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Anaesthetic management of sleep-disordered breathing in adults

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ABSTRACT

Anaesthesia and sleep are different states of unconsciousness with considerable physiological common ground. Because of their shared depressant effects on muscle activation and ventilatory drive, patients with anatomically compromised airways will tend to obstruct in either state and those with impaired ventilatory capacity will tend to hypoventilate. Breathing behaviour in one state is predictive of that in the other. An essential difference is that while arousal responses are preserved during sleep, they are depressed during sedation and abolished by anaesthesia. This renders patients with sleep-related breathing disorders vulnerable to hypoventilation and asphyxia when deeply sedated. Addressing this vulnerability requires a systematic approach to identification of patients and circumstances that magnify this risk, and methods of managing it that seek to reconcile the need for safety with cost-effective use of resources.

Key words: anaesthesia, obstructive sleep apnoea, perioperative management, sleep, sleep-disordered breathing.

Abbreviations: AHI, apnoea–hypopnoea index; BMI, body mass index; BPAP, bi-level PAP; CPAP, continuous PAP; GABA, gamma-aminobutyric acid; ICU, intensive care unit; OSA, obstructive sleep apnoea; PACU, post-anaesthesia care unit; PAP, positive airway pressure; Pcrit, critical closing pressure; REM, rapid eye movement; SDB, sleep-disordered breathing; STOP-BANG, loud *S*noring, daytime *T*iredness, *O*bserved obstructions during sleep, presence of high blood *P*ressure, *B*MI of >35 kg/m^2, *A*ge over 50 years, *N*eck circumference >40 cm and *M*ale gender; UA, upper airway.

INTRODUCTION

Preservation of upper airway (UA) patency is fundamental to survival. It is for good reason that in the 'ABC' of resuscitation 'A' for airway comes first, because without it, the 'B' for breathing will not happen and the 'C' for circulation becomes irrelevant.[1] Airway patency is challenged by unconsciousness in all its forms—sleep, sedation, anaesthesia and neurological injury. The first requirement of general anaesthesia is to ensure that a stable airway is established and maintained. This can be a major challenge in some individuals because of anatomical or physiological predisposition to UA collapse when unconscious. This vulnerability is often evident during sleep in such individuals as the associated muscle relaxation provides the conditions for partial or complete obstruction of the predisposed airway, usually necessitating a brief arousal to terminate it. This is the basis of obstructive sleep apnoea (OSA).

The anatomical factors that predispose to OSA are those that also predispose to difficulties with airway maintenance (either with face mask ventilation or with tracheal intubation) under anaesthesia.[2,3] These factors include a crowded narrow, oropharyngeal airway or restricted skeletal confines, as seen with retrognathia.[4,5] Hence, knowledge of the presence of OSA can help forewarn the anaesthesiologist of the possibility of a 'difficult airway'. Furthermore, the presence of OSA forewarns of vulnerability to UA obstruction beyond the heavily supervised environment of the operating room, with increased risk of post-operative asphyxia, respiratory arrest and death.[6–8] In the post-operative period, the risk is particularly exacerbated when under the influence of sedatives and opioid analgesics.[9,10] These medications depress the arousal responses that protect against prolonged episodes of partial or complete obstruction during sleep.[11,12] The severity of OSA as measured by the apnoea–hypopnoea index (AHI) may worsen in OSA patients during the post-operative period. It is well established that surgical patients with OSA are at increased risk of post-operative complications. Apart from their immediate consequences, these events have medico-legal implications.[13]

It is likely that the presence of other forms of sleep-disordered breathing (SDB), such as sleep hypoventilation, also helps identify patients at increased risk of perioperative respiratory complications. For example, patients with obesity hypoventilation syndrome appear

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to be at increased risk of post-operative respiratory failure.[14,15] Hypoventilation during sleep from other causes, such as respiratory neuromuscular disorders or advanced chronic obstructive lung disease, may also indicate a vulnerability to (wakeful) respiratory failure post-operatively in circumstances where ventilation is further compromised by pain, sedatives, opioid analgesics, atelectasis, respiratory infection or where there is an increased requirement for it from fever or disordered gas exchange.[16] While this paper primarily considers OSA and anaesthesia, sleep hypoventilation is another sleep-related breathing disorder which is likely to have significant implications for perioperative management. Conditions that commonly predispose to sleep hypoventilation, such as obesity and neuromuscular disease, also predispose to OSA and so the problems can frequently coexist.

THE NATURE OF OSA

OSA is characterized by recurrent episodes of partial (hypopnoea) or complete (apnoea) obstruction of the UA during sleep. The number of apnoeas and hypopnoeas divided by hours asleep yields the AHI, an index of OSA severity, with (in adults) ≤5 events/h considered normal, 5–14.99 events/h mild disease, 15–29.99 events/h moderate disease and ≥30 events/h severe disease.

The pathophysiology of OSA is based on a number of 'phenotypic' factors that vary between individuals, accounting for the different disease patterns observed amongst them. These factors include predisposing anatomical characteristics, patterns of UA muscle activation, arousal thresholds and stability of ventilatory control.[17] It is likely that these factors influence individual vulnerability of OSA patients to opioids and sedatives.

The apnoeic and hypopnoeic events occur because of the combined influences of an airway which is anatomically predisposed to obstruction and loss of the stabilizing effects of wakeful UA muscle activity with transition to unconsciousness.[18] Narrow, crowded airways through genetic predisposition, obesity, restricted skeletal confines or obstructing pathologies such as tonsillar or adenoidal hypertrophy all predispose to the problem. When a hypopnoea or apnoea occurs, it is usually terminated by a brief (<15 s) arousal, which is accompanied by muscle activation and momentarily disrupts sleep. Sometimes, these events cause awakenings. Occasionally, they resolve without cortical arousal. The threshold for arousal varies between individuals—some with a low threshold having a propensity to arouse with only minor degrees of obstruction, while others with a high arousal threshold have events that are longer and associated with greater degrees of hypoxaemia.[11,12]

The nature of the ventilatory response that follows relief of obstruction is a further individual phenotypic characteristic that predicates the subsequent course of events.[19] Those with exaggerated ventilatory responses following a period of obstruction—either because of relative brisk chemoreceptor/mechanoreceptor-mediated ventilatory responsiveness (high controller gain) or under-damped responses, for example through low oxygen or carbon dioxide stores which reduces the capacity to buffer changes in blood gas tensions with changes in ventilation (high plant gain)—are prone to increased breathing instability and repetition of events. The combined influences of controller gain and plant gain are sometimes collectively referred to as 'loop gain'.[17,18]

In its milder forms, OSA may only be manifest in particular circumstances, such in rapid eye movement (REM) sleep, where muscle relaxation is most profound, or where other aggravating factors are present, such as sleep in the supine posture, or after alcohol.[20]

Hence, it is the combined influence of a number of factors that determines the nature and severity of OSA. These are of relevance to perioperative management. The anatomical factors that predispose to OSA also predispose to difficulties with airway maintenance when sedated or anaesthetized, including difficulties with tracheal intubation.[2] The decrease in muscle activation and ventilatory drive that occur during sleep are also seen during sedation and anaesthesia, so that those with a tendency to obstruct or hypoventilate in one state are prone to do so in the other. Those with high arousal thresholds, manifest as lengthier obstructive events, with consequently deeper arterial oxygen desaturations, are likely to be more vulnerable to the depressant effects on arousal responses of sedative drugs, including opioid analgesics.[11]

ANATOMICALLY DIFFICULT AIRWAYS

The American Society of Anesthesiologists taskforce has defined a difficult airway as the clinical situation in which a conventionally trained anaesthesiologist experiences difficulty with facemask ventilation of the UA, difficulty with tracheal intubation or both.[21] Anatomical predispositions to this situation include: oropharyngeal crowding from familial factors; macroglossia (e.g. from Down syndrome); tonsillar and adenoidal hypertrophy; other masses within the UA; obesity; increased neck circumference (not necessarily obesity related) and narrowed skeletal confines (e.g. from retrognathia).[5] These problems also predispose to OSA and oropharyngeal appearances as judged from reduction in width and increased Mallampati score have predictive capacity for both conditions.[2] Furthermore, the conditions are related: Patients who are difficult to intubate are at substantial risk of OSA and, conversely, OSA is a risk factor for difficult intubation.[2,22] In a case–control retrospective study of 253 patients, difficult intubation was found to occur eight times more often in OSA patients than controls.[23] A 44% prevalence of difficult intubation has been found in OSA patients undergoing ear, nose and throat surgery.[24] Patients with severe OSA (AHI >40) have been found to have a higher prevalence of difficult intubation.[22] Patients with OSA may be difficult to mask ventilate as well.[25,26]

PHYSIOLOGICAL EFFECTS SHARED BETWEEN SLEEP AND ANAESTHESIA

A number of physiological changes are shared by sleep and anaesthesia. These include reductions in: (i) hypoxic and hypercapnic ventilatory drive[27–29]; (ii) muscle activation (with preferential inhibition of UA vs respiratory pump muscles and increased

diaphragm dependence)[30–32]; (iii) cortical influences (with removal of volitional control and of the stimulatory effect of wakefulness); (iv) gain of UA pharyngeal and other load compensation reflexes[31,33] and (v) lung volume.[34,35] These physiological changes render UA vulnerable to collapse and predispose to hypoventilation. The fact that they are present in both states suggests a potential relationship in UA collapsibility and hypoventilation between them.

TENDENCIES TO UA COLLAPSE AND HYPOVENTILATION IN SLEEP AND ANAESTHESIA ARE RELATED

Indeed, it is the case that there is a direct relationship between increased UA collapsibility under general anaesthesia, as quantified by measurement of pharyngeal critical closing pressure (Pcrit), and presence and severity of OSA, as quantified by AHI.[36]

THE NARCOTIC SWITCH: ABRUPT TRANSITIONS WITH LOSS OF CONSCIOUSNESS

In airways that are anatomically predisposed to obstruction, UA muscle activity is vital in maintaining UA patency during wakefulness.[37] This activity decreases quite abruptly with transition to unconsciousness both at sleep onset and with induction of anaesthesia.[38,39]

The electroencephalographic hallmark of sleep onset is a relatively sudden transition in its frequency content from alpha to (slower) theta rhythm. At this transition, there is an abrupt diminution of phasic UA dilator muscle (genioglossus) activity that promptly reappears with return to alpha rhythm.[38] The abruptness of this change with transition to unconsciousness is consistent with the notion of a 'narcotic switch' in which the neural networks involved are organized to produce stable states of wakefulness or sleep with a distinct threshold effect observed in moving from one state to the other. To be either stably awake or stably asleep is important behaviourally so as to avoid inconvenient intrusions of sleep into wakefulness or wakefulness into sleep. Structurally, this narcotic switch consists of a network of neural centres in the hypothalamus and brain stem, some of which are stimulatory in nature, generating ascending activating stimuli to the cortex to maintain wakefulness, and others which are inhibitory, suppressing this ascending arousal system during sleep.[40] During wakefulness, the activity of the stimulatory centres predominates and, furthermore, inhibits the activity of the inhibitory, sleep-promoting centres. During sleep, the reverse applies. It is this mutual inhibition between stimulatory and inhibitory centres that is the basis for the relative stability of either wakefulness or sleep with a well-defined switch-like transition between them.

The neurophysiology of wakefulness and sleep is highly relevant to anaesthesia as key components of the narcotic switch are activated by anaesthetic and sedative drugs. It is important to note that the unconsciousness of anaesthesia is due, at least in part, to activation of this switch. For example, the hypothalamic ventrolateral preoptic nucleus, which is the foremost inhibitory centre, is replete with GABA (gamma-aminobutyric acid)-ergic neurons. Its neuronal targets are activated by common anaesthetics, such as propofol and inhalational anaesthetics, and sedatives, such the benzodiazepines, as they are GABA-ergic in their activity.[40] Alternatively, rather than activating inhibitory centres, dexmedetomidine, which is an alpha 2 agonist, produces its sedative effects by inhibiting the locus coeruleus, an important wakefulness promoting centre which is noradrenergic in its activity.[40] It is perhaps not surprising, therefore, that when these wake-sleep centres are activated by anaesthetic and sedative drugs, a similar sharp switch-like decrease in pharyngeal muscle activation is observed during induction of anaesthesia at transition from consciousness to unconsciousness to that observed at sleep onset.[38,39]

While this transition from consciousness (with all its protections) to unconsciousness (with all its associated vulnerabilities to UA obstruction and hypoventilation) occurs by similar neurophysiological means with sleep and anaesthesia, an obvious and critical difference exists between them: transition back to consciousness is readily made from natural or drug-induced sleep (mild sedation), as arousal responses are preserved, whereas arousal responses are suppressed during deep sedation and anaesthesia and drug metabolism, and elimination is required for the return of consciousness.

CAPACITY TO AROUSE IS THE CRITICAL DIFFERENCE BETWEEN SLEEP/MILD SEDATION AND DEEP SEDATION/ANAESTHESIA

The capacity to arouse is a vital protection against harm in the sleeping individual. In general, obstructive events and periods of hypoventilation are terminated by arousals which are triggered by changes in mechanical load or in blood gas tensions. Suppression of arousal responses is associated with danger of asphyxia, cardiopulmonary arrest and death in those prone to UA obstruction when unconscious.[11] These vulnerabilities are adequately managed during procedural sedation and anaesthesia by a vigilant attending anaesthesiologist. Close monitoring is required until there is an assured return of arousal responses following emergence from anaesthesia. Danger of perioperative asphyxia recedes with the return of rousability. However, subsequent use of sedatives or opioids for postoperative pain may induce a recurrent impairment of protective arousal responses. This may well explain why OSA is an established risk factor for cardiopulmonary complications during in-patient surgery, but appears to be associated with relatively low risk beyond initial emergence in ambulatory surgery.[41,42] However, while the requirement for opioids for postoperative pain may be less following ambulatory surgery, there are reported cases of respiratory arrest related to use of opioids in this setting. Assuring return of prompt, stable protective arousal responses is a key consideration in anaesthetic management of patients with SDB. Until the risk of recurrent arousal

suppression (e.g. with subsequent opioid or sedative use) has receded, monitoring of ventilatory adequacy (oximetry, oro-nasal airflow by capnography or other means) is recommended.[43]

OPIOIDS AND THE AT-RISK PATIENT

Opioids have a central role in post-operative pain management for many procedures. In sufficient dose, they pose particular problems for ventilation in patients with SDB. These problems arise because they acutely depress: (i) hypercapnic and hypoxic responses[44]; (ii) pharyngeal negative pressure and other load compensating reflexes[45]; (iii) arousal and awakening responses[46]; (iv) volitional modulation of respiration[47] and (v) pharyngeal dilator muscle activity.[48]

These opioid-induced changes are potentially dangerous in predisposed patients as they can provoke UA obstruction, hypoventilation and arousal failure. These effects may be aggravated by coexistent sleep, hypercapnia and use of sedatives. Multimodal analgesia and regional anaesthesia should be used whenever possible. However, while an admirable aim, circumvention by minimizing use of opioid drugs preoperatively is not always practicable. Where not, the vulnerable patient should be continuously monitored until the need for such drugs has passed or, where there is continued, non-parenteral use in lower dose, stable return of prompt rousability has been demonstrated.

OSA AS A PERIOPERATIVE RISK FACTOR

It is likely that the patients most at risk from perioperative ventilatory complications are those who have a low tolerance to the sedative effects of these drugs and/or who have an inherently high threshold to arousal from obstructive events, as evident from their behaviour during sleep.[11] Just as there are inter-individual difference in drug sensitivities, so are there inter-individual differences in thresholds to arousal from sleep triggered by UA obstructive episodes: some arouse very readily to relatively minor degree of obstruction and changes in blood gas tensions while others can have prolonged apnoeas with deep desaturations before the eventual occurrence of a life-saving arousal response.[11,49] In such cases, these lengthy events are most often observed during REM sleep, as arousal responses can be at their most sluggish in this sleep stage. A particular subset of patients with OSA, characterized by decreased chemoreflex responsiveness and high arousal thresholds, may be more susceptible to opioid-induced ventilatory impairment.[49]

STRATIFYING PERIOPERATIVE RISK

Hence, a variety of factors potentially contribute to perioperative risk associated with OSA and attempts to stratify this risk should take into account the procedure undertaken, relevant patient characteristics and post-operative sedative and opioid analgesic requirements.

Procedures associated with extra risk are likely to include operations involving the UA, brain, chest wall/respiratory pump (thoracic and upper abdominal) and/or those associated with a substantial post-operative analgesic requirement. *Patient factors* likely to be important include: (i) severity of OSA, as reflected by both AHI and presence of lengthy events and deep desaturations[12,50]; (ii) associated obesity, neuromuscular disease or advanced lung disease, all of which can predispose to both UA obstruction and sleep hypoventilation[16]; (iii) the presence of sleep hypoventilation either demonstrated by direct observation (sleep study) or inferred from otherwise unexplained elevation of wakeful serum bicarbonate levels (which suggests buffering of overnight CO_2 accumulation)[51] or (iv) the presence of (wakeful) respiratory failure as documented by arterial blood gas analysis. Any *post-operative opioid analgesic or sedative requirement* is potentially problematic but likely to be particularly so where there is a requirement for higher doses, as defined by parenteral use, oral use >200 mg morphine equivalents/day in the case of chronic use, or less in patients naïve to opioids or with particular sensitivities to their effects.[52]

MANAGING PERIOPERATIVE RISK

The most widely promulgated guideline for management of perioperative risks associated with OSA is the 'Practice Guidelines for the Perioperative Management of Patients with Obstructive Sleep Apnea' published by the American Society of Anesthesiologists in 2014.[53] This provides much sound advice, but is constrained by a relative paucity of published data on which to base its recommendations. As a result, the guideline provides little practical advice as to how to balance notions of best practice with effective and cost-effectiveness use of expensive monitoring resources. Anaesthesiologists remain faced with the difficulty of identifying those most at risk of perioperative ventilatory complications for particular attention including prolonged use of expensive monitoring facilities post-operatively: a process of risk stratification.

A more recent guideline, published by the Society of Anesthesia and Sleep Medicine in 2016 ('Society of Anesthesia and Sleep Medicine Guidelines on Preoperative Screening and Assessment of Adult Patients with Obstructive Sleep Apnea') addresses this issue.[54] Among other matters, it recognizes that identification of patients at risk may occur late in preoperative work-up. In most cases, this is too late and too imprecise to reasonably delay surgery. Hence, the emphasis is on how to best assess risk within the limited time frame available, and manage thereafter.

GENERAL CONSIDERATIONS IN PERIOPERATIVE MANAGEMENT OF PATIENTS WITH KNOWN OR SUSPECTED OSA

Many surgical patients with OSA are undiagnosed.[55,56] Of those who are diagnosed, some are fully compliant with treatment while others are either partially treated

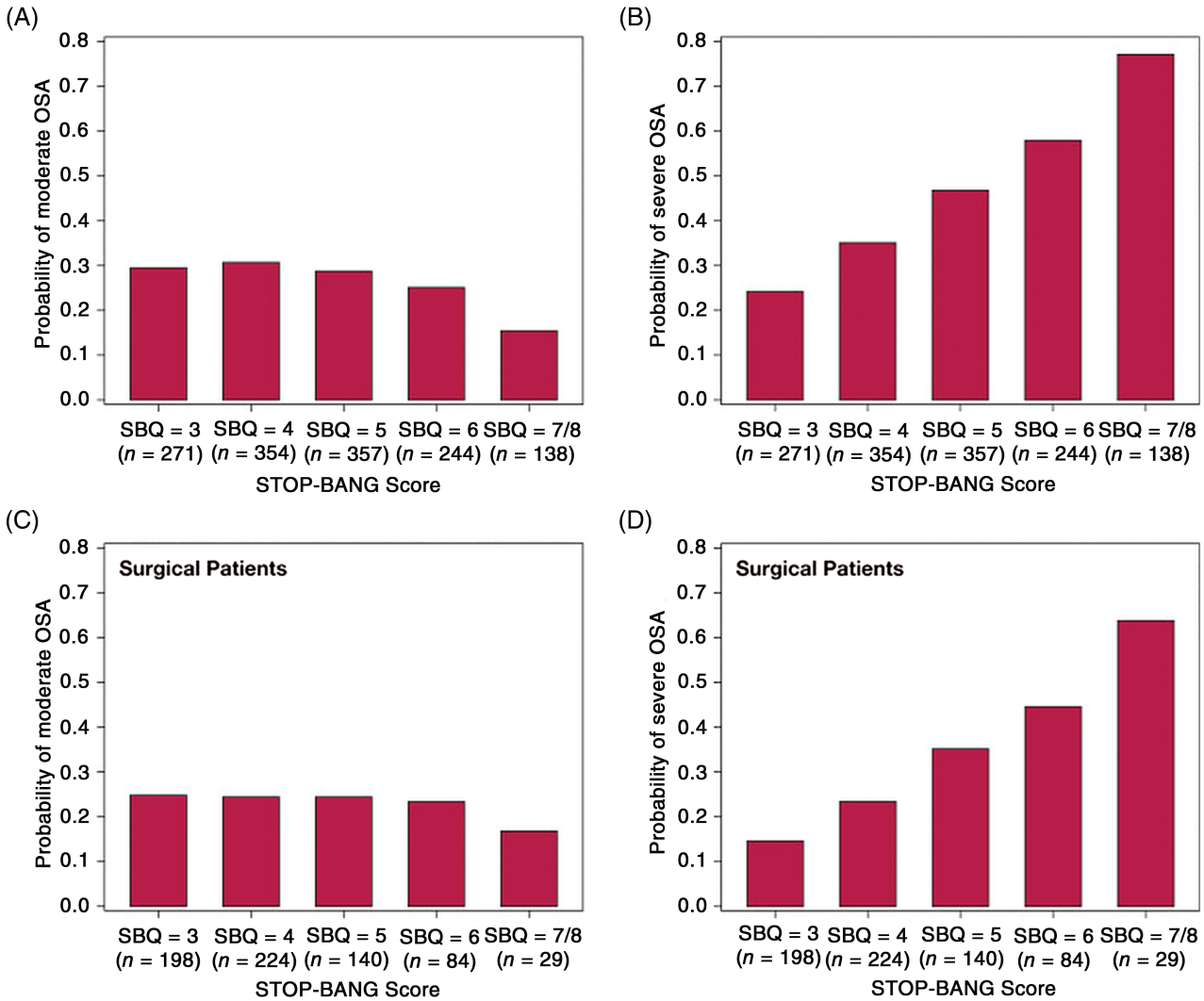

Figure 1 The relationship between the STOP-BANG (loud *S*noring, daytime *T*iredness, *O*bserved obstructions during sleep, presence of high blood *P*ressure, *B*MI of >35 kg/m^2, *A*ge over 50 years, *N*eck circumference >40 cm and *M*ale gender) Questionnaire score and probability of obstructive sleep apnoea (OSA). (A) The STOP-BANG Questionnaire score and the probability of moderate OSA (apnoea–hypopnoea index (AHI): >15–30) in sleep clinic patients. (B) The STOP-BANG Questionnaire score and the probability of severe OSA (AHI >30) in sleep clinic patients. (C) The STOP-BANG Questionnaire score and the probability of moderate OSA (AHI: >15–30) in surgical patients. (D) The STOP-BANG Questionnaire score and the probability of severe OSA (AHI >30) in surgical patients (Adapted from Chung *et al.*,[64] with permission).

or untreated. Perioperative management strategies need to take this into account.[53] General considerations include: (i) preoperative assessment for OSA with an attempt to identify and stratify risk and to initiate therapy where feasible in patients not already under treatment; (ii) intraoperative use of local or regional anaesthetic techniques where applicable to obviate the need for sedatives and parenteral opioids; (iii) selection of short-acting drugs with rapid metabolism and elimination for early return of consciousness where general anaesthesia is employed; (iv) extubation awake, with full reversal of neuromuscular blockade and (where practicable) non-supine; (v) further assessment of vulnerability to UA obstruction and hypoventilation in the post-anaesthesia care unit (PACU)[57]; (vi) provision to monitor oxygen saturation (SpO2) and ventilation continuously while risk of obstruction or hypoventilation persists (essentially while arousal responses remain subject to compromise); (vii) minimization of use of opioids and sedatives through use of regional analgesia and non-opioid analgesics, exercising particular caution with opioid infusions[58]; (viii) avoidance of the supine posture where possible and (ix) use of positive airway pressure (PAP) therapies where indicated and feasible.[59–62]

IDENTIFYING PATIENTS AT RISK

OSA is widely prevalent, particularly amongst surgical populations, and is under-diagnosed.[55,56] Hence, many patients will present for surgery with this risk factor unidentified. The first step in addressing this issue is to organize systematic collection of historical and anthropometric information that identifies this risk. There are

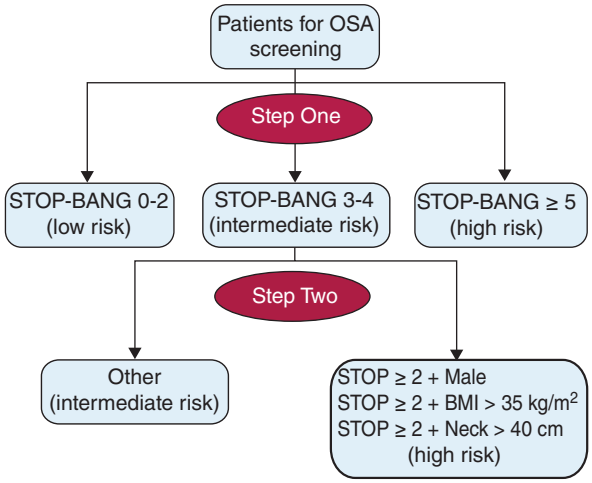

Figure 2 STOP-BANG (loud *S*noring, daytime *T*iredness, *O*bserved obstructions during sleep, presence of high blood *P*ressure, *B*MI of >35 kg/m^2, *A*ge over 50 years, *N*eck circumference >40 cm and *M*ale gender) algorithm with a two-step scoring strategy (Adapted from Chung *et al.*,[64] with permission).

a number of questionnaires designed to do this, the most widely used currently being the eponymous STOP-BANG Questionnaire.[63,64] This involves eight questions relating to loud *S*noring, daytime *T*iredness, *O*bserved obstructions during sleep, presence of high blood *P*ressure, *B*MI of >35 kg/m^2, *A*ge over 50 years, *N*eck circumference >40 cm and *M*ale gender. One point is scored for each positive answer and a score of ≥3 is 84% sensitive and 56% specific for the presence of OSA (AHI >5 events/h).[63] The high sensitivity, combined with its simplicity, makes it an attractive screening tool as a score of 0–2 allows OSA to be reasonably confidently excluded. However, the modest specificity means a high proportion of those ruled in (score ≥3) will be false positives. Other preoperative questionnaires such as the Perioperative Sleep Apnea Prediction Score, Berlin Questionnaire and American Society of Anesthesiologists OSA check-list share this problem of modest specificity.[65] In surgical populations, a higher STOP-BANG score is associated with a greater probability of moderate to severe OSA (Fig. 1).[66,67] Various combinations of STOP-BANG score components may be helpful in refining risk. For example, a combination of a STOP score ≥2 + BMI > 35 kg/m^2, or male gender or neck circumference >40 cm is more specific for the presence of OSA than a STOP-BANG score of ≥3 (Fig. 2).[64] The inverse relationship between sensitivity and specificity with varying STOP-BANG diagnostic thresholds influences the relative rates of missed diagnoses and wasted resource utilization in diagnosing OSA.[54] The optimal cut-off score of STOP-BANG should be determined in each specific setting: in practice, many preoperative clinics use STOP-BANG score of 5 or greater in an effort to improve specificity, but at the cost of sensitivity.[68]

In addition to their problems with diagnostic accuracy, it is important to note that the questionnaires are primarily designed to identify the presence of OSA rather than quantify its severity. Hence, other methods have to be considered in more accurately determining the presence *and* severity of OSA. If time allows and the level of suspicion is sufficiently high, then comprehensive polysomnography can be considered. However, there are other simpler portable monitoring alternatives that supply valuable additional information readily and relatively inexpensively.[69]

PORTABLE PREOPERATIVE MONITORING

The simplest portable monitors record various combinations of oximetry, nasal airflow and chest wall motion continuously overnight. Providing adequate sleep is obtained (which is not directly assessed by these simpler devices, in contrast to polysomnography) a reasonable estimate of the presence and severity of OSA can be obtained including estimated AHI, length of events and severity of associated arterial oxygen desaturations. The addition of this information substantially improves on the diagnostic accuracy of questionnaires with favourable combinations of sensitivity and specificity for the presence of OSA.[69] Furthermore, the additional information allows the severity of OSA to be better quantified.

Even though simpler and more readily available than polysomnography, time is still needed to arrange overnight monitoring, retrieve data and analyse the results. This is problematic where patients at potential risk are identified late in the preoperative work-up, as is often the case. The Society of Anesthesia and Sleep Medicine Guideline suggests that there is insufficient evidence to support cancelling or delaying surgery in order to undertake further testing to diagnose OSA in patients identified as being at high risk of it preoperatively, unless there is evidence of associated significant or uncontrolled systemic disease or additional problems with ventilation or gas exchange.[54] These problems include, but may not be limited to: (i) hypoventilation syndromes; (ii) severe pulmonary hypertension and (iii) resting hypoxaemia not attributable to other cardiopulmonary disease.[54] Where at-risk patients are identified, risk can be mitigated by following the measures described earlier, including modification of anaesthetic technique, multi-modal pain management, postoperative monitoring and PAP therapy where required.

THE VALUE OF FURTHER SYSTEMATIC OBSERVATION IN THE PACU

A valuable additional strategy to determine risk of post-operative ventilatory complications is to make systematic observations early post-operatively in the PACU, as described by Gali *et al.*[57] This takes advantage of the close and continuous monitoring facilities available in PACU to identify behaviours that indicate particular vulnerability to UA obstruction and/or hypoventilation in at-risk patients. Such patients are monitored for up to 90 min in this environment, so observations can be made well beyond immediate emergence from anaesthesia. The presence of

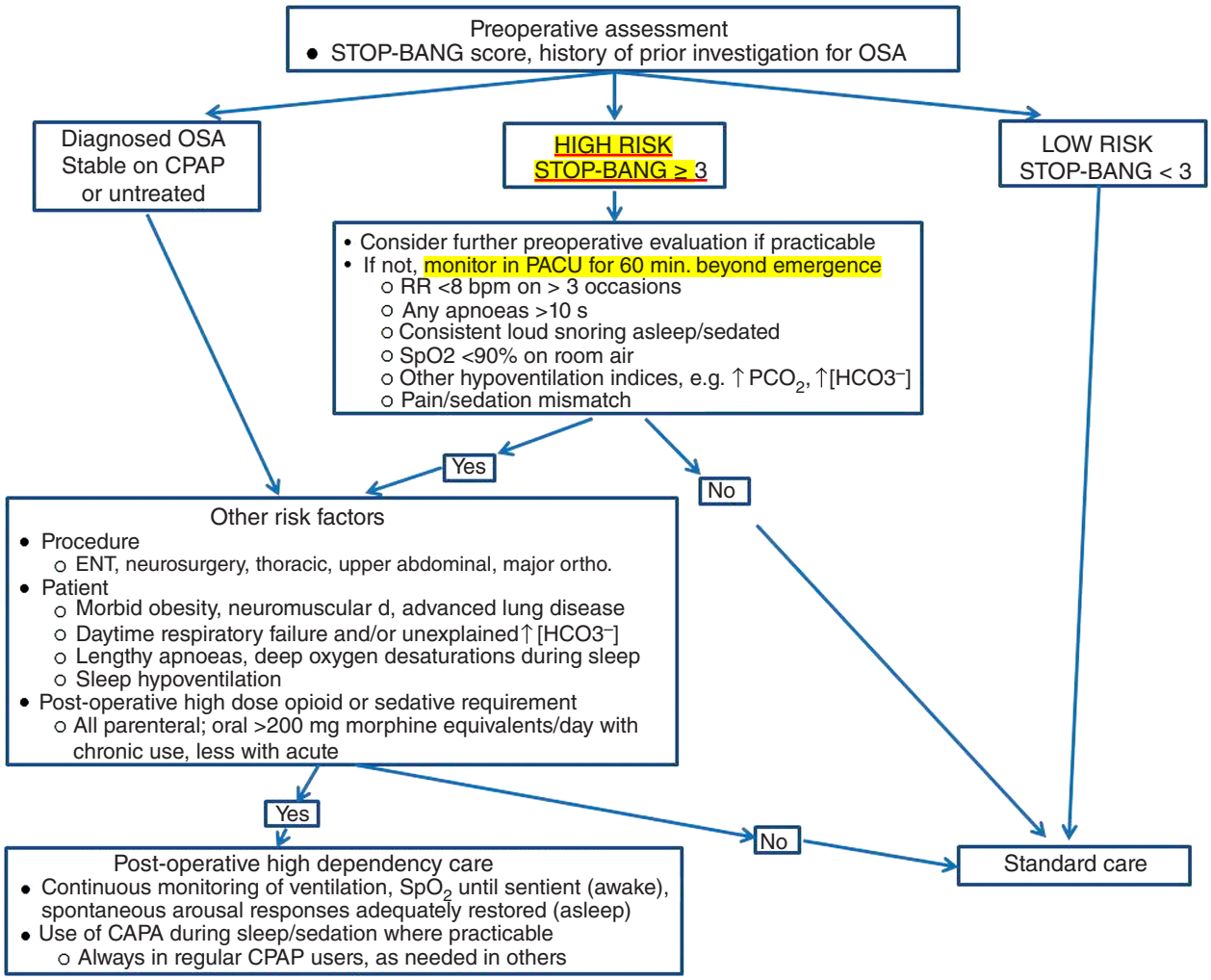

Figure 3 A flow chart incorporating principles for perioperative management of adult patients with sleep-disordered breathing.

recurrent bradypnoea; witnessed obstructive events; consistent loud snoring when asleep/sedated; persistent hypoxaemia (accounting for use of oxygen therapy); other indices of hypoventilation such as elevated bicarbonate levels or hypercapnia; and mismatch between complaints of pain and levels of sedation are factors that may highlight vulnerability to ventilatory problems beyond PACU. Patients with suspected OSA and the recurrent respiratory events in PACU were shown to have a greatly increased likelihood of post-operative respiratory complications.[57] Detection of these recurrent respiratory events demands consideration of further continuous oximetry and monitoring of ventilation on discharge from PACU until such time that the patient is sentient when awake and has adequate and stable restoration of arousal responses when asleep.

USE OF PAP THERAPIES POST-OPERATIVELY

There is a limited literature suggesting that PAP therapy may have a role in attenuating post-operative complications in patients with OSA, with a meta-analysis showing a trend towards shorter length of hospital stay (continuous PAP (CPAP) vs no CPAP: 4.0 ± 4 days vs 4.4 ± 8 days, $P = 0.05$).[62] Two recent large retrospective studies suggest potential efficacy of CPAP in reducing such complications in patients with diagnosed OSA.[50,59] In one of these studies, those diagnosed with OSA and who had CPAP therapy prescribed before their surgery had a significantly reduced risk for cardiovascular adverse events as compared with those undiagnosed OSA patients (OR = 0.34, 95% CI = 0.15–0.77, $P = 0.009$).[50] In the other studies, untreated OSA patients were found to have higher cardiopulmonary complication rates than those prescribed CPAP therapy (risk-adjusted rates 6.7% vs 4%; adjusted OR = 1.8, $P = 0.001$).[59] These studies provide some evidence to support preoperative diagnosis of OSA and initiation of CPAP therapy.

While the use of PAP therapies in patients with OSA or sleep hypoventilation when asleep or sedated is highly desirable, it must not be thought of as obviating the need for monitoring of such patients while they remain vulnerable to failure to arouse post-operatively: PAP therapies are not always used continuously and are subject to misapplication or other problems with

implementation, particularly for the time when the patient is not sentient.

For patients already on CPAP and compliant with it, perioperative use is generally straightforward and its deployment should be routine in the post-operative period. Where time allows preoperatively, an advantage of detailed work-up of previously undiagnosed patients is to familiarize them with CPAP where indicated, which will simplify its use post-operatively.[60,61]

Its use should certainly be considered where obstructive events or hypoventilation are observed post-operatively. In most circumstances, the form of PAP therapy used to treat OSA is CPAP whereas bi-level PAP (BPAP) is used where hypoventilation coexists. Successful implementation depends on experienced, well-motivated staff who understand the equipment involved, can apply it confidently and readily troubleshoot difficulties with its use. Where PAP therapies are not tolerated alternative, less efficacious strategies include positional therapy (e.g. lateral posture with upper body elevation) and renewed efforts to eliminate opioid/sedative use. Other strategies such as use of oral devices/airways remain to be explored in the perioperative arena, although oral appliances are second line therapy to PAP in the management of OSA at home.[70]

CONCLUSIONS

Figure 3 summarizes the principles for the perioperative management of adult patients with SDB discussed in this paper, in a flow chart format. This is based on current knowledge and much work remains to be done to test some of its propositions and develop better management methods. The fact that OSA is common and associated with increased risk of adverse events perioperatively, including occasional fatal outcomes, provides a strong motivation for doing so.[13] Some obvious areas for improvement include: (i) development of better perioperative risk stratification with wider use of portable monitoring; (ii) use of other metrics of OSA severity besides AHI (e.g. those indicating high arousal thresholds such as length of events and depth of associated arterial oxygen desaturations); (iii) closer regard for the possibility of coexistent sleep hypoventilation; (iv) development of cost-effective methods to continuously monitor ventilation outside high dependency areas such as PACU and intensive care unit (ICU), including adaptation of sleep study methods such as transduction of nasal pressure to derive nasal flow; (v) the use of telemetered data particularly for patients are not being nursed in a high dependency area and (vi) increased use of PAP therapies post-operatively.

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