Adverse outcomes have been reported in the perioperative setting in patients with known or unrecognized obstructive sleep apnea syndrome (OSAS). Although epidemiologic data report a prevalence of OSAS at about 5%,1 patients presenting to surgery have an estimated prevalence of 1–9%, or even higher in certain surgical categories.2 Ashton et al. studied 1487 men older than 40 years undergoing non-cardiac surgery for risk of perioperative myocardial infarctions (MI) and surprisingly did not report a single case of obstructive sleep apnea. Assuming the lowest reported prevalence of 1%, at least 15 patients in this large series of patients could have had OSAS prior to the surgical intervention.3 This leaves open the possibility that some of these patients who had perioperative MIs had unrecognized OSAS. In a cross-sectional study of 170 consecutive patients presenting for bariatric surgery the prevalence of OSA in the severely obese group (BMI 35–39.9) was 83.3% and 73.6% of patients in the morbidly obese (BMI 40–49.9%) category.4 Another study evaluating 40 consecutive patients for bariatric surgery reported an obstructive sleep-related breathing disorder in 88% by polysomnography, 71% had OSA and majority of the patients were women with mean BMI of 47kg/m2.5

Sleep studies in patients undergoing major abdominal surgery and cardiac surgery have shown suppression of rapid eye movement (REM) and slow wave sleep (SWS) after surgery.6,7,8,9 The REM sleep returns or rebounds in the late postoperative period (when oxygen may have been discontinued) and has been linked to significant respiratory abnormalities in a group of elderly patients who underwent abdominal vascular surgery.10 In REM sleep, the neural drive to the pharyngeal muscles is at a minimum and the atonia of antigravity muscles predisposes to airway instability causing episodic hypoxemia.11 Reduction in REM sleep, SWS and the lack of inherent rhythmicity are more pronounced after major surgery than after minor surgery even and less after laparoscopic surgery.9 Sedatives or analgesics, as well as the residual effects of anesthetic agents may worsen OSAS by decreasing pharyngeal tone and thereby increase upper airway resistance as well as attenuate the ventilatory and arousal responses to hypoxia, hypercarbia and obstruction.12

There is a limited amount of data that has focused specifically on OSAS and its impact on postoperative outcomes. In patients undergoing hip and knee replacement, up to one-third of those with OSAS developed substantial respiratory or cardiac complications including arrhythmias, myocardial ischemia, unplanned ICU transfers and reintubation.13 In another small prospective study evaluating the incidence of arrhythmia in patients with OSAS undergoing coronary artery bypass surgery, those with an oxygen desaturation index (ODI-defined as the number of desaturations ≥ 4% per hour) ≥ 5 had a relative risk of 2.8 for the development of atrial fibrillation postoperatively.14 In our own recent retrospective series of 25,587 patients that underwent cardiac surgery, 37 were confirmed to have sleep apnea by polysomnography.15 Higher incidence of encephalopathy (p=0.008), postoperative infection (0.028) and increased ICU length of stay (p=0.031) were noted in the group with OSAS after cardiac surgery. The difference in the rates of infection was mostly accounted for by the presence of mediastinitis (8.1% vs 1.6%). Differences in the rates of reintubation, tube time, and overall postoperative morbidity were not statistically significant.

Challenges in addressing the relative impact of OSAS on perioperative outcomes begin often times with the difficulty in diagnosing OSAS in the first place. The symptomatology of sleep apnea may be difficult to distinguish from normal variations in sleep behavior. Clinical examination at best carries a diagnostic sensitivity and specificity of only 50–60% for sleep apnea, when performed by experienced sleep physicians.16 Physical examination may reveal characteristic stigmata of OSAS including short thick neck, nasal obstruction, tonsillar hypertrophy, retrognathia and obesity. The degree of difficulty in visualizing the faucial pillars, soft palate and the base of the uvula can predict difficulty with intubation and should increase the suspicion of OSAS.17 In patients with these findings and history of daytime somnolence, snoring, and observed apneas, a presumptive diagnosis of OSAS can be made in the absence of a sleep study. Since the severity of these historical items correlates with the severity of sleep study-proven OSAS, use of a simple screening questionnaire for OSAS appears reasonable. However, none have so far been validated for use in the preoperative setting. Some studies suggest routine overnight polysomnography in all patients undergoing bariatric surgery regardless of BMI.5 Clinical suspicion for sleep apnea may also arise intra-operatively in some patients. Airway obstruction out of proportion to the apparent degree of sedation, pronounced tendency for upper airway obstruction during or upon recovery from anesthesia can suggest sleep apnea that has not been recognized perioperatively.18

Data guiding perioperative management of patients with known sleep apnea or those suspected of having this condition is limited. Increased awareness for close monitoring of high risk patients is recommended. For patients with OSAS having abdominal or other major surgery, significant expected pain or opioid requirement, severe OSAS at baseline needing CPAP at home, or with observed obstruction or episodic desaturations evident in recovery room continued inpatient moni-
toring is advised after the patient is moved out of PACU. 19
Routine ICU admission after surgery may not be necessary except in patients with co-existing cardiopulmonary disease or difficult airway. Patients at increased perioperative risk from OSAS should be extubated while awake and after full reversal of neuromuscular blockade is verified. Benzodiazepines should be avoided altogether and narcotics limited. Alternative forms of analgesia, such as non-steroidal anti-inflammatory medications, nerve blocks or local analgesics should be considered. If narcotics are required for pain control, patients should be in a monitored setting. Patient controlled analgesia with no basal rate may help limit dosing. 20 General anesthesia with a secure airway is preferable to deep sedation without a secure airway, particularly for procedures that may mechanically compromise the airway. Respiratory arrest has been reported in those with OSAS receiving epidural opioids at 2 to 3 days postoperatively. 21 If neuraxial analgesia is planned, local anesthetics alone should be preferred over opioids in combination. Case series and limited data suggest that use of CPAP in the perioperative setting for known cases of OSAS may help reduce postoperative complications. Until additional information is available to guide decision making, screening for OSAS should be incorporated as part of the preoperative assessment of patients subjected to surgery.

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References