REVIEW ARTICLE

Obstructive Sleep Apnea— a Perioperative Risk Factor

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SUMMARY

Background: Obstructive sleep apnea (OSA) is a common disorder of breathing but is probably underappreciated as a perioperative risk factor.

Methods: This review is based on pertinent articles, published up to 15 August 2015, that were retrieved by a selective search in PubMed based on the terms “sleep apnea AND anesthesia” OR “sleep apnea AND pathophysiology.” The guidelines of multiple specialty societies were considered as well.

Results: OSA is characterized by phases of upper airway obstruction accompanied by apnea/hypoventilation, with hypoxemia, hypercapnia, and recurrent overactivation of the sympathetic nervous system. It has been reported that 22% to 82% of all adults who are about to undergo surgery have OSA. The causes of OSA are multifactorial and include, among others, an anatomical predisposition and/or a reduced inspiratory activation of the bronchodilator muscles, particularly when the patient is sleeping or has taken a sedative drug, anesthetic agent, or muscle relaxant. OSA is associated with arterial hypertension, coronary heart disease, and congestive heart failure. It can be assessed before the planned intervention with polysomnography and structured questionnaires (STOP/STOP-BANG), with sensitivities of 62% and 88%. The utility of miniaturized screening devices is debated. Patients with OSA are at risk for perioperative problems including difficult or ineffective mask ventilation and/or intubation, postoperative airway obstruction, and complications arising from other comorbid conditions. They should be appropriately monitored postoperatively depending on the type of intervention they have undergone, and depending on individually varying, patient-related factors; postoperative management in an intensive care unit may be indicated, although no validated data on this topic are yet available.

Conclusion: OSA patients need care by specialists from multiple disciplines, including anesthesiologists with experience in recognizing OSA, securing the airway of OSA patients, and managing them postoperatively. No randomized trials have yet compared the modalities of general anesthesia for OSA patients with respect to postoperative complications or phases of apnea or hypopnea.

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Obstructive sleep apnea (OSA) is a common disorder of breathing but is probably underappreciated as a perioperative risk factor. This review article, based on a selective literature search, is intended to illuminate current pathophysiological concepts of OSA and, in particular, its perioperative management. The information presented here is derived largely from prospective and retrospective observational studies, as well as a few randomized and controlled trials (RCTs), experimental studies, expert opinion, and guidelines.

Epidemiology and predisposing factors

The reported prevalence of obstructive sleep apnea (OSA) in the overall population is 3–7% in men and 2–5% in women (1), although one recent study yielded much higher figures (49.7% and 23.4%, respectively) (2). 22–82% of all patients in adult preoperative cohorts have OSA (3, 4, e1). It is noteworthy that 82–93% of preoperative patients with OSA have not received the diagnosis preoperatively, to say nothing of being treated for it (e2). The anesthesiologist is thus often the first doctor to be confronted with an undiagnosed OSA patient. Untreated OSA is associated with a higher risk of comorbidity (*Table 1*) (6, e3, e4, e12–e14), which is explicable at least in part by increased sympathetic activity resulting from repetitive nocturnal bouts of hypoxemia, hypercapnia, and arousal episodes (e15). Daytime sleepiness in persons with OSA also increases the risk of traffic accidents (odds ratio [OR]: 6.3; 95% confidence interval [CI]: [2.4; 16.2]) (e16). The identified risk factors for OSA (*Box*) include, in particular, obesity—70% of obese persons have OSA (e24, e25). Aging is also associated with increased collapsibility of the upper airway. The prevalence of OSA nearly quadruples from the third to the sixth decade of life, rising from 1.5% to 5.7% (8, e26). Congestive heart failure in persons with OSA leads to nocturnal redistribution of edema fluid into the head and neck, which, in turn, aggravates OSA (9). The three risk factors just mentioned, along with the high prevalence of obesity and the aging of the population, will probably make OSA an increasingly common problem in the years to come.

As recommended in the guidelines of the American Academy of Sleep Medicine, the severity of OSA is graded by the number of episodes of apnea and hypopnea per hour of sleep (the apnea-hypopnea index, AHI). OSA is considered mild if the AHI is in the 5–15 range, moderate if between 15 and 30, and severe if over 30 (10).

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TABLE 1

Comorbidities and perioperative complications in patients with obstructive sleep apnea

Organ system	Comorbidities	Odds ratio	Perioperative complications	Odds ratio
Lungs			hypoxemia hypercapnia (aspiration) pneumonia ARDS pulmonary embolism	7.9 4.2 1.37–1.41 1.58–2.39 0.9–1.22
Cardiovascular system	arterial hypertension congestive heart failure coronary heart disease	1.42–2.89 2.2 2.13	cardiac arrhythmia perioperative myocardial ischemia	1.25 2.6
Central nervous system	stroke	1.58–3.3	delirium	4.3
Metabolism	abnormal glucose tolerance metabolic syndrome	1.44 9.1		
Effect on treatment			need for NIV/reintubation unplanned ICU stay prolonged hospitalization	1.95–9.2 4.4 1.7

adapted from (3, 5, e3–e11)

ARDS, acute respiratory distress syndrome; NIV, non-invasive ventilation; ICU, intensive care unit

The pathophysiology of obstructive sleep apnea

Subatmospheric inspiratory airway pressure can lead to airway obstruction if the forces opposing airway collapse are diminished, particularly in small airways subject to elevated extraluminal pressure (*Box*). The most important upper airway opener is the genioglossus muscle, which is activated in synchrony with the inspiratory muscles of respiration; it dilates the airway and prevents collapse (e27, e28). When awake, patients with OSA can compensate for the increased tendency of their airways to collapse by increased activation of the airway-opening muscles (e29). In many OSA patients, the inspiratory activation of the airway-opening muscles is depressed in sleep to a greater extent than in persons without OSA and is too weak to prevent airway obstruction, especially during the “rapid eye movement” (REM) phase of sleep and when the patient is supine (11).

Sedation and anesthesia weaken the activation of the airway openers just as sleep does, potentially leading to airway obstruction (e30). In persons without OSA, this can be observed in the residual effects of muscle relaxants (12, 13) (*Figure*) as well as the anesthetic agents isoflurane (14) and propofol (15). Local anesthesia of the upper airway also promotes airway obstruction (e31) by blocking afferent neural impulses from the airway, thereby inactivating the protective reflexes that keep it open.

The narrowest parts of the upper airway in patients with OSA are the retroglottal and/or retropalatal spaces; the resulting abnormality of inspiratory gas flow is designated as “Starling” resistance (e32). The airway pressure below which the airway collapses is called the critical closing pressure (Pcrit). The Pcrit is a quantitative measure of the severity of airway collapsibility. It indicates the amount of continuous positive airway pressure (CPAP) that will be needed to eliminate inspiratory airway obstruction (16, 17, e32, e33).

Perioperative morbidity in patients with obstructive sleep apnea

Patients with OSA have an elevated risk of perioperative complications (*Table 1*) (5, 18, e5). OSA is also an independent predictor of difficult airway management (19). Thus, intubation was difficult in 22–44% of patients with OSA, compared to 2–3 % of patients without OSA (20, e34); in 5% of cases, the attempt to intubate was unsuccessful (e35). An analysis of medicolegal databases in the USA (1991–2010) has shown that difficult airway management and respiratory arrest without respiratory monitoring in patients with presumed or confirmed OSA are the complications that most commonly lead to legal proceedings (21).

Hypnotic, sedative, analgesic, and muscle-relaxing agents given during general anesthesia suppress the activation of the airway muscles and increase airway collapsibility in healthy patients, and even more so in patients with OSA (13, 15, 16, e36, e37). They increase the risk of postoperative airway obstruction.

The perioperative complications of patients with OSA (3, 5, 18, e6) are apparently not limited to the period when intraoperatively administered drugs are still exerting their effects. In one study, the postoperative AHI actually increased from the first to the third night (22), indicating a likely REM rebound effect (23, e38).

Preoperative screening and evaluation

Identifying patients with OSA before surgery

According to epidemiological studies, many persons about to undergo surgery have OSA but have not been given the diagnosis preoperatively (e2); such patients are at greater risk of complications than those with already diagnosed OSA. It is, therefore, the interdisciplinary, guideline-recommended responsibility of anesthesiologists, specialists in sleep medicine, and

surgeons to identify patients with OSA before surgery and, if the operation is elective, to proceed to further diagnostic assessment of OSA before it is performed (24). This is so both for medicolegal reasons and because knowing the patient has OSA will probably help the physicians and surgeons optimize the perioperative management, and with it the clinical outcome.

Particular attention must be paid to clues to OSA that can be found in the patient's medical records, the history taken from the patient and/or another person, and a detailed physical examination, which is an obligatory prerequisite to anesthesia in any case (Table 2). A history of possible sleep apnea should be obtained from the patient's bed partner as well. Questionnaires that have been validated in prospective observational studies (Table 3), such as the STOP and STOP-BANG questionnaires, simplify and standardize the identification of patients with OSA (25, 26, e40–e42). Their sensitivity ranges from 65% to 80% (25, e42, e43). If the patient answers “yes” to five items on the STOP-BANG questionnaire, there is a high likelihood of moderate (OR: 4.8 [2.80; 8.03]) to severe OSA (OR: 10.4 [4.46; 24.26]). If fewer than three questions are answered “yes,” the likelihood of OSA is low (e42).

Diagnostic evaluation and the role of polysomnography

Polysomnography is the gold standard for the diagnosis of OSA, according to sleep medicine guidelines (27, e44). It involves the continuous recording of multiple biological signals and is thus technology-intensive, time-consuming, and expensive. It is, nonetheless, the only way to assess the severity of OSA definitively, so that suitable treatment options can be chosen and tested. One such option is the application of positive airway pressure (PAP), either continuously (CPAP) or in a manner that is automatically titrated to the inspiratory flow of respiratory gases (APAP), through a nasal or oral-nasal mask. It remains unknown, however, whether the definitive diagnosis of OSA by polysomnography in fact lowers the perioperative risk.

To simplify screening for OSA and help select patients for polysomnography, miniaturized devices have been developed that measure a smaller number of biological signals simultaneously. These can be used by the patient at home (e45), but their clinical utility is still unclear. The latest S3 guideline of the German Sleep Society (Deutsche Gesellschaft für Schlafforschung und Schlafmedizin, DGSM) entitled “Non-Restorative Sleep and Sleep Disturbances” contains the recommendation that such systems should be used to evaluate OSA only under certain precisely defined conditions, and when there is a high pretest probability of OSA, as implied, e.g., by the history, physical examination, and patient questionnaires (28). Nonetheless, such devices can be useful, among other things, for the detection of severe OSA, or of OSA in need of treatment, immediately before surgery. It remains to be seen whether they can truly detect OSA with greater sensitivity and specificity than questionnaires alone. Surgeons and anesthesiologists still have to decide, in each individual

BOX

Factors that promote and prevent upper airway collapse*

Factors that increase airway collapsibility

- Negative inspiratory pressure
- Extraluminal pressure, caused (e.g.) by:
 - prevertebral fat deposition
 - a small mandible
- Narrow airway, due to (e.g.):
 - obesity
 - macroglossia (in trisomy 21 or other conditions)
 - enlarged (“kissing”) tonsils
- Further risk factors:
 - age
 - male sex
 - pregnancy
 - menopause
 - alcohol use, cigarette smoking
 - (residual) effect of sedatives, anesthetic drugs, muscle relaxants

Factors that decrease airway collapsibility

- tonic and inspiratory phasic contraction of the upper airway musculature (genioglossus m., m. tensor veli palatini, other muscles)
- increased lung volume (longitudinal pull on trachea)
- being awake

* adapted from (7, e15, e17–e23)

case, with what degree of certainty the diagnosis of OSA should be ruled in or out before surgery.

Patient monitoring and treatment strategies

Preparing patients with obstructive sleep apnea for surgery

Weight loss for severely obese patients (24), albeit rarely practical, and the optimal management of comorbid illnesses are indicated. Patients should be asked whether they are already being treated with PAP and, if so, at what pressure level, as this varies widely from patient to patient. Postoperative PAP treatment should already be started in the recovery room as soon as the patient is able to cooperate; thus, patients should be instructed to bring their PAP devices along when they are admitted to the hospital. It is helpful for the ward staff to be knowledgeable about the workings of PAP devices, though this goal is hard to attain given the wide variety of devices now in use.

In an RCT conducted in Canada, the application of PAP treatment for one to three nights before surgery in patients who had never had PAP before significantly lowered the postoperative AHI from 30.4 (25th–75th percentiles, 23.2–41.9) to 3.0 (1.0–12.5), in comparison to a control group without PAP (AHI 29.0 [18.8–40.8] versus 31.9 [13.5–50.2]). Fewer than half of the patients, however, were able to tolerate PAP for more than four hours (29). The rate of postoperative complications, and hypoxemic phases in particular, was

The minimal retroglottal airway diameter during forced inspiration in awake healthy subjects under partial neuromuscular blockade

Data acquisition by magnetic resonance imaging, triggered by respiratory gas flow, under control conditions and in the setting of partial neuromuscular blockade with train-of-four (TOF) ratios of 0.5 and 0.8, upon recovery to the normal value, and 15 minutes after neuromuscular blockade is no longer demonstrable.

The residual effects of muscle relaxants given during anesthesia increase upper airway collapsibility by impairing the function of the airway-opening musculature. A similar impairment is seen in patients with obstructive sleep apnea during natural sleep. Adapted from (13).

* $p < 0.05$ versus control

the same in both groups (48.3% in both). Another RCT of PAP revealed no benefit (30). A very recent RCT, however, documented the efficacy of postoperative monitored CPAP in obese patients with OSA after bariatric surgery (31). In its guidelines, the American Society of Anesthesiologists (ASA) recommends considering treating newly diagnosed OSA patients (AHI >30) with PAP (24).

Poor compliance with PAP is not surprising when the pressure levels used are not individually adjusted. PAP that is too low does not prevent airway collapse, while PAP that is too high increases pulmonary volume and gives the patient an uncomfortable feeling of overexpansion, while making the work of breathing more difficult. Only the recently introduced “smarter” devices adjust the level of PAP optimally for the individual patient; at present, however, such devices are scarcely available in German hospitals for the perioperative setting. It is unknown whether postoperative PAP treatment lowers not only AHI values, but also the rate of serious complications.

Premedication

Pathophysiological studies have shown that sedatives such as benzodiazepines impair airway function (e36). They may also elevate the arousal threshold for the mechanisms that enable persons with OSA to wake up when their airways are obstructed. It is, therefore, recommended in pertinent review articles that great caution should be exercised in giving benzodiazepines

preoperatively to patients with OSA (e46). This recommendation is not based on hard data. Potential alternatives include centrally acting, sedating α_2 -agonists such as clonidine and dexmedetomidine, whose safe use has at least been documented in case reports (32, e47, e48).

Techniques of anesthesia

As a general principle, the technique of anesthesia in a patient with OSA, as in any patient, should be optimally tailored to the patient's comorbidities and individual risks, and to the type of procedure that is to be performed. The ASA has developed a simple (though as yet unvalidated) score for risk stratification, based on a small number of variables (24) (eTable). Unfortunately, there is a lack of prospective, randomized trials concerning the optimal anesthesia regimen for patients with OSA. The ASA's current recommendations are, therefore, based on expert opinion rather than hard evidence. It seems intuitively reasonable to use short-acting, easily managed drugs. The authors' own, as yet unpublished data suggest that a combination of propofol and remifentanyl, or of sevoflurane and remifentanyl, enables safe general anesthesia for OSA patients without elevating the postoperative AHI over its preoperative value.

Local and regional anesthesia should be used rather than general anesthesia if possible (3, 24), although elevated AHI values have been described after regional anesthesia as well (33). If the patient is to be sedated, the potentially adverse effect of sedation on airway integrity must be kept in mind. CPAP treatment is sometimes useful intraoperatively and can be delivered by any good anesthesiological ventilating machine. As recommended in the guidelines, the patient's spontaneous respiration must be continuously monitored with capnography and oximetry (24). Snoring indicates partial airway obstruction. If deeper sedation is needed, the timely induction of general anesthesia via an endotracheal tube or laryngeal mask may well be a less risky procedure overall. Analogous considerations presumably apply to endoscopies.

Securing the airway

Patients with OSA have a higher rate of difficult intubation during the induction of general anesthesia, and a higher rate of intubations that are not possible at all by conventional means (19, 20, e34, e35). The technique of intubation should be based on the ASA Difficult Airway Algorithm (e49). A well-trained anesthesiologist can choose among a broad palette of techniques, including the use of a special video laryngoscope or fiber-optic awake intubation under surface anesthesia, in order to lessen the risk of an unsuccessful attempt to intubate the patient.

Review articles and clinical-experimental studies have led to the conclusion that patients with OSA should be extubated only when fully awake. In other words, the patient should not only be breathing adequately through the endotracheal tube, but should

also open his or her eyes when spoken to, and should be oriented. Whenever possible, the patient is put in the lateral position, or else the head of the table or bed is raised, and this position is maintained in the recovery room (3, 34). Before extubation, any clinically significant residual muscle relaxation must be ruled out by quantitative neuromuscular monitoring: in a train of four (TOF) supramaximal peripheral nerve stimulations administered over two seconds, the amplitudes of the muscle contractions evoked by the first and last stimuli should be very close (TOF-ratio 0.9–1.0). Residual neuromuscular blockade should be antagonized with cholinesterase inhibitors or with sugammadex, a cyclo-dextrin. The only alternative is to keep the patient on the ventilator (13, 16, 35, e50). On the other hand, the “well-meant” pre-emptive antagonism of merely suspected residual muscle relaxation is likely to do more harm than good. Cholinesterase inhibitors can impair neuromuscular transmission, threatening the integrity of the upper airway (17). In view of its pathophysiological mechanism, any residual neuromuscular blockade has implications similar to those of inadequately treated myasthenia gravis for the integrity and function of the upper airway (12, 13, 16, 36, e50).

Postoperative Treatment

Analgesia: The widespread notion that patients with OSA should not be given opioids after surgery has no basis in pathophysiology. Opioids depress the central respiratory drive, but they have no effect on airway integrity unless given in an excessively sedating dose. Patients with OSA have a diminished pain tolerance (37). Thus, even when non-opioid analgesic drugs are given, opioids may still need to be added on for effective pain relief (38, e36). Review articles support the efficacy, for adjuvant use, of centrally active α_2 -agonists such as clonidine or dexmedetomidine (32). Patient-controlled intravenous analgesia (PICA) can be used in the postoperative setting, but continuous opioid drips should be avoided, and sedatives as well.

Oxygen supplementation: Postoperative oxygen supplementation raises the arterial partial pressure of oxygen and thereby prolongs the apnea phases of patients with OSA, not always leading to relative hypoxemia that can be detected by pulse oximetry. Routine oxygen administration is nonetheless recommended until the patient can reach and maintain the same individual oxygen saturation value on room air as was present before anesthesia (24). Patients who were treated with nocturnal PAP before surgery should resume this treatment right after surgery, in the recovery room if possible.

Postoperative Monitoring: Although the association of OSA with postoperative pulmonary complications is well documented (18, 39, e5), there are as yet no evidence-based recommendations concerning the intensity and duration of postoperative monitoring. The ASA guidelines imprecisely state that OSA patients with an elevated risk of pulmonary complications should continue to be monitored after leaving

TABLE 2

The preoperative evaluation of patients with possible obstructive sleep apnea

Past medical history / documented conditions	History from patient and bed partner	Physical findings
history of “difficult airway”	loud snoring	“suspect” airway, Mallampati score 3 or 4*
arterial hypertension	witnessed periods of apnea	neck circumference: >43 cm in men >40 cm in women
other cardiovascular diseases	repeated nocturnal awakening	tonsillar enlargement
prior evaluation in a sleep laboratory	marked daytime sleepiness	obesity, particularly BMI >35 kg/m ²
metabolic syndrome	Headache on awakening in the morning	

adapted from (24)

* The Mallampati score (e39) is an estimate of the difficulty of endotracheal intubation based on the visibility of pharyngeal structures with the mouth open and the tongue protruded when the patient is sitting upright (Grade 1: full visibility of soft palate, uvula, and lateral palatal arches; intermediate grades to Grade 4: only the hard palate is visible).

BMI, body-mass index.

TABLE 3

The STOP and STOP-BANG questionnaires and the items they contain

Snoring	Do you snore loudly (loudly enough to be heard through closed doors at night)?
Tiredness	Do you often feel tired or sleepy during the day?
Observed apnea	Has anyone observed you to stop breathing, choke, or gasp in your sleep?
Pressure	Do you have high blood pressure, or are you being treated for it?
BMI	Is the body-mass index over 35 kg/m ² ?
Age	Is the patient over 50 years old?
Neck circumference	Is the patient's neck circumference >43 cm (men) or >40 cm (women)?
Gender	Is the patient male?

adapted from (25, 26) BMI, body-mass index; BANG, “body-mass index, age, neck circumference, gender”; STOP, “snoring, tiredness, observed apnea, high blood pressure.”

the recovery room. Monitoring can take place on an intermediate care unit, by telemetry on a normal inpatient ward, or by means of an observer assigned to keep watch over the patient in the hospital room. It remains unclear how OSA patients who are at an elevated risk of pulmonary complications are supposed to be identified, how they should be monitored and for how long, and whether the phenomenon of REM rebound (mentioned above) plays any role in the matter. In practice, patient safety must be weighed against the need for economically sensible mobilization of sparse monitoring resources. One possible algorithm for the postoperative monitoring of patients

TABLE 4

A proposed algorithm for the postoperative monitoring of patients with obstructive sleep apnea

Mode of monitoring / Type of postanesthetic management	Normal ward without monitoring	Normal ward with telemetric monitoring	Intensive care unit
CPAP after anesthesia (yes)			
– opioids (no)	acceptable	desirable	not necessary
– opioids (yes)	not recommended	acceptable	desirable
CPAP after anesthesia (no)			
– opioids (no) – oxygen (yes)	acceptable	desirable	desirable
– opioids (no) – oxygen (no)	not recommended	not recommended	desirable
– opioids (yes)	not recommended	not recommended	desirable

adapted from (34)

CPAP yes/no: Is it possible for the patient to be treated with continuous positive airway pressure (CPAP) after anesthesia?

with OSA, based on expert opinion, is shown in *Table 4* (34). Patients with OSA who are unproblematically extubated after surgery generally do not need to be monitored in an intensive care unit thereafter merely because they have OSA. Innovative methods of telemetric monitoring outside the intensive care unit, e.g., via WLAN, will probably be increasingly used in the near future.

Outpatient surgery for patients with obstructive sleep apnea

Patients with OSA can generally undergo surgical procedures on an outpatient basis, except those who have moderate to severe OSA and are not under treatment with PAP, and those with poorly controlled comorbid conditions (24, 40). Surgery on the airways themselves should not be performed on an ambulatory basis in patients with OSA. For outpatients whose preoperative screening arouses suspicion of previously undiagnosed OSA, the guidelines recommend that opioids should not be used for postoperative analgesia (24, 40), or, at least, that the necessary opioid dose should be meticulously titrated.

Research prospects in perioperative care

Clarification is needed with respect to the proper mode of valid preoperative short-term assessment of patients with suspected OSA. The proper role of miniaturized screening devices for OSA is unclear. More evidence is also needed for any definitive recommendation whether patients with suspected moderate to severe OSA should undergo polygraphy or polysomnography preoperatively. Furthermore, it remains to be determined whether all patients in whom OSA is identified before surgery must be treated with PAP, and, if so, with what modality of PAP. The appropriate use of telemedicine for the postoperative monitoring of patients with OSA, or a high risk for OSA, is unclear as well. Finally, we still lack a valid conception of the technical and logistical hospital infrastructure that will be needed to address all of these issues.

KEY MESSAGES

- Obstructive sleep apnea (OSA) is an important risk factor in perioperative medicine.
- OSA is associated with a wide variety of perioperative complications.
- The special situations and complications associated with OSA require expertise and meticulous care from the anesthesiologist in the perioperative setting.
- No recommendations for the anesthesiological treatment of OSA patients can yet be given on the basis of evidence from randomized trials.
- Patients with OSA do not generally need to be monitored postoperatively in an intensive care unit.

Conflict of interest statement

Prof. Eikermann owns stock in Calabash Bioscience Incorporated as well as patents for the use of acyclic curcubituril (also known as calabadiene) to reverse pharmacological muscle relaxation. He has received third-party research funding from Merck.

Prof. Teschler has served as a paid consultant for the ResMed company, from which he has also received lecture honoraria, third-party research funding, and reimbursement of meeting participation fees and travel expenses.

Dr. Fassbender, Dr. Herbstreit, and Prof. Peters state that they have no conflict of interest.

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Supplementary material
 For eReferences please refer to:
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Obstructive Sleep Apnea – A Perioperative Risk Factor

by Philipp Fassbender, Frank Herbstreit, Matthias Eikermann, Helmut Teschler, and Jürgen Peters

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eTABLE

Perioperative risk estimation in patients with obstructive sleep apnea

Variable		Points
A	OSAS severity*	
	no OSAS	0
	mild (AHI 5–15)	1
	moderate (AHI 15–30)	2
	severe (AHI >30)	3
B	Invasiveness of surgical procedure/anesthesia	
	superficial procedure under local anesthesia or peripheral nerve blockade, without sedation	0
	superficial procedure under moderate sedation or general anesthesia	1
	peripheral procedure under spinal or epidural anesthesia (with or without moderate sedation)	1
	peripheral procedure under general anesthesia	2
	procedure involving the airways under moderate sedation	2
	major surgery under general anesthesia	3
	procedure involving the airways under general anesthesia	3
C	Postoperative opioid requirement	
	none	0
	low-dosed oral opioids	1
	high-dosed oral, parenteral, or neuraxial opioids	3
D	Perioperative risk estimation A score plus B or C score, whichever is higher	
	moderate perioperative risk due to OSA	4
	marked perioperative risk due to OSA	5–6

AHI, apnea-hypopnea index; OSA, obstructive sleep apnea; OSAS, obstructive sleep apnea syndrome; PAP, positive airway pressure

* if PAP therapy has already been initiated and is to be continued postoperatively, −1 point; PaCO₂ >50 mm Hg in moderate or severe OSA, +1 point; adapted from (24)