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Chapter 7

Management of Acute Pain in Obese Patients with Sleep Apnea

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Abstract

Perioperative pain management for obese patients is daily challenges for anesthesiologists especially if complex comorbidities such as Obstructive Sleep Apnea and cardiovascular disease coexist. Limitations to effective pain management in this group are multifactorial, that includes technical difficulty with regional techniques, limited expertise, unavailability of standardized guidelines and lack of familiarity with recent multimodal analgesic regimens. Opioid-related complications such as narcotic-induced ventilatory depression in these group of patients poses another critical concern for both trainees and the experienced anesthesiologists. This chapter is intended for residents, fellows, as well as senior perioperative physicians, and will explore various regional and pharmacological options for acute pain management in this special population based on recent advances and available evidence.

Keywords: acute pain, obstructive sleep apnea (OSA), OSA pathology, opioid interaction with OSA, multimodal pain management

1. Introduction

Obesity is one of the growing worldwide epidemics that has doubled since 1980. In 2016, about 39% of women and men across the world were overweight, and 11% of men and 15% of women were obese [1]. Obesity is a chronic systemic disease characterized by abnormal and excessive fat deposition with a significant impact on individual's quality of life and life expectancy in addition to its burden on public health resources. Overweight and obesity are defined as BMI ≥ 25 kg/m^2 and ≥ 30 kg/m^2, respectively. Beyond that BMI-centric definition, complications-centric approach

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is more comprehensive and clinically relevant to assess the adiposity-based chronic disease (ABCD). Airway obstruction is a common comorbidity in the obese population, with an estimated prevalence of 40% in moderate obesity, and up to 90% in patients with severe obesity. The grade of airway obstruction proportionally correlates with the severity of obesity. It has been shown that each 1-SD increase in BMI increases the risk of OSA four folds [2], moreover a 10% change in body weight increases the severity of OSA by 30%, as measured by apnea–hypopnea index [3]. OSA on top of obesity is associated with worse perioperative outcomes in terms of difficult intubation, postoperative respiratory, atrial fibrillation and other perioperative cardiac events [4]. Given the increasing prevalence of obesity, anesthesiologists are very likely to find themselves caring for obese patients with sleep apnea. This necessitates anesthesiologists and pain physicians to acquire a clear understanding of OSA pathology, familiarity with multimodal pain modalities, experience with regional anesthesia and structured pain management protocols. This chapter is an OSA-centered review of the commonly used pain medications and the most clinically relevant topics in OSA pathology and pain management. It is intended to be a quick review for residents, fellows in training and pain physicians and is equipped with the latest evidence-based management options.

2. Obstructive sleep apnea

Obstructive Sleep Apnea (OSA) is one of four types of breathing problems that occur during sleep. These include sleep apnea due to a central cause, hypoventilation, and hypoxemia during sleep. OSA presents as daytime sleepiness, loud snoring, witnessed apneic episodes, or frequent awakenings due to airway obstruction. In OSA, at least one of the obstructive respiratory events such as apneas, hypopneas or increased respiratory effort related arousals occur per hour of sleep. The alternating episodes of snoring and silence lasting 20–30 seconds are usually associated with a reduction in blood oxygen saturation and interruption of sleep pattern [5]. Risk factors include obesity, male gender and postmenopausal state [6]. It is prevalent in 4% of men and 2% of women, with predominance in 40–60 years of age. It is believed that the number of patients suffering from OSA is underestimated, given the lack of an accurate screening tool [6]. The National Sleep Foundation (NSF) found 26% of American healthy adults at a high risk of OSA based on Berlin questionnaire assessment [6].

OSA could be mild, moderate or severe, depending upon the respiratory disturbance index (RDI). RDI between 5 and 15 is mild, between 16 and 30 is moderate and more than 30 per hour is severe [5, 7]. The severity of OSA requires an objective testing with sleep study using polysomnography or portable monitors at home or drug induced sleep endoscopy (DISE), as it impacts treatment decisions.

3. OSA pathophysiology and its interaction with opioid pharmacology

OSA is a systemic disease with multiorgan involvement that is not limited to obstruction of the upper airways. It is associated with significant negative functional and cognitive outcomes

apart from a higher morbidity and mortality. OSA is associated with cardiovascular morbidities including pulmonary and systemic hypertension, heart disease, ischemic stroke, diabetes and metabolic syndrome [8–12]. During sleep, OSA patients have depressed ventilation, which worsens with any perioperative opioids. It is known that arousal is a protective mechanism.

OSA is the result of interaction between normal sleep physiology and abnormal upper airway anatomy. The narrowing of upper airways can be due to either soft tissues or bony structures. Soft tissues factors include an enlarged tongue, increased soft tissue volume, fat deposition in upper airways or enlarged tonsils in pediatric patients. The latter can be heritable and seen in the absence of obesity [13]. Bony structures can also affect patency of the airway. Increased mandibular length was associated with less risk for OSA development in male individuals [14]. Patients with OSA tend to have an impaired arousal in response to airway obstruction [15].

REM stage of sleep causes a generalized loss of muscle tone including muscles responsible for patency of the upper airway; genioglossus, palatal muscles and hyoid muscles [6–8]. Under physiological conditions, sleep causes narrowed airway caliber, increased airway resistance and increased upper airway compliance [16, 17]. This could increase the work of breathing causing hypoventilation and increased arterial partial pressure of carbon dioxide ($PaCO_2$). During wakefulness, the decreased airflow through a narrowed airway is compensated by increasing respiratory muscle effort thus creating greater negative intrathoracic pressure, a reflex known as mechanical load compensation [18]. This mechanism is normally lost during sleep and is more exaggerated in OSA patients.

Such physiologic changes during sleep may be well tolerated in individuals with an acceptable airway anatomy. But with an already narrowed upper airway, sleep-induced hypotonia can potentially cause complete obstruction of the airway. The interaction between these physiologic and pathologic factors leads to recurrent airway collapse resulting in reduced (hypopnea) or complete cessation of airflow (apnea) despite patient's breathing efforts. This eventually leads to intermittent blood gas disturbances (hypoxia and hypercapnia) and interrupted sleep. The typical feature of OSA is a cyclic breathing pattern that develops, with alternating cycles of obstructive breathing events (sleep) and arousal (wakefulness).

Breathing during sleep is dependent on the chemoreceptor and mechanoreceptor inputs to the brain. Hypoxia and hypercapnia stimulate the central chemoreceptors and peripheral carotid and aortic bodies chemoreceptors which in turn stimulate ventilation and cause cortical arousal. Increased respiratory muscle effort per se can cause cortical arousal [19]. Once arousal occurs, inspiratory upper motor neurons are stimulated resulting in a rapid recovery of dilator muscle activity, return of muscular tone in the genioglossus and pharyngeal muscles with resulting relief of airway obstruction [20, 21].This subsequently leads to restoration of blood oxygen levels and clearance of the accumulated carbon dioxide. When the patient falls asleep, the cycle of obstruction and hypoxia recurs and so on. Arousal is the predominant protective mechanism in OSA, after which oxygen and carbon dioxide levels return to normal. All of the opioids cause varying degrees of sedation and sleep-disordered breathing, that could be central sleep apnea and peripheral OSA [22, 23]. As patients with OSA are at a higher risk of oxygen desaturation, opioids sparing analgesic techniques are preferred [24].

4. Causes of acute pain in patients with OSA

Patients with OSA may experience acute pain in a nonsurgical or a surgical setting. Listed below in **Table 1** are some of the common causes:

Nonsurgical causes	**1.**	**Headache**: mostly morning headache, very common, 50% of OSA patients [25], unknown cause.
	2.	**Coronary artery disease (CAD)**: OSA is associated with higher thrombotic risk [26].
	3.	**Tonsillitis**: pain and difficulty in swallowing [27].
Common surgical causes	**1.**	**Tonsillectomy**: "Post-tonsillectomy pain" due to disruption of mucosa and glossopharyngeal nerve irritation the pharyngeal muscles spasms [28].
	2.	**Uvulopalatoplasty**: [29].
	3.	**Bariatric surgery**: laparoscopic bariatric surgeries which are less painful than open surgeries [30].

Table 1. Some common causes of acute pain in patients with OSA.

5. Drug treatment options

The choice of one analgesic over the others is dependent on its safety profile and the interaction with OSA and its complications. Interestingly, the systemic effects of OSA like chronic hypoxemia and systemic inflammation can increase analgesic sensitivity to opioids [31]. Chronic intermittent hypoxemia activates hypoxemia inducible factor-1 alpha which in turn increases expression of mu opioid receptor and delta opioid receptor expression, augmenting opioid sensitivity [32].

5.1. Opioid medications

5.1.1. Opioid effects on ventilation

Opioids depresses the respiratory drive by inhibition of respiratory centers in the brain stem, thus decreasing the respiratory rate and the tidal volume [33]. Opioids could also obstruct the upper airway through loss of muscle tone due to sedation or through direct inhibition of central neurons responsible for maintaining upper airway muscle tone [34]. Moreover, it alters the upper airway reflexes and responses to ventilatory depression [35].

The respiratory depression results in hypoventilation with subsequent increase in $PaCO_2$ [33]. These effects are dose-dependent and could be life-threatening with higher doses of opioids or multiple boluses that are commonly used in perioperative period or after major trauma. Those additive risks in OSA patients with already depressed ventilation and airway obstruction could increase the risk of respiratory events and mortality after recovery from anesthesia. Therefore, it is prudent to start with the lowest dose recommended and carefully titrate to effect.

5.1.2. Opioid-induced sleep disturbances

Patients without OSA who are on chronic opioid therapy have been reported to have sleep disturbances, impaired self-reported sleep and poor sleep quality [36]. These effects might exacerbate already disturbed sleep in OSA patients.

5.1.3. Opioid effects on the cardiovascular system

Opioids have unfavorable hemodynamic effects in OSA patients such as hypotension, orthostatic hypotension, and syncope of various degrees. The negative hemodynamic effects are due to central vasomotor depression, direct myocardial depression, and arteriovenous dilatation in higher doses [37]. In general, cautious use of opioids should be considered in patients with severe OSA associated with hypertension, arrhythmias and heart failure. Fentanyl has a relatively favorable cardiovascular profile compared to other opioids. For anesthetic management, it has modest effects on blood pressure and myocardial contractility. It is well tolerated as an analgesic regarding cardiovascular adverse effects compared with other opioids. Morphine has the greatest potential for histamine release, compared to other opioids, resulting in hypotension. Hydromorphone can cause a greater drop-in blood pressure compared to equipotent doses of morphine. Tramadol has a lower risk of cardiovascular adverse events, but it can lead to serotonin syndrome and cardiac arrhythmias. Meperidine administration leads to significant decrease in blood pressure and cardiac output due to direct myocardial depressant effects and peripheral vasodilatation. It can predispose patients to serotonin crisis [38]. Methadone is recently becoming popular in chronic pain management. However, it has been associated with QTc prolongation with a risk of torsade de pointes [35, 39].

5.2. Nonsteroidal anti-inflammatory drugs (NSAIDs)

These are foundational analgesics and highly effective as they have lower numbers needed to treat (NNT) comparable with other medications. Ketorolac and Ibuprofen are the commonly used NSAIDs in a perioperative setting. The American Society of Anesthesiologists (ASA) taskforce recommends the use of perioperative NSAIDs to decrease narcotic consumption [40]. A reduction in narcotic consumption in the postsurgical period was achieved by regular administration of Ketorolac in morbidly obese patients [41, 42]. Likewise, intravenous intraoperative Ketorolac infusion has been shown to reduce pain scores in the same population [42]. NSAIDs should be used with caution in patients with hypertension, arrhythmias, heart failure and chronic kidney disease. They can increase the risk of serious cardiovascular events, myocardial ischemia, and stroke and the risk is proportional to the duration of their use. Selective COX 2 inhibitors carry a higher risk than nonselective COX inhibitors. If NSAIDs use is necessary for high-risk patients, it is advisable to use the lowest dose possible for the shortest duration. NSAIDs could precipitate acute kidney injury in patients with compensated heart failure or diabetic nephropathy and also further worsen underlying chronic kidney disease (CKD) [43, 44]. The hyperkalemia is mild, but could be critical in patients with elevated K^+ serum levels due to CKD or with concomitant use of ACE inhibitors. The inhibition of platelet aggregation through decreased production of thromboxane A_2 could potentially increase bleeding in patients already on anticoagulants.

5.3. Acetaminophen

Acetaminophen is a commonly used foundational analgesic with perioperative opioid sparing properties. It offers the distinct advantage of being relatively safe and devoid of sedative properties. Addition of acetaminophen to an intravenous opioid PCA regimen was associated with a 20% opioid sparing effect in a meta-analysis of mixed surgical population [45]. Similar opioid dose reduction expected in patients with OSA could therefore potentially reduce opioid-induced ventilator problems. Obese bariatric surgical patients receiving postoperative intravenous acetaminophen 1 gram every 6 hours required fewer morphine equivalents [46]. Acetaminophen lowers cumulative narcotic consumption, regardless of the route of administration [47, 48]. It has the most favorable effect on blood pressure and should be considered as the first line treatment option in patients with hypertension or cardiovascular disease.

5.4. Anticonvulsant agents

Anticonvulsant agents in clinical use for their analgesic benefits include Pregabalin and Gabapentin. The preoperative use of Pregabalin as premedication significantly lowered immediate postoperative requirement of pain medications among bariatric surgical patients. [49] Preoperative Gabapentin has also significantly lowered immediate postoperative pain scores in morbidly obese bariatric surgical patients [50]. However, the most common side effects of these drugs are dizziness and somnolence, which potentially add to the OSA induced somnolence. Postoperative respiratory depression with the use of Pregabalin has been reported in a patient with undiagnosed obstructive sleep apnea [51]. Concomitant opioid use could increase the risk of respiratory depression and caution should be considered. The Ottawa hospital algorithm for Pregabalin use recommends either avoiding or cautiously titrating low doses depending on the clinical setting and patient characteristics [51]. Pregabalin and Gabapentin could cause peripheral edema which is not related to cardiac, hepatic or kidney failure [52].

5.5. Alpha $_2$ agonists

Alpha $_2$ agonists such as Clonidine and Dexmedetomidine are used in perioperative setting for their analgesic properties. A meta-analysis conducted in a mixed general surgical population suggested that perioperative alpha $_2$ agonists reduce narcotic consumption in the postoperative period [53]. However, very limited data is available regarding their use in obese patients with OSA. Nonopioid anesthetic with Dexmedetomidine in obese patients with OSA resulted in low pain scores in Post Anesthesia Care Unit (PACU) and no perioperative events [54]. Among patients with morbid obesity, substitution of intraoperative opioids with an intraoperative Dexmedetomidine infusion resulted in reduced perioperative opioid requirements [55]. Also, perioperative Dexmedetomidine infusion were found to significantly reduce postoperative opioid consumption (24-hours) as compared to conventional perioperative analgesic regimens [56]. Alpha $_2$ agonists can cause sedation which could interfere with arousal in OSA patients. However, their opioid sparing benefits may offer greater benefit than the risks. Clonidine causes adverse effects like hypotension, bradycardia, sinus and AV nodal block [57]. Dexmedetomidine causes hypotension and bradycardia more often than hypertension

and tachycardia. It may also reduce atrial fibrillation (AF) induction among adult patients with history of paroxysmal AF.

5.6. Ketamine

Ketamine, an *N*-methyl-d-aspartate (NMDA) antagonist has recently received increasing attention as a relatively safe adjuvant analgesic. In patients with morbid obesity, low-dose Ketamine added to intravenous Morphine PCA resulted in a significant reduction in opioid consumption. Importantly, subjects receiving ketamine in addition to morphine had fewer episodes of desaturation postoperatively [9]. A preinduction dose of ketamine of 0.5 mg kg^{-1} together with Clonidine significantly lowered pain scores and perioperative opioids consumed [58]. Intra-operative Ketamine at doses up to 1 mg kg^{-1} was shown to decrease opioid consumption in recovery but at the cost of significant drowsiness [59].

5.7. Tramadol

It is an analgesic of intermediate potency with possible advantages in OSA patients due to its multimodal mechanism and a relatively lower risk of respiratory depression [60].

6. Multimodal strategies for pain management in obese patients with OSA

Multimodal analgesia is the use of a combination of different analgesic medications and techniques with an aim to provide optimal pain control, thus allowing reduction in opioid requirements. They may have additive or synergistic effects by acting through diverse mechanisms either peripherally or centrally in the nervous system [61]. Multimodal analgesia with a combination of IV Paracetamol and IV Ketorolac in bariatric surgery was found to reduce the postoperative opioid consumption by about 70% [62]. A nonopioid analgesic regimen that employed a combination of nonopioid analgesics (Ketorolac, Clonidine, Ketamine, Lidocaine and Magnesium) in thirty morbidly obese patients undergoing bariatric surgery resulted in lesser PACU opioid consumption and less sedation [63]. The opioid-sparing multimodal techniques are summarized in **Table 2**.

Magnesium, intravenous local anesthetic infusions (lidocaine), and Clonidine are some additional drugs that have been used perioperatively in patients with morbid obesity to enhance analgesia [64, 65] but more scientific evidence is needed before adopting these agents into routine clinical use.

6.1. Regional analgesia techniques in patients with obstructive sleep apnea

Narcotic-based pain regimens for OSA patients have a risk of opioid-induced respiratory depression. Hypoxia, sleep disturbance, pain, and disturbed opioid responses in OSA contribute to that risk [66, 67]. Neuraxial techniques and peripheral nerve blocks are effective

Nonopioid analgesics include Nonsteroidal anti-inflammatory drugs (NSAIDs), Acetaminophen, Analgesic adjuvants such as Ketamine, Dexmedetomidine, and Clonidine may also decrease postoperative opioid requirements.
Regional analgesia with local anesthetic (e.g., peripheral nerve blocks, epidural analgesia)

Table 2. Opioid-sparing techniques include a combination of the following.

interventions and superior alternatives in pain management toolkits. It offers a superior analgesic effect and minimizes the need for systemic analgesics [68, 69].

Perioperative management by regional analgesic techniques rather than systemic opioids has been recommended by the American Society of Anesthesiologists since 2014, with an aim to reduce the likelihood of OSA-related perioperative adverse outcomes. The beneficial effects of perioperative regional analgesic techniques on patient outcomes have been proved in general surgical population [70, 71]. Still, the evidence in OSA patients is inconclusive as it was driven mainly from case reports or small retrospective case–control studies [67, 72].

6.2. Neuraxial analgesia techniques

Neuraxial analgesia is a modality with high efficacy and can be used effectively as a sole analgesic approach. Its beneficial effects on respiratory functions such as superior spirometry in the immediate postoperative period and lower postoperative pulmonary complications have been consistently documented in many studies [73, 74]. The risks in morbidly obese patients include respiratory depression secondary to a rostral spread of neuraxial opioids that could lead to a postoperative respiratory arrest [75, 76]. Therefore, the ASA task force recommends that expected benefits should be weighed against the potential risks.

Technical difficulty with procedure failure has been proposed as another challenge in OSA patients, who are often obese. Yet, these concerns lack conclusive evidence, and most have been driven from opinion-based reports. Studies in obese pregnant have showed that the incidence of technical difficulty for epidural anesthesia is overrated. The success was correlated with optimal positioning prior to placement and good quality of palpable surface landmarks [77]. Preprocedural ultrasonography (US) of the spine could accurately identify the intervertebral space and predict the needle insertion depth in intrathecal space thus facilitating placement of an epidural catheter. Systematic review and meta-analysis have shown that spine US has a greater accuracy than manual palpation of surface anatomical landmarks [78]. This could lead to a decreased risk of technical failures and the number of needle punctures.

6.3. Peripheral nerve blocks (PNB)

PNB modality is another pillar of opioid-sparing analgesic techniques. It includes upper extremity, lower extremity, and planar blocks such as transversus abdominis plane (TAP) block, paravertebral block, and erector spinae plane block (ESP). Initially, studies showed that obesity (BMI > 25 kg/m^2) is independent risk factor for block failure. [45] However, other

studies showed not much of a difference in success rate between obese and nonobese and proved that success mainly depends on experience of anesthesiologist and the use of ultrasound-guided techniques [46]. Upper extremity blocks include brachial plexus block that could be performed at different levels. It offers good analgesia for patients with OSA who have a respiratory compromise. Phrenic nerve block is a theoretical risk with inter-scalene block. There is insufficient evidence to recommend its use in this group of patients [79]. Ultrasound-guided transversus abdominis plane (TAP) block is performed in laparoscopic and open abdominal surgeries as it effectively blocks T10 to L1 segments. A successful bilateral TAP block is effective for abdominal midline incisions, especially in nonobese and in situations of failed or difficult epidurals [63]. Paravertebral block is another option for thoracoabdominal surgeries. It could be administered as a single shot or continuous technique, either intra or postoperatively. Multilevel paravertebral block has shown to be successful and opioid-sparing in obese females undergoing breast surgery [80]. The erector spinae plane block (ESP) is a recently evolved simple technique that could be performed under ultrasound guidance. It is gaining popularity for its effective pain relief for somatic and visceral pain. However, further clinical investigation is needed to clearly establish its efficacy in OSA patients. Perioperative analgesia for OSA patients could be carefully planned in the perioperative period. Many effective regional analgesia modalities are currently available for intraoperative and postoperative.

7. Combined strategies for analgesia

Preemptive analgesia could obtund nociceptive responses prior to surgical stimulus and possibly decrease postoperative pain. Moreover, it could possibly decrease the probability of conversion of acute pain to chronic pain [81]. Postoperative patient-controlled intravenous analgesia (PCA) or epidural analgesia (PCEA) have been shown to decrease narcotic consumption and provide a high degree of patient satisfaction [82]. Other techniques like long-acting local anesthetic infusions at the surgical wound site or even intraperitoneal infusion have shown promising results [83, 84].

Poor pain management of morbidly obese patients increases postoperative complications [85]. Therefore, the use of multimodal analgesia can solve this problem, consequently improving patient satisfaction and reduce postoperative morbidity.

Developing an optimal evidence-based pain management protocol tailored to obese patients with OSA is a challenging task. The majority of current recommendations are either based on studies with small sample size or lacking a scientifically rigorous study design. Given the paucity of literature in obese patients with sleep apnea, it is difficult to draw a definitive conclusion. Yet, the obvious benefits of multimodal analgesic regimen make it popular in regular clinical practice.

8. Nonpharmacological options to pain management

The nonpharmacological options are summarized in **Table 3:**

Peripheral therapies (physical skin stimulation)	
Transcutaneous electrical nerve stimulation (TENS)	Application of electricity to stimulate the skin can be used to manage different types of pain. The mechanism could possibly be explained by Gate control theory. There is a lack of strong evidence to suggest benefits of TENS in labor pain
Hot-cold treatment	Can be used in acute musculoskeletal injury [86].
Acupuncture and acupressure	It could reduce cancer pain intensity [87]
Exercise	To relieve pain by preventing spasms and contractures [13].
Positioning	Adequate position in the postoperative period by pillows and special beds. [13]
Restriction of movement/resting	Prolonged bed rest is not recommended—the bed rest for about two days may be beneficial after back surgeries by reducing edema.
Cognitive-behavioral therapies	
Distraction by music	Decreases pain sensitivity and increase pain tolerance
Reflexology	Special techniques employed to apply pressure at reflex points on feet corresponding to various body parts decreases pain perception by altering physiology [13].
Meditation, Yoga, Hypnosis, Herbal treatments, Aromatherapy, Relaxation—respiration techniques	

Table 3. Summarization of the nonpharmacological options.

9. Monitoring, oxygen and positive airway pressure therapy for obese patients with sleep apnea

9.1. Monitoring

Oxygen saturation and respiratory rate should be closely monitored in these group of patients receiving opioids or other potentially sedative medications [88]. For continuous assessment of the adequacy of ventilation in the postoperative setting, end-tidal CO_2 measurement has become the standard of care [89, 90]. End-tidal CO_2 detects hypoventilation earlier than any other physiological monitors. OSA patients at high risk should be continuously monitored with pulse oximetry until patient maintains oxygen saturation to their preoperative baseline levels [91, 92]. Studies have shown that pulse oximetry monitoring in postoperative patients is very useful in detecting hypoxemic episodes [40, 93].

9.2. Oxygen therapy

Obese patients with suspected or confirmed OSA should be transferred with supplemental oxygen from operation room to the post-anesthesia care unit (PACU) after receiving general anesthesia. In the PACU, patient head end elevation by at least 30 degrees is recommended. Though there is not enough literature that shows the effect of supplemental oxygen in postoperative settings, the recommendation is that supplemental oxygen should

be continued in all postoperative OSA patients until they maintain preoperative (baseline) oxygen saturation on room air [91].

9.3. Positive airway pressure therapy

Use of CPAP in PACU is recommended for OSA patients who were using it preoperatively at home and also when patients get frequent attacks of airway obstruction in the recovery room [91, 94]. It should also be continued on the inpatient units. CPAP has shown to reduce the incidences of apnea and hypopnea episodes when compared to the preoperative baseline. CPAP is associated with improved ventilation in postoperative OSA patient and also it has shown reduced hospital stay [95–97].

Patients who receive long-acting opioids should be monitored closely and may need high dependency unit admission for postoperative monitoring. Indications for obese patients with OSA for HDU or ICU admission are preexisting co-morbidities, limited functional capacity, major surgery, poorly controlled OSA requiring systemic opioids. The patient should be discharged to the unmonitored settings only when adequate oxygen saturation is maintained to baseline level on room air and with no more risk of respiratory depression (apnea or hypopnea) that can be determined ideally when the patient is asleep [91].

10. Recommendations from guidelines in obese patients with OSA

High-risk OSA patients should be managed by a dedicated specialized anesthesia team. Regional anesthetic technique or nonopioids analgesics should be used to reduce the need for systemic opioids. Meticulous monitoring is required when sedatives and opioids are used in suspected or OSA patients due to high risk of respiratory depression. Even HDU or ICU admission for monitoring could be considered if obesity is associated with other co-morbidities. OSA and its complications should be anticipated in obese patient. Postoperative use of supplemental oxygen is recommended as it reduces the incidence of hypoxemic episodes, in addition to CPAP or NIPPV, that could relieve the upper airway obstruction.

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