increased risk from OSA. While polysomnography represents the gold standard of OSA diagnosis, the time and resource requirements have rendered this measure unsuitable for routine preoperative use. Instead, essential clinical predictors of OSA can be evaluated through patient interview, review of medical records, physical examination, and the use of screening tools. In particular, OSA screening questionnaires, including the STOP-Bang (Snoring, Tiredness, Observed apnea, blood Pressure, Body mass index, Age, Neck circumference and Gender) Questionnaire, the Perioperative Sleep Apnea Prediction Score (P-SAP), the Berlin Questionnaire, and the ASA checklist, have been validated and established as feasible for preanesthetic risk stratification. These screening tools have therefore increasingly become the standard of preanesthetic OSA evaluation.[19]

Patients with a diagnosis of OSA as well as screen positive patients with high threshold values should be assumed to have moderate to severe OSA.[19] To that effect, all stakeholders, including the patient, the family, and caregivers, should be advised about the heightened perioperative risk while precautionary measures should be put into place. Although firm scientific data are still lacking, most experts agree that in the absence of additional risk from any uncontrolled systemic comorbidity such as cardiopulmonary disease, the delay of surgery for further investigation and diagnosis is not recommended if the presence of basic clinical safety measures is assured. This also applies to preoperatively diagnosed, untreated patients and individuals noncompliant with OSA treatment when risk mitigation strategies are in place. In contrast, preoperative optimization of the patient's health condition should be considered in severe OSA or if the associated risk is deemed high. This may include the initiation of positive airway pressure (PAP) therapy, the implementation of mandibular and oral appliances, and preoperative weight loss. However, the impact of these interventions, although pathophysiologically sound, remains ill defined.[12] Furthermore, uncontrolled systemic or cardiopulmonary comorbidities compounding OSA (eg, hypoventilation, pulmonary hypertension, unclear resting hypoxemia) may require further preoperative evaluation and treatment. Additional key decision factors include surgical urgency and invasiveness and the postoperative requirement for narcotics and sedatives. Importantly, PAP utilization is strongly recommended throughout the entire hospital stay pre- and postoperatively in patients previously adherent.[11] A summary of recommendations on the preoperative decision process is provided in the Figure.[19]

Furthermore, while the implementation of ambulatory surgery is on the rise, the suitability of OSA

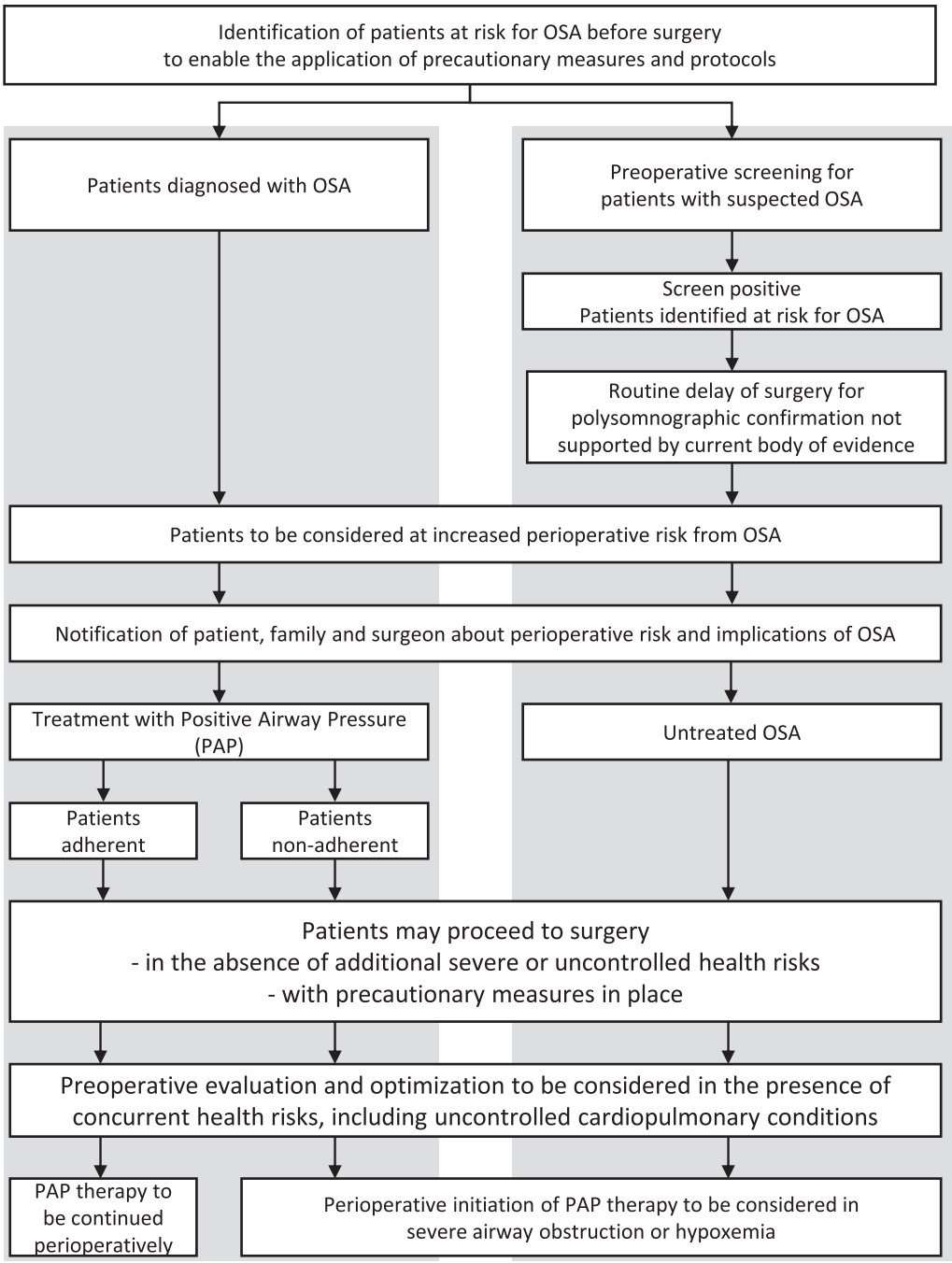

Figure. Recommendations for the preoperative management of OSA from the Society of Anesthesia and Sleep Medicine and the American Society of Anesthesiologists Task Force on Perioperative Management of Patients with OSA. Content was derived from Gross et al[11] and Chung et al.[19] OSA indicates obstructive sleep apnea.

patients for such setting remains controversial based on concerns for serious postdischarge complications and unexpected death. In this context, the Society of Ambulatory Anesthesia has issued evidence-based recommendations, suggesting the consideration of ambulatory surgery only if postoperative pain can be predominantly managed with nonopioid analgesic techniques or in settings where postoperative PAP therapy is feasible. On the contrary, caution is warranted in OSA patients with coexistent nonoptimized comorbid medical conditions, who should not be considered as candidates appropriate for ambulatory surgery.[20]

INTRAOPERATIVE MANAGEMENT

Airway Management

Typical features of OSA, including obesity and variations in upper airway and craniofacial anatomy, predispose patients to a narrow and crowded airway

with a higher risk for difficult airway management, perioperative laryngeal injury and airway trauma, rapid oxygen desaturation, aspiration, unexpected intensive care unit (ICU) admission, and death. Common abnormalities of the upper airway anatomy include a large tongue, overcrowding of oropharyngeal structures, increased fat deposition in the pharynx, a reduced upper airway diameter as well as a larger neck circumference.[21] It is therefore not surprising that a significant association between OSA and difficult intubation has been widely established.[18] A recent analysis demonstrated that during intubation of OSA patients, the occurrence of poor visualization of the glottis (Cormack and Lehane III/IV), the requirement of intubation aids, and the need for multiple intubation attempts were increased by more than a 3-fold (odds ratio [OR] = 3.46; confidence interval [CI], 2.32-5.16) compared to the general population. Similar findings were reported for mask ventilation, with challenges involving a higher incidence of inadequate oxygenation, requirement of additional airway adjuvants or 2 providers as well as impossible mask ventilation (OR = 3.39; CI, 2.74-4.18).[21] OSA should therefore be considered a risk factor for airway complications and it is recommended to manage these patients according to the practice guidelines for the management of the difficult airway.[12,22]

Anesthetic Medications

Airway patency is challenged by any form of unconsciousness, including sleep, sedation, and anesthesia, but while arousal responses are preserved during sleep, they are depressed during sedation and abolished by anesthesia.[23] The impact of anesthetic drugs and agents therefore presents a significant perioperative challenge as hypnotics and narcotics depress upper airway function at various degrees. Critical, dose-dependent effects of anesthetic and narcotic medications include worsening of upper airway collapsibility, depression of central respiratory activity with reduced output to upper airway dilator muscles, delayed respiratory arousal response to airway occlusion, and diminished ventilatory response to hypercarbia and hypoxia.[24,25] While these medications are designated to suppression of alertness and pain perception, residual effects constitute a disadvantage after emergence from anesthesia when spontaneous breathing returns and patients move beyond the operating room environment with reduced or terminated surveillance. To prevent the occurrence of respiratory arrest, the ASA and SASM have therefore emphasized the need for consideration of sustained drug effects and their impact on postoperative OSA severity when determining intraoperative medication regimens, including drug dosing, action time, and metabolism.[11,12]

Hypnotic and Sedative Medications

General anesthetic agents confer dose-dependent attenuations of upper airway muscle activity. In this context, the vulnerability to upper airway collapse has been suggested to increase with diminished pharyngeal dilator muscle activity.[24,25] This dose-related effect results from the depression of central respiratory output to upper airway dilator muscles and suppression of upper airway reflexes.[25] The primary site of occlusion in the majority of OSA patients appears to be at the level of the soft palate during sleep.[26] Moreover, during procedural sedation, OSA severity and higher levels of obesity have been associated with airway collapse at multiple sites as well as total airway obstruction and hypoxemic incidents.[27–29]

The upper airway is systematically more collapsible during anesthesia than during sleep, implying greater vulnerability in the anesthetized state.[30] Patients with OSA should therefore be considered at increased risk for adverse respiratory effects based on the impact of hypnotic agents, including propofol, benzodiazepines, and inhalational drugs.[31,32] These medications should be utilized carefully in OSA patients with particular caution during procedural sedation and postoperative recovery given the absence of a secured airway.[12] Furthermore, intraoperative monitoring of anesthesia depth by bispectral indexing or A-line autoregression may facilitate the titration of hypnotic agents and permit a reduction of cumulative anesthetic dosages.[33,34]

Neuromuscular Blocking Agents

Neuromuscular blocking (NMB) medications are frequently needed for endotracheal intubation and surgical relaxation during mechanical ventilation. Complete reversal of this effect, however, is required before emergence from anesthesia to ensure efficient spontaneous respiratory function, maintenance of a patent airway, and recovery of protective airway reflexes. Despite the understanding that even partial residual NMB without respiratory symptoms can impair upper airway dilator muscle function and cause respiratory complications, residual NMB appears to be common with a reported incidence of up to 64% among patients in the postanesthesia recovery unit (PACU).[35,36]

Current evidence indicates that OSA patients may be at significantly higher risk for postoperative events related to residual neuromuscular blockade with a higher propensity for pulmonary complications, hypoxemia, and respiratory failure.[37] Full reversal of neuromuscular blockade should therefore be verified even after administration of reversal agents to rule out residual effects that promote pharyngeal dysfunction, airway obstruction, and aspiration.[11,12,38] Although in the United States, sugammadex is still used with a

similar frequency as neostigmine, a recent multicenter cohort analysis (the STRONGER trial) found that sugammadex was associated with a 30% reduced risk for pulmonary complications, a 47% reduced risk for pneumonia, and a 55% reduced risk for respiratory failure when compared to neostigmine. Given the detriment of residual neuromuscular blockade and the unfavorable effects of systemic cholinergic activity, these findings provide strong support for a wider implementation of sugammadex versus neostigmine for reversal of neuromuscular blockade.[39]

Opioids

Evidence indicates that OSA is associated with a 50% increased risk for opioid-induced respiratory depression.[40] This is likely based on the interference of narcotics with chemical and motor control of ventilation. Opioids depress the central ventilatory drive by diminishing the respiratory response to hypoxia and hypercarbia.[41] Furthermore, they are implicated in reducing the pharyngeal motor tone and thereby precipitate impaired airway patency, obstructive apnea events, and alveolar hypoventilation. As a result, the risk for delayed or failed ventilatory arousal in response to hypoxia and hypercarbia is particularly increased in OSA. Clinical evidence further indicates an association between cumulative opioid consumption dose and postoperative severity of the apnea-hypopnea index (AHI) and central apneas.[42,43] Moreover, intra- and postoperative parenteral opioid use have been linked to significantly higher odds for respiratory failure in patients with OSA.[10,44–46] Studies investigating the occurrence of delayed postoperative respiratory depression have confirmed these findings and demonstrated a significant association between postoperative naloxone requirement and OSA status.[47,48] Furthermore, in the presence of patient-controlled morphine analgesia, OSA status was identified as a predictor of severe postoperative respiratory depression with the requirement of naloxone administration.[47,49]

OSA patients should therefore be considered at increased risk for adverse respiratory events from the use of opioid medication.[12]

Pain and Opioid analgesia

Basic components of OSA have been described to progressively alter pain sensitivity and opioid potency.[50] Chronic recurrent nocturnal hypoxia and sleep disruption appear to enhance the sensitivity to pain, while perpetual hypoxia may additionally precipitate potentiated opioid analgesic effects.[51,52] In this context, a significant reduction in postoperative opioid consumption has been observed in OSA patients exhibiting intermittent nocturnal hypoxia.[51] The underlying mechanism could be a hypoxia-triggered upregulation of opioid receptors, ultimately causing increased sensitivity to narcotics.[53] In pediatric patients with OSA, Brown et al[54] discovered an association between the severity of preoperative hypoxia and postoperative respiratory morbidity. Thus, the Spo_2 level may potentially constitute an accurate measure for preoperative risk stratification, rather than the AHI with poor association to hypoxemia.[50] The ASA has therefore encouraged the adjustment of perioperative opioid dosing in pediatric OSA patients that exhibit hypoxemia.[11]

On the other hand, chronic fragmentation of sleep due to perpetual arousal has been associated with enhanced pain perception. This was observed in patients exhibiting hyperalgesia as a result of insomnia, while improved sleep continuity with PAP treatment was found to reduce pain sensitivity in severe OSA.[55]

Alterations in pain perception and opioid requirement should therefore be anticipated in the perioperative management of OSA, as opioid requirements could be substantially lower in this patient population. In this context, preoperative nocturnal hypoxia may be critical for postoperative opioid pharmacology. Careful titration of opioid analgesia according to individual risk and clinical appearance could help prevent respiratory complications.[12]

Anesthesia Technique

Consistent with data from the general population, evidence indicates improved patient safety with the utilization of regional over general anesthesia in OSA.[56–58] In orthopedic surgery, the use of neuraxial compared to general anesthesia was associated with a decrease in pulmonary complications, mechanical ventilation, ICU admissions, length of stay (LOS), and hospitalization cost. Moreover, the addition of peripheral nerve blockade showed a further reduction in the requirement for mechanical ventilation, ICU admissions, and LOS, respectively.[56] General anesthesia has been identified as a risk factor for hypoxemia in OSA, which in turn predicted major respiratory complications, ICU admissions, and increased LOS.[59] Chung et al[60] discovered that OSA patients postoperatively experience a worsening of sleep-disordered breathing and changes in sleep architecture, expressed in an increased AHI and the exacerbation of nocturnal hypoxemia and hypercapnia. While this deterioration appears to peak on the third postoperative day, changes may persist for 7 days.

Interestingly, Chung et al[43] also identified general anesthesia as an independent driver of an increased postoperative central sleep apnea index, while postoperative AHI severity was associated with 72-hour cumulative opioid consumption in polysomnography confirmed OSA patients.

In contrast, regional techniques may facilitate the reduction of systemic anesthetic consumption and permit the avoidance of airway manipulation. This is significant in a patient population prone to difficult airway management.[21,61] Furthermore, the avoidance of neuromuscular blockade diminishes the heightened risk for postoperative residual NMB and pulmonary complications.[37]

Regional anesthesia also offers efficient pain relief with reduced opioid consumption, a major advantage given the underlying changes in pain behavior and opioid sensitivity that progressively result from chronic intermittent hypoxia and sleep fragmentation.[62,63]

Perioperative societies therefore strongly recommend that whenever feasible, regional anesthesia techniques should be preferred over general anesthesia in patients at perioperative risk from OSA.[11,12] For superficial procedures, the utilization of local anesthesia and peripheral nerve blockade with or without moderate sedation is recommended, while neuraxial anesthesia should be considered for peripheral procedures. General anesthesia with a secure airway is however generally preferred over deep sedation without a secure airway.[11] Whenever general anesthesia is performed in OSA patients, the risk for postoperative compromise should be considered according to the anesthetic regimen, including the dosing, action time, and metabolism of drugs. Moreover, multimodal analgesia pathways present additional important strategies in the context of increasing patient safety.[11]

In OSA, the combination of various analgesic modalities, including peripheral nerve blockade, acetaminophen, nonsteroidal anti-inflammatory drugs, cyclooxygenase-2 inhibitors, steroids, and ketamine, was associated with opioid sparing and a decrease in critical complications. This impact was observed in the context of a dose-response gradient, as the utilization of increasing analgesic modalities was associated with stepwise beneficial outcome effects related to opioid sparing, reduced postoperative mechanical ventilation, and decreased critical care admissions.[64]

Caution, however, is warranted with regards to the perioperative use of gabapentinoids as adjuncts in multimodal analgesia regimens because of the risk for serious respiratory depression in conjunction with sedating medications.[65] To this effect, the Food and Drug Administration (FDA) has recently issued a warning on gabapentinoid use and respiratory complications, recommending an increased level of respiratory vigilance in recipients of such drugs.[66]

Emergence From General Anesthesia

After extubation, the risk for life-threatening airway obstruction, accelerated arterial oxygen desaturation, and gastroesophageal reflux is increased in patients with OSA.[67] In OSA corrective surgery, postextubation airway obstruction has been reported with an incidence of 5%.[68] Moreover, airway collapse resulting from premature extubation can lead to a rapid development of severe negative pressure pulmonary edema from spontaneous ventilation against an obstructed airway.[18,68] Therefore, to avoid pulmonary complications, patients with OSA should be extubated while awake and able to respond to commands when there is no contraindication. Importantly, full NMB reversal is critical and should be verified before extubation, even after the utilization of antagonizing drugs.[11,12]

Furthermore, the collapsibility of the upper airway is primarily influenced by body positioning, rather than sleep stages.[69,70] In particular, the soft palate of the upper airway is highly susceptible to collapse in the supine position after the administration of anesthetics, sedatives, and opioids.[71] Rostral fluid shifts may further worsen airway obstruction due to edema.[72] Lateral positioning of the patient can structurally improve the maintenance of the passive pharyngeal airway in OSA.[73] Moreover, upper body elevation or 30° reverse Trendelenburg position improves upper airway stability during sleep and may allow therapeutic PAP levels to be substantially reduced postoperatively by minimizing the abdominal compression against the diaphragm.[69,73] Therefore, whenever possible, extubation and anesthesia recovery should be performed in lateral, semiupright, or other nonsupine positions.[11,12]

POSTOPERATIVE MANAGEMENT

Supplemental Oxygen

Postoperative supplemental oxygen has been shown to improve oxygenation and decrease the AHI without increasing the duration of apnea-hypopnea events.[74] To ensure adequate oxygenation, the ASA recommends that all patients at increased perioperative risk from OSA should receive supplemental oxygen continuously during recovery from anesthesia until the ability to maintain baseline oxygen saturation is reached while breathing room air.[11]

Nevertheless, caution is warranted because a subset of OSA patients may retain carbon dioxide based on the role of hypoxemia in triggering respiratory arousal. Thus, when supplemental oxygen abolishes hypoxemia, the apnea duration may increase in some patients and result in hypoventilation and hypercarbia with the risk for life-threatening respiratory depression. Continuous postoperative supplemental oxygen therapy should therefore be suspended as soon as baseline oxygen saturation can be maintained with room air aiming to prevent hypoventilation, prolonged apneic episodes, and atelectasis, which could remain undetected by pulse oximetry during oxygen therapy in the absence of additional monitoring (eg, respiratory rate monitoring or capnography).[11]

Positive Airway Pressure

PAP therapy serves as a pneumatic splint aimed to prevent airway collapse during sleep. Long-term PAP therapy can restore quality of life in OSA by improving ventilation and enhancing vigilance and cognitive function through the mitigation of sleep fragmentation. Nevertheless, due to common discomfort, the compliance to PAP is generally low, rendering the role of perioperative PAP utilization less clear. In cardiac and thoracoabdominal surgery, postoperative PAP therapy has conferred diminished hypoxemia and reduced pulmonary morbidity including the need for reintubation.[75–78] In OSA specifically, limited but growing evidence indicates that PAP use may attenuate postoperative cardiovascular complications, including cardiac arrest and shock,[79] while patients without PAP use may experience a higher incidence of postoperative complications.[80] Liao et al[81] reported that the perioperative randomization of OSA patients to auto-titrated PAP initiation resulted in significant AHI reduction and improved oxygen saturation. However, others have failed to demonstrate a reduction of complications.[82,83]

The lack of robust evidence specifically on the perioperative efficacy of PAP therapy presents a challenge difficult to overcome, given the impracticability of randomization of such standard of care treatment. Furthermore, there is large heterogeneity in current literature with regards to study conduct and OSA severity. In terms of perioperative safety, the optimal duration of preoperative PAP treatment or the ideal timing for PAP initiation remains unknown. It is therefore not surprising that <20% of the patients appear to be receiving PAP interventions perioperatively.[84]

Nevertheless, given the presumably beneficial effect of PAP utilization and the lack of expectable harm, the ASA and SASM recommend its preoperative initiation, particularly if OSA is severe.[11,19] In patients not responding adequately to PAP therapy, mandibular advancement devices and/or preoperative weight loss may be considered. Intraoperatively, the ASA recommends PAP administration or oral appliances during sedation in patients previously treated with these modalities. Importantly, patients preoperatively adherent to PAP therapy should continue its utilization pre- and postoperatively throughout the entire hospital stay. However, in those previously untreated, the initiation of PAP treatment may be considered if frequent or severe airway obstruction or if hypoxemia is observed during postoperative monitoring.[11]

Postoperative Monitoring and Risk for Deterioration

The period of emergence and recovery from anesthesia is of high concern owing to residual effects of hypnotics and narcotics and their impact on respiratory drive and airway patency. Moreover, worsening of sleep-disordered breathing with exacerbated nocturnal hypoxia and hypercapnia is anticipated in OSA.[60] Patient monitoring is therefore essential for the early identification of clinical deterioration and the stratification of patients requiring extended care.[85] To prevent the occurrence of asphyxia, cardiopulmonary arrest, and death in patients prone to upper airway obstruction, close surveillance is required until there is an assured return of arousal responses after emergence from anesthesia. However, caution and vigilance are essential with subsequent use of sedatives and opioids for postoperative pain management, as a recurrent impairment of protective respiratory arousal should be anticipated.[23]

According to the ASA, patients at increased risk for respiratory compromise from OSA should receive continuous pulse oximetry monitoring and possibly capnography after discharge from the recovery room. This may be provided in critical care or step-down units, by telemetry, or by professional observers in the patient's room and should be maintained as long as patients are considered at increased risk.[11] To establish the ability to maintain adequate oxygen saturation levels, patients can be observed in an unstimulated environment, breathing room air, and preferably while asleep. Currently, pulse oximetry is the most common technique for postoperative respiratory surveillance. However, end-tidal carbon dioxide monitoring can detect adverse respiratory events significantly earlier than the occurrence of oxygen desaturation, even in patients receiving supplemental oxygen.[86] Based on the higher sensitivity and accuracy, capnography may emerge as a measure of increased postoperative safety, presenting an early warning instrument for detecting the vulnerability to respiratory complications beyond the PACU environment.[23,87] Other technologies, such as the impedance-based noninvasive respiratory volume monitoring, have been developed, but require more research in terms of utility and feasibility.[88,89] Furthermore, evidence on opioid-induced respiratory depression also suggests the importance of repeated assessment of sedation levels.[49] As demonstrated by Gali et al,[90] the PACU setting provides an opportunity for a systematic observation of clinical indicators of increased vulnerability to upper airway obstruction.

Despite the significance of postoperative respiratory and sedation monitoring in patients with OSA, current literature is insufficient to guide the appropriate timing of discharge to unmonitored settings. This presents a significant clinical challenge given the emergence of an increasing number of studies that have identified preventable lapses in monitoring as the predominant drivers of life-threatening adverse outcome in OSA.[8,9,61] Nevertheless, resources required to monitor the growing OSA population remain a major concern for health care systems worldwide.

Postoperative Critical Events and Unexpected Death

Critical perioperative complications directly related to OSA are increasingly acknowledged in the legal field, making up a growing percentage of cases adjudicated in the court of law. The particular importance for the anesthesiologic field is evident, given that the majority of adverse events occur perioperatively due to difficult airway management, premature extubation, and monitoring lapses.[61] Adding to the severity of these unfortunate outcomes is the fact that, in retrospect, they are often deemed preventable with appropriate measures of precaution.[61] Studies on postoperative death and near-death events in OSA have consistently confirmed the critical role of postoperative opioids, sedatives, and insufficient patient monitoring in this context.[8] Moreover, closed claims literature revealed that in 50% of postoperative opioid-induced respiratory depression cases, opioids were concurrently administered by more than 1 modality.[9] While the highest risk for opioid-induced respiratory compromise persists within 24 hours of opioid utilization, consumption of typical or less than typical opioid doses was found in 80% of life-threatening and deadly events.

Case reports and malpractice literature often describe hospitalized patients with OSA, that unexpectedly experience death or near-death events, despite acceptable dosing of narcotics and previous satisfactory alertness. Notably, these patients commonly share a unique clinical course, starting off with being awake, alert, and stable, while after going to sleep, patients are found in a critical state or dead.[17,91] This has raised the question of whether or how these events can be prevented. Evidence has shown that subgroups of OSA patients may demonstrate occult arousal failure, expressed in severely delayed arousals in response to repetitive apneas, that allow for extreme desaturation levels to occur before recovery. Given the nature of OSA, this only happens during sleep and can be evidenced by high-resolution oximetry which reveals a distinct respiratory pattern of repetitive reductions in airflow and Spo_2 from cycling collapses and reopening of the upper airway.[17,92] However, when awake, patients with profound arousal failure exhibit no indicative symptoms or signs and therefore remain concealed within the surgical population. In this setting, any perioperative factors that can additionally delay arousal, such as hypnotics and narcotics, significantly increase the risk for respiratory arrest and unexpected death. While the cause and incidence of arousal failure in OSA are not well established so far, acquired arousal failure in response to repetitive hypoxemias over the years presents a viable mechanism. This path has particularly been suggested for severe OSA or obese patients who on average experience more profound cyclic desaturations that may progressively attenuate the arousal response to arterial hypoxemia.[17] To date, there is no conventional way to perioperatively identify arousal failure by any screening methods. However, obesity and disease severity could be indicative of a patient's vulnerability.

Challenges and Future Research

Population-based data indicate that the current implementation of OSA-targeted interventions, including the use of regional anesthesia, the administration of supplemental oxygen, perioperative PAP therapy as well as pulse oximetry monitoring is limited.[84] In this context, 70% of US and Canadian anesthesiologists have reported a lack of departmental policies. Furthermore, significant inconsistencies in monitoring practices persist.[93,94] Underlying reasons are likely linked to the scarcity of evidence on the efficacy of safety measures. Moreover, the increasing prevalence of OSA poses a significant constraint on the feasibility of costly monitoring interventions.[95] The poor specificity of monitoring alarms is further burdened by alarm fatigue and patient discomfort.[95] Concurrently, the lack of adequate monitoring has been identified as a primary cause of catastrophic outcome in OSA.[8,9,61] As health care institutions continue to allocate resources to increase patient safety in the growing OSA population, more accurate screening and monitoring algorithms are needed to stratify particular OSA phenotypes that are susceptible to cardiorespiratory arrest and other life-threatening complications that require advanced surveillance.[95,96] Notably, the prevention of complications is preferable to rescue after complication from both the health care perspective and economically. Nevertheless, current OSA screening tools exhibit high false-positive rates and limited specificity, which can promote the waste of resources by failing to confine enhanced monitoring and treatment to patients truly at increased risk from OSA.[95] As such, the identification of accurate metrics of risk with stronger predictive value than the AHI (eg, Spo_2 or blood CO_2 levels, high-resolution pulse oximetry, cardiac biomarkers, indicators of arousal thresholds) is long needed to facilitate increased patient safety in OSA.[17,97–100] Moreover, new methods and technologies should be investigated because early patterns of clinical instability and evolving death may be too complex for early detection by any unifying numeric threshold.[17]

In conclusion, the diversity and vulnerability of OSA demand a systematic clinical approach for the implementation of aligned and effective measures that provide increased perioperative safety for this growing patient population.

DISCLOSURES

Name: Crispiana Cozowicz, MD.
Contribution: This author helped design the study, conduct the study, write the manuscript and has approved the final manuscript.
Conflicts of Interest: None.
Name: Stavros G. Memtsoudis, MD, PhD, MBA.
Contribution: This author helped design the study, conduct the study, write the manuscript and has approved the final manuscript.
Conflicts of Interest: S. G. Memtsoudis is a director on the boards of the American Society of Regional Anesthesia and Pain Medicine (ASRA) and the Society of Anesthesia and Sleep Medicine (SASM). He is a 1-time consultant for Sandoz Inc and the holder US Patent Multicatheter Infusion System. US-2017-0361063. He is the owner of SGM Consulting, LLC and Centauros Healthcare Analytics and Consulting. S. G. Memtsoudis is also a shareholder in Parvizi Surgical Innovations LLC and HATH. None of the above relations influenced the conduct of the present project.
This manuscript was handled by: Toby N. Weingarten, MD.

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