WELCOME MESSAGE

On behalf of The Society of Anesthesia and Sleep Medicine (SASM) and The University of Chicago, we would like to welcome you to our **Annual Conference: OSA, Anesthesia and Sleep - the Common Ground.**

The SASM is an international society organized to advance standards of care for clinical problems shared by Anesthesiology and Sleep Medicine, including perioperative management of sleep disordered breathing, and to promote interdisciplinary communication, education and research in matters common to anesthesia and sleep. Consider joining us as a member of *SASM.*

We truly hope you enjoy The OSA, Anesthesia and Sleep - the Common Ground and will take time to meet our conference speakers, delegates and exhibitors.

Thank you for joining us; and we look forward to a rewarding and scientifically enriching conference.

Sincerely,
Conference Directors:
Frances Chung, MBBS
Babak Mokhlesi, MD

CONFERENCE DESCRIPTION

This conference, presented by The Society of Anesthesia and Sleep Medicine (SASM) and The University of Chicago Pritzker School of Medicine, has been developed as an educational opportunity to present and discuss the basic and more controversial areas of sleep apnea and anesthesia.

The objective of this meeting is to provide a forum for discussions pertaining to the common grounds between obstructive sleep apnea, sleep and anesthesia. The goal is to promote excellence in medical care, research and education in anesthesia, sleep medicine, and perioperative medicine.

LEARNING OBJECTIVES

Upon completion of this activity, participants will be able to:

- *Review the shared pathogenetic mechanisms of increased upper airway collapsibility during anesthesia and sleep;*
- *Interpret the neurophysiological correlates of loss of consciousness, unconsciousness and recovery of consciousness under general anesthesia;*
- *Determine the perioperative management in adenotonsillectomy in a child with OSA;*
- *Formulate how to implement screening for OSA in a Preoperative Clinic;*
- *Analyze the evidence of postoperative complications of OSA patients in medical literature;*
- *Determine which postoperative patients with OSA need monitoring;*
- *Formulate algorithms for the perioperative management of OSA patients;*
- *Appraise the impact of sleep apnea and episodic hypoxemia on ventilatory control and hemodynamics;*
- *Review the influence of opioids, anesthetics and their interaction on the control of breathing;*
- *Determine that recognition and treatment of obesity hypoventilation syndrome in the perioperative period can potentially reduce complications;*

TARGET AUDIENCE

The target audience includes anesthesiologists, critical care physicians, residents, fellows in-training, general medicine physicians, pulmonary physicians, sleep medicine physicians, surgeons, scientists and allied health care professionals.

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This activity has been planned and implemented in accordance with the Essential Areas and Policies of the Accreditation Council for Continuing Medical Education through the joint sponsorship of The University of Chicago Pritzker School of Medicine and The Society of Anesthesia and Sleep Medicine. The University of Chicago Pritzker School of Medicine is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

The University of Chicago Pritzker School of Medicine designates this live activity for a maximum of 6 *AMA PRA Category 1 Credit(s)*™. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

Nurses and other health professionals will receive a Certificate of Attendance. For information on applicability and acceptance, please consult your professional licensing board.

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Mission

The mission of SASM is to advance standards of care for clinical problems shared by Anesthesiology and Sleep Medicine, including peri-operative management of sleep disordered breathing, and to promote interdisciplinary communication, education and research in matters common to anesthesia and sleep.

Benefits of Membership in SASM include:

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- *Gives a forum to evaluate and discuss the latest research, education, and clinical practices pertaining to OSA and patients with other sleep disordered breathing.*
- *Get advice and counsel from other members regarding various practice paradigms.*

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Active Members:	Physician and Scientist members with the ability to vote, hold office, and serve as Directors of Board.	$100 per annum** **This fee is not required for Founding Members for the year 2011.
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3. Mail checks for membership to:

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c/o Dept of Anesthesia
2500 MetroHealth Drive
Cleveland, OH 44109

FRIDAY, OCTOBER 14, 2011

7:00 am	**REGISTRATION, CONTINENTAL BREAKFAST AND POSTER VIEWING**
7:30	**POSTER VIEWING** **Poster Session Facilitators:** *Dennis Auckley, Yandong Jiang, Ralph Lydic, Jean Mantz, Satya Krisna Ramachandran*
8:00	**WELCOME ADDRESS,** *Frances Chung, MBBS*
SESSION I	**Unconsciousness and the Upper Airway - Shared Considerations for Anesthesiology and Sleep Medicine,** Moderators: *Shiroh Isono, MD; Ralph Lydic, MD*
8:05	**KEYNOTE: Effects of Sleep and Anesthesia Induced Unconsciousness on Upper Airway Patency,** *David Hillman, MBBS*
8:55	**The Dynamics of Loss and Recovery of Consciousness Under General Anesthesia,** *Emery Brown, MD*
9:40	**Perioperative Management in Adenotonsillectomy in a Child With OSA,** *Karen Brown, MD*
10:10	**Question & Answer**
10:30	**REFRESHMENT BREAK AND POSTER VIEWING**
SESSION II	**Obstructive Sleep Apnea – A Perioperative Challenge,** Moderators: *Norman Bolden, MD; Dennis Auckley, MD*
11:00	**From Screening to Diagnosis of OSA Preoperatively:** *Chicago's Experience, Bobbie Jean Sweitzer, MD*
11:20	**Perioperative Complications in OSA Patients: Evidence-Based Review of the Literature,** *Frances Chung, MBBS*
11:40	**Who Are the Patients That Require Postoperative Monitoring?,** *Roop Kaw, MD*
12:00	**Clinical Response Pathways When the Monitor Alarms Go Off: Use of Positional Therapy, Oxygen, and Various PAP Devices,** *Peter Gay, MD*
12:20	**Question & Answer**
12:30	**LUNCH AND POSTER VIEWING**
1:00 – 1:30	**POSTER VIEWING** **Poster Session Facilitators:** *Yandong Jiang, John Loadsman, Shiroh Isono, Jeremy Weingarten, Max Kelz*
1:30	**Presentation of Poster Awards** - *Presenter: Yandong Jiang, MD*
SESSION III	**Sleep, Anesthesia and Ventilatory Control,** Moderators: *Matthias Eikermann, MD; Max Kelz, MD*
2:00	**Mechanisms of Recurrent Apnea,** *Kingman P. Stroh, MD*
2:35	**Influence of Opioids and Anesthetics on the Control of Breathing and Upper Airway Obstruction,** *Albert Dahan, MD*
3:10	**Unique Considerations of Patients with Obesity Hypoventilation Syndrome,** *Babak Mokhlesi, MD*
3:45	**Question & Answer**
3:45 - 4:00	**REFRESHMENT BREAK**
4:00	**ADJOURN**
4:30 - 5:30	**FIRST SASM ANNUAL MEETING**
5:15 - 6:15 pm	**RECEPTION with CASH BAR**

SPEAKER ABSTRACTS

TABLE OF CONTENTS

KEYNOTE:
Effects of Sleep and Anesthesia Induced Unconsciousness On Upper Airway Patency

David R Hillman, MBBS
Head, Department of Pulmonary Physiology,
Director, West Australian Sleep Disorders Research Institute
Sir Charles Gairdner Hospital, Perth, Australia

Sleep is a fundamental need with restorative functions and an important role in information processing, consolidation of memory and learning. Specific neural circuits are involved in the induction of sleep. The brain's sleep and arousal pathways exert inhibitory influences on each other, acting like a flip-flop switch to produce relatively stable states of sleep or wakefulness.[1] Hence a threshold exists that is traversed with induction of sleep. As this threshold is crossed a precipitate reduction in muscle activation occurs that predisposes the vulnerable upper airway to collapse.[2]

In recent years it has become increasingly apparent that activation of these same sleep pathways is a mechanism by which many anesthetic agents induce unconsciousness.[3] Similarly to sleep onset, anesthetic induction is associated with a profound reduction in upper airway muscle activity with precipitate changes occurring at the point at which consciousness is lost.[4] This loss of activation is accompanied by an increase in airway collapsibility that persists until consciousness is restored.

Understanding this threshold behavio r is likely to be a key to ensuring safe perioperative management of patients with obstructive sleep apnea. Vulnerability exists where drug-induced unconsciousness is present as a result both of loss of upper airway muscle activation and suppression of arousal responses. Risk is likely to be less where rousability is present, such as during conscious sedation, or once it is restored during emergence from anesthesia. This may explain the apparent low post-operative morbidity in OSA patients presenting for day surgery[5] yet more evident risk for those having major surgery,[6] where postoperative sedation and narcotic analgesia may re-induce unconsciousness and suppression of arousal, particularly if compounded by marked hypercapnia.

Perioperative morbidity needs to be examined with these factors in mind. This may allow the establishment of effective, cost-effective and safe perioperative management strategies based on more precise definition of the circumstances associated with particular risk of upper airway obstruction or hypoventilation.

References

1. Fuller PM et al. J Biol Rhythms 2006;21:482-93.
2. Worsnop C et al. J Appl Physiol 1998;85:908-20.
3. Brown EN et al. N Engl J Med 2010;363:2638-50
4. Hillman DR et al. Anesthesiology 2009; 111:63-71
5. Sabers C et al. Anesth Analg 2003;96:1328-35
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THE UNCONSCIOUS BRAIN UNDER GENERAL ANESTHESIA

Department of Brain and Cognitive Sciences
Harvard-MIT Division of Health-Sciences and Technology
Massachusetts Institute of Technology

Emery N. Brown, MD, PhD.
Department of Anesthesia, Critical Care and Pain Medicine
Massachusetts General Hospital
Harvard Medical School, Cambridge, MA

General anesthesia is a drug-induced, reversible condition comprised of five behavioral states: unconsciousness, amnesia (loss of memory), analgesia (loss of pain sensation), akinesia (immobility), and hemodynamic stability with control of the stress response. The mechanisms by which anesthetic drugs induce the state of general anesthesia are considered one of the biggest mysteries of modern medicine. We have been using three experimental paradigms to study general anesthesia-induced loss of consciousness in humans: combined fMRI/EEG recordings, high-density EEG recordings and intracranial recordings. These studies are allowing us to establish precise neurophysiological, neuroanatomical and behavioral correlates of unconsciousness under general anesthesia. We will discuss the relation between our findings and two other important altered states of arousal: sleep and coma. Our findings suggest that the state of general anesthesia is not as mysterious as currently believed.

OUTCOME, RISK, AND ERROR AND THE CHILD WITH OBSTRUCTIVE SLEEP APNEA

Karen A. Brown
Department of Pediatric Anesthesia
McGill University Health Centre/Montreal Children's Hospital
Montreal, QC, Canada

Summary

Adenotonsillectomy is the mainstay of treatment for pediatric obstructive sleep apnea syndrome (OSAS). However, there is evidence that the child with severe OSAS is at increased risk of respiratory compromise. The most difficult risk factor to assess is the severity of OSAS, and these difficulties are reviewed.

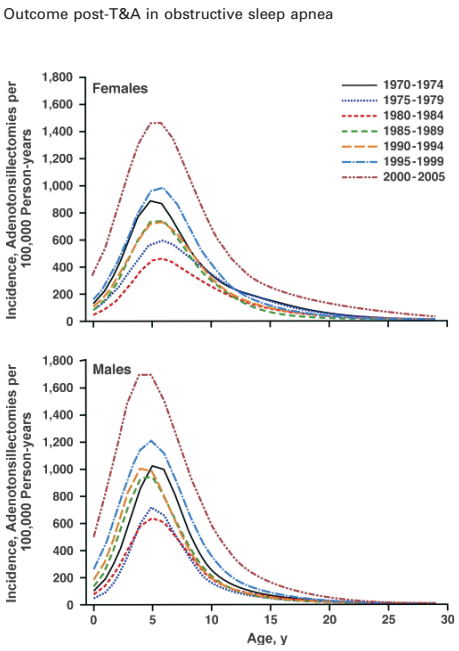

Figure 1 The rate of adenotonillectomy (T&A), normalized to 100 000 person years, in Olmsted County, Minnesota, USA between 1970 and 2005. The nadir surgery occurred 1980–1984, but in the 1990s there has been a resurgence in the rate of T&A. Reproduced with permission from Erickson *et al.* (7).

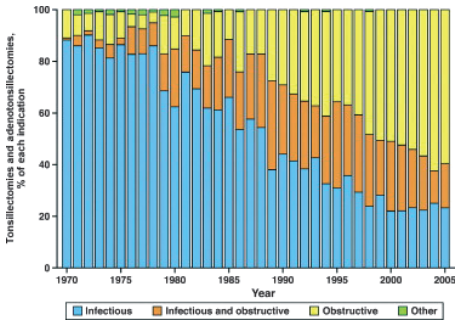

Figure 2 Surgical indications for adenotonsillectomy (T&A) in Olmsted County, Minnesota, USA between 1970 and 2005. Reproduced with permission from Erickson *et al.* (7).

Introduction

In 1976, Guilleminault reported sleep apnea in eight children (1). The sleep disturbance in the pediatric obstructive sleep apnea syndrome (OSAS) is characterized by loud snoring, episodic hemoglobin desaturation, hypercapnia, and repeated arousals from sleep. Sleep-disordered breathing may lead to a spectrum of symptoms including neurocognitive and behavioral disturbances, poor school performance, cardiovascular dysfunction, and pulmonary disease (2–5). Whereas, primary benign snoring occurs in 5–27% of children and pediatric OSAS affects only 1–3% (3,6). Pediatric OSAS is as prevalent as childhood asthma. This article discusses Outcome, Risk, and Error in the perioperative management of the child with the severe obstructive sleep apnea who is to undergo tonsillectomy with or without adenoidectomy (abbreviated T&A). The focus is on respiratory morbidity.

Adenotonsillar hypertrophy is a major factor in the development of obstructive breathing during sleep in children, and therefore, T&A is the mainstay of the treatment for OSAS (3), exhibiting a bimodal age distribution with peaks at 5–8 and 17–21 years (7). There has been a resurgence in the rate of T&A. (Figure 1)

Adenotonsillectomy represents the most frequently performed ambulatory pediatric surgery in the United States (7). For the year 2005, the American National Health Statistics reported that 530 000 ambulatory T&As were performed in children (under 15 years) in American ambulatory programs including hospitals and freestanding ambulatory surgery centers (8).

Erickson et al. examined trends for T&A registered in the Rochester Epidemiology Project database of Olmsted County, Minnesota. Whereas in 1970, the indication for T&A in the majority (88%) was infection, today, the majority indication is pharyngeal obstruction (77%) (Figure 2) (7). (Concordance between the surgical indication reported from the electronic search and a chart review was 91%, in a randomly selected subset of 5%). Worldwide, however, there seem to be important differences. The National Prospective Tonsillectomy Audit of practice in the United Kingdom (https://www.tonsil-audit.org/documents/ta_finalreport.pdf) reported that the indication for T&A in only 10% of patients was pharyngeal obstruction / obstructive sleep apnea in 2003/2004. Furthermore, in young children (<5 years), only a third reported pharyngeal obstruction as the indication for T&A (9). As discussed below, the risk of post-T&A respiratory morbidity is higher if the surgical indication is OSAS. Thus global differences in the surgical indication for T&A may underlie regional differences in perioperative morbidity.

Outcome

Deficiencies, highlighted in 1979 by Pratt and Gallagher (10), in the reporting of complications following T&A, have limited accurate reporting of outcomes. Even today, centralized reporting of patients readmitted after discharge from outpatient facilities may not be available, and therefore, statistics relating to postoperative complications are lacking (11). In a recent report of lethal post-tonsillectomy hemorrhage from Germany (12), the method for retrieval of subjects was voluntary reporting.

An estimate of mortality following T&A is 0.6 per 10 000 (12). Although lethal hemorrhage following T&A occurs, less than one-third of tonsillectomy mortality is attributed to bleeding (12,13). The Medical Liability Mutual Insurance Company in New York State reported 36 court trials for malpractice claims of death/major brain injury following T&A, between 1985 and 2007 (13). Nineteen subjects (53%) of death/major brain injury were because of airway complications and postoperative airway events accounted for the majority (60%) of death/major brain injury in children. Compared with adults, children had a twofold higher incidence of fatal respiratory events in the postoperative period following T&A (Table 1). Common postoperative airway complications included airway obstruction and respiratory arrest of unclear etiology. Court trials represent a minority of malpractice claims, and therefore, data should be interpreted cautiously. Nonetheless, it would seem that an additional factor is acting on children to increase the risk of respiratory mortality in the postoperative period. A discussion of risk factors for respiratory complications following T&A is warranted.

Table 1 Frequency of death or profound brain injury from airway and bleeding complications following adenotonsillectomy (T&A). Modified with permission from Morris *et al.* (13)

	Death/brain injury (%)
Children (n = 25)	
Airway (intraoperative)	4 (16)
Airway (postoperative)	11 (44)
Post-T&A hemorrhage	8 (32)
Adult (n = 11)	
Airway (intraoperative)	2 (18)
Airway (postoperative)	2 (18)
Post-T&A hemorrhage	4 (36)

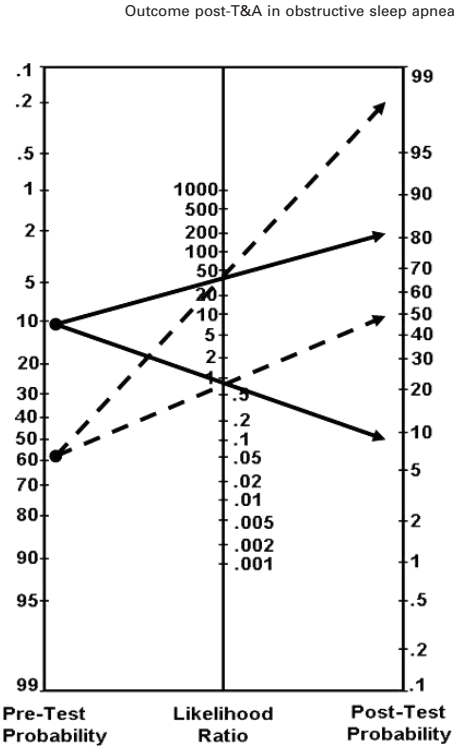

Figure 3 Normogram showing the Pre-Test probability, likelihood ratios, and Post-Test probabilities for pulse oximetry trend studies predicting obstructive sleep apnea syndrome (positive) or not (negative). In otherwise healthy children a positive pulse oximetry trend graph has a likelihood ratio of 43 and a negative pulse oximetry trend graph had a likelihood ratio of 0.6. The Post-Test probability predicting obstructive sleep apnea high, 60%, (—) and low, 10%, (—) Pre-Test probabilities are shown. See text for further explanation. Adapted from Brouillette *et al.* (23).

Risk of post-T&A respiratory complications

Obstructive sleep apnea syndrome

There is overwhelming evidence that children with OSAS have a higher incidence of postoperative respiratory complications including post-obstructive pulmonary edema, pneumonia, airway obstruction, and respiratory failure (14–20). There is very strong evidence that the severity of the sleep apnea is an important determinant of this risk. Clinicians become more adept in the identification of the at-risk child if an assessment of OSAS severity is available.

The gold standard for determining OSAS severity requires an assessment of obstructive breathing during sleep with either the apnea hypopnea index (AHI) or respiratory disturbance index (RDI). In children, an AHI that exceeds one event per hour sleep is abnormal (5), but in children with OSAS, the AHI may range from 1 to over 100 events per hour. It is an AHI threshold value above 10 events per hour that has been linked to the risk of respiratory complications following T&A (14,15,17–21). Children with severe OSAS also exhibit hypoxemia during the sleep-related obstructive apnea and hypopnea. Profound recurrent hypoxemia during sleep, below a threshold value of 80%, increases the risk of major medical interventions following T&A (14,17–20).

The predictive value of nocturnal oximetry has been evaluated with likelihood ratios, the ratio of true positives and false positives, and a likelihood ratio above 10 indicates a conclusive change from the pretest to posttest probability of disease (22). Brouillette et al. determined likelihood ratios for the oximetry trend graphs and the probability that an abnormal study would be associated with polysomnographic evidence of OSAS (AHI > 1 event per hour) (23). They determined the likelihood ratio for otherwise healthy children (n = 165) with only adenotonsillar hypertrophy as the explanation of their sleep-disordered breathing. An abnormal oximetry trend graph had a likelihood ratio of 43, and a negative/inconclusive result had a likelihood ratio of 0.6. In the population referred to the sleep laboratory, the pretest probability of OSAS was high (64%), and the high likelihood ratio of 43 gave a 99% posttest probability of having OSAS (Figure 3).

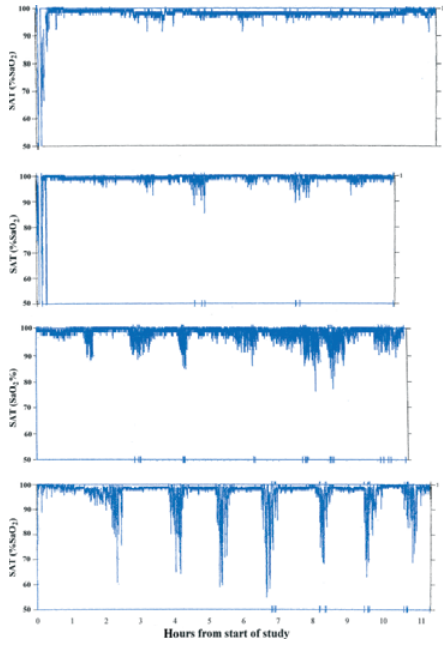

Figure 4 Examples of the oxygen saturation trend graphs from overnight oximetry tests used to develop the McGill Oximetry Score (MOS): from top to bottom, MOS1, MOS2, MOS3 and MOS4. Reproduced with permission from Nixon *et al.* (19) and from Hannallah RS, Brown KA, Verghese ST. Otorhinolaryngologic Procedures. In: Coté CJ, Lerman J, Todres ID, eds. *A practice of anesthesia for infants and children*, 4th edn. Philadelphia, PA: Saunders, 2009:657–683.

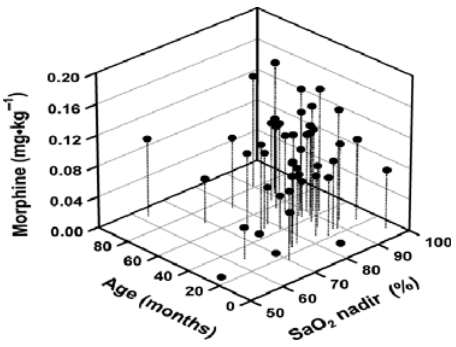

Figure 5 Age (months) and preoperative arterial oxygen saturation (SaO_2) nadir (%) are significantly correlated with the cumulative postoperative morphine dose (mg/kg) required for analgesia after adenotonsillectomy. The correlation of the combined variables with the cumulative postoperative morphine dose is shown. Estimates for each child are depicted with a filled circle. The magnitude of the cumulative postoperative morphine dose in the three-dimensional scatter plot is depicted by the height of the stem supporting each circle. Reproduced with permission from Brown *et al.* (25).

Nixon et al. extended this work to develop an OSAS severity score, the McGill Oximetry Score (MOS) (19). An abnormal oximetry trend graph was defined as one with ‡3 clusters of desaturation. Three levels of severity (MOS2, MOS3, and MOS4) were identified from the nadir saturation in at least three clusters: <90%, <85%, and <80%, respectively (Figure 4). There are some important take-home messages arising from centers using nocturnal oximetry to assess sleepdisordered breathing which may be relevant to clinicians.

(1) In populations of otherwise healthy children, with only adenotonsillar hypertrophy as the explanation of their sleep-disordered breathing, a high pretest probability of having OSAS combined with the high likelihood ratio of 43 yields a probability that an abnormal oximetry trend graph is associated with OSAS of 99%. In our sleep laboratory, these children require no additional testing to establish the diagnosis of OSAS (19).

(2) The nomogram provided in Figure 3 can be used to estimate the posttest probability of having OSAS for any surgical population of otherwise healthy children for any pretest value. In surgical practices with a high incidence of OSAS, i.e., 77%, the likelihood ratio of 43, means that an abnormal oximetry trend graph yields a posttest probability of having OSAS of 100%. Even in surgical practices with a low incidence of OSAS, i.e., 10%, the high likelihood ratio of 43, means that an abnormal oximetry trend graph has a posttest probability of having OSAS above 80%.

(3) The nadir saturation during sleep is inversely correlated with the AHI (Table 2). We reported that children exhibiting recurrent hypoxemia during sleep (i.e.,MOS2, MOS3, and MOS4) have values or the AHI above 10 events per hour (19). An AHI > 10 is the threshold value predictive of respiratory complications following T&A (15).

(4) The risk of postoperative respiratory complications increases as the severity of preoperative nocturnal hypoxemia worsens (14,17–20) (Table 2).

Table 2 Correlation of the preoperative McGill Oximetry Score (MOS) with the preoperative apnea/ hypopnea index and the post-adenotonsillectomy (T&A) respiratory compromise. Modified with permission from Nixon *et al.* (19)

	Apnea/hypopnea index (events per hour)	Post-T&A respiratory compromise %
MOS2	12.6	35
MOS3	13.3	60
MOS4	39.9	62

(5) Children with profound hypoxemia (i.e., nadir saturation <80% and MOS4) demonstrate a heightened respiratory (24) and analgesic (25,26) sensitivity to opioids (Figure 5). We have linked this heightened analgesic sensitivity to opioids with the report that intermittent hypoxia increases mu-opioid receptor density in piglets (27) and speculate that the molecular process mediating this response involves oxygen-sensitive gene regulation (28). Children living at altitude (chronic hypoxia) also demonstrate a decreased analgesic opioid requirement (29). However, the molecular mechanisms underlying their heightened opioid sensitivity may differ as the stimuli of intermittent and chronic hypoxia illicit distinct physiological and molecular responses (30,31).

(6) The surgical population for T&A exhibits a very high prevalence of hypoxemia during sleep. A third of an unselected surgical population of 44 children with adenotonsillar hypertrophy assessed the night prior to T&A exhibited baseline

saturations below 90% and/or episodic hypoxemia during sleep (32). The incidence of recurrent hypoxia during sleep in a surgical population of 334 children referred for preoperative evaluation of sleep-disordered breathing was 30% (33). In comparison, only 2.4% of an unselected population of German primary school children demonstrated recurrent episodic desaturation during sleep (6).

Ethnicity

Although there is, as yet, no evidence that ethnicity is an independent risk factor for respiratory morbidity following T&A, there is strong evidence that African American ethnicity is a risk factor for OSAS. Sleepdisordered breathing is almost twice as prevalent in young African Americans compared with Caucasians (34) and these children are three times more likely than controls to have OSAS (35). African American children with sleep-disordered breathing report more difficulty with nasal breathing during wakefulness than controls. In addition, African American children with OSAS had significantly lower oxygen saturations during obstructive airway events compared with other ethnicities, and the median (interquartile range) of the nadir saturation during rapid eye movement sleep in African American children was 80% (24,36).

Multisystem disease

Pediatric OSAS is a multisystem disease, and severe OSAS is associated with abnormalities that may of themselves increase the risk for perioperative complications. These pathophysiologies include pulmonary and systemic hypertension, ventricular hypertrophy, and lower airways disease (2,3,16). Alterations in ventilatory control may also be present. Don et al. remarked that children with OSAS demonstrate a higher than expected central apnea rate (average 2.5 per hour) and suggested this may reflect abnormalities of ventilatory control (16). Shine et al. reported a high incidence of central apnea in obese children with OSAS (21). Children with OSAS demonstrate a blunted responsiveness to carbon dioxide (37). Alterations in ventilatory control may be linked to the heightened respiratory sensitivity to halothane and opioids reported by Waters et al. (24).

Age

Young age is an independent risk factor for respiratory complications following T&A (14,15,17). In addition, hypoxemia during sleep-disordered breathing is common in young children. Figure 6 shows that hypoxemia during sleep is more common in young children with OSAS than older children (16). Constantine et al. reported that 39% of children with OSAS who were under 3 years had hypoxemia during sleep-disordered breathing and for each 1-year increase in age, the odds of having hypoxemia during sleep decreased by 17% (33).

Medical co-morbidity

The presence of a medical co-morbidity is an independent risk factor for respiratory complications following T&A (14,15,17). This is true regardless of the nature or the number of the co-morbidities be they prematurity, neuromuscular disease, seizure disorder, Down syndrome, craniofacial anomalies, cardiac disease, asthma, or obesity. Because the overall risk is the product, not the sum, of independent risk factors, combinations of young age, medical co-morbidity, and severe OSAS identify a child at high risk of post-T&A respiratory complications. One study found that 7 of 61 (11%) children under 2 years, with medical co-morbidity and severe OSAS, required a major medical intervention for respiratory complications following T&A (38).

Obesity is a medical co-morbidity that deserves special mention as it is both a cause and consequence of OSAS. Both OSAS and obesity are systemic inflammatory diseases, and there is evidence of metabolic disturbances in children when OSAS and obesity coincide (5). The risk of sleep-disordered breathing in obese children increases fourfold (39) and for every BMI increment of 1 kg/m2, beyond the mean BMI, the risk of OSAS increases 12% (40). The prevalence of severe OSAS in obese children is 46% (41). However, in a retrospective comparison between otherwise healthy obese and non-obese children with polysomnogram (PSG) proven OSAS, there was no evidence for a significant correlation between the degree of obesity and the severity of OSAS (40). Mallampati scores were higher in the obese children. Shine et al. (21) reviewed 26 consecutive morbidly obese (BMI > 95th percentile) children (2–17 years) admitted to intensive care following T&A for the treatment

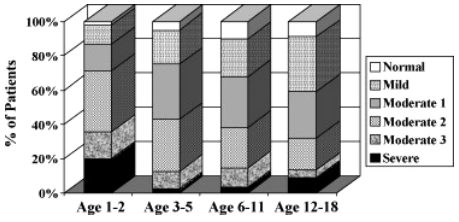

Figure 6 Bar graph representing the percentage of patients by age in the normal, mild, moderate and severe obstructive sleep apnea syndrome categories. Children in the moderate and severe categories demonstrated recurrent episodic hypoxia during sleep. Reproduced with permission from Don *et al.* (16).

of OSAS. Whereas no threshold of RDI was associated with postoperative morbidity, a preoperative sleep study with a saturation nadir below 70% and/or central apnea was associated with respiratory compromise following T&A. The severity of OSAS in obese children can only be determined by preoperative testing.

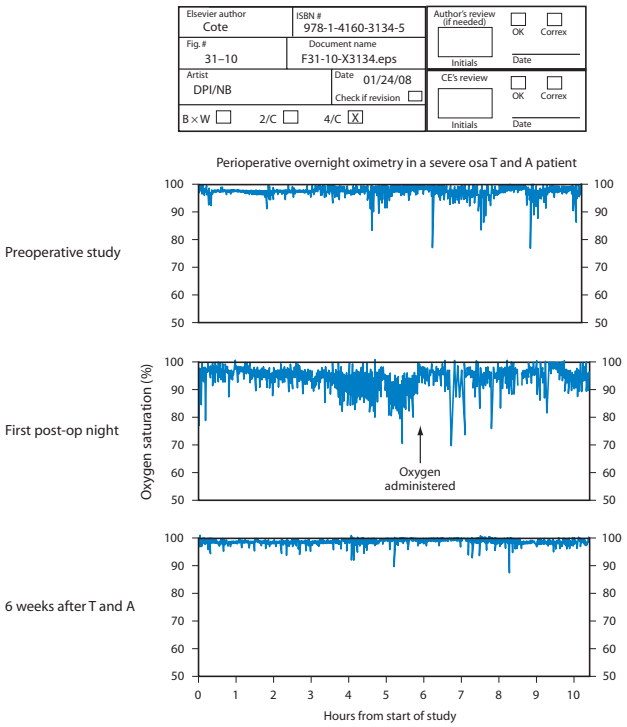

Figure 7 Overnight oximetry trend reports for an otherwise healthy 2.8 year old boy. Clusters of desaturation are seen in the preoperative study, reflecting obstructive events in rapid eye movement sleep. On the first postoperative night, obstructive apneas and hypopneas occurred frequently from sleep onset, leading to repetitive desaturation below 80%. Note administration of supplemental oxygen 12 h after T&A. He was admitted for an additional 2 nights until desaturation during sleep had resolved. Oximetry performed 6 weeks after surgery is within normal limits. Reproduced with permission from Nixon *et al.* (42) and from Hannallah RS, Brown KA, Verghese ST. Otorhinolaryngologic Procedures. In: Coté CJ, Lerman J, Todres ID, eds. *A Practice of Anesthesia for Infants and Children*, 4th edn. Philadelphia, PA: Saunders, 2009:657–683.

Residual OSAS

Nixon et al. recorded abbreviated cardiorespiratory studies on the night following T&A in otherwise healthy children, with OSAS, who were aged over 2 years,. All children demonstrated obstructive airway events on the first night following T&A (Figure 7). These events were four times more likely in those children with severe nocturnal hypoxemia on the preoperative oximetry trend graph. Virtually, all desaturation events were associated with obstructive events. The duration of the mixed obstructive apnea, recorded on the first night following T&A, exceeded 20 s in the severe group (42) (Table 3).

Table 3 Parameters [mean (range)] of sleep and breathing on preoperative and first night following adenotonsillectomy in children with the obstructive sleep apnea syndrome (OSAS) by severity of preoperative nocturnal hypoxemia. Modified with permission from Nixon *et al.* (42)

	Severe group (n = 5)	Mild group (n = 5)	P value
Nadir saturation on preoperative overnight oximetry (%)	76 (54–79)	87 (86–91)	0.009
First postoperative Night			
Mixed/obstructive Apnea/hypopnea index (events per hour)	21.5 (15.1–112.1)	6.9 (2.2–9.8)	0.009
Nadir saturation (%)	82.9 (73.5–89.5)	88.0 (81.0–90.0)	0.46
Desaturation events due to obstructive events (%)	99.6 (94.0–99.8)	66.9 (19.9–89.5)	0.02
Longest mixed/obstructive apnea (sec)	24.3 (23.1–27.2)	14.0 (10.8–19.2)	0.05

A multinational retrospective study involving nine pediatric centers reported that whereas two-thirds of children with mild and moderate OSAS show complete resolution of their sleep-disordered breathing six weeks following T&A, one-quarter of children with severe OSAS do not. Residual disease was more likely in older children (>7 years) and in the obese (3). In obese children compared with non-obese children, the odds ratio for persistent OSAS (defined by an RDI > 5) was 4 (43). Indeed Dayyat et al. propose 2 types of OSAS disease exist in children, one associated primarily with obesity and the second associated with marked adenotonsillar hypertrophy (5). In non-obese children, asthma and severe OSAS were more likely to be associated with residual disease (3) and a preoperative AHI > 19 events per hour was associated with residual disease (44).

Anesthetic management

There is little published evidence to recommend a specific anesthetic technique for T&A in the child with OSAS. Helfaer et al. (45) reported no difference in respiratory outcome between a halothane anesthetic technique and a balanced technique which included fentanyl in a children with mild OSAS (AHI = 5 events per hour). However, in children with severe OSAS, reports of an altered ventilatory control, an increased respiratory sensitivity to halothane, and a heightened opioid sensitivity suggest that these children may be more vulnerable to respiratory depression during anesthesia and recovery. A management strategy individualized to the severity of preoperative hypoxemia reduced the incidence of respiratory complications following T&A in children (46).

Error

Management of OSAS on an outpatient basis

Our criteria for hospitalization following T&A include medical co-morbidity, young age, a bleeding diathesis, disadvantaged social environment, excessive distance from a hospital, excessive pain, poor oral intake, postoperative vomiting, and an awake (room air) saturation below 95%. It is likely that the children represented in Figure 7 and Table 3 would have met the discharge criteria of most outpatient facilities, as these children were awake and taking oral fluids in the afternoon following T&A. However, at least 5 h following surgery, with sleep onset, all children demonstrated obstructive respiratory events on the first night following T&A (42). Any child with a history of severe OSAS, young age, and/or medical co-morbidity must be admitted following T&A and observed in the safety net of the hospital environment.

All children exhibiting hypoxemia on the preoperative sleep study should be monitored with continuous oximetry during sleep following T&A. Helfaer et al. concluded that the improvement in saturation and obstructive respiratory events in children with mild OSAS (AHI = 5) did not justify intensive monitoring on the first night following T&A (45). Many advocate elective admission to the intensive care unit for children with severe OSAS (AHI > 10, nadir saturation <80%, carbon dioxide retention) (15,20,21). However, we have reported that when the perioperative management is individualized to the severity OSA, utilization of the intensive care unit for children with severe OSAS decreased (46). Our local practice recommends that post-T&A admission to the intensive care unit be reserved for children with severe recurrent episodic hypoxia (MOS4) plus young age (<2years) and/or medical co-morbidity. In the absence of intraoperative complications, our practice is to monitor otherwise healthy children with severe recurrent episodic hypoxia (MOS4), overnight in the recovery room with continuous oximetry.

Whereas, young age and medical co-morbidity are easily identifiable risk factors, OSAS is more difficult to diagnose in children. Health care systems with access to pediatric sleep laboratories are able to identify children with severe OSAS and exclude them from ambulatory T&A programs. In the absence of preoperative testing, guidelines published by the American Society of Anesthesiology suggest that clinical criteria may be used to establish a diagnosis of OSAS (2). However, agreement between clinical criteria and the gold standard polysomnography is only 55% (47), and preoperative questionnaires have a sensitivity of only 40% for detecting hypoxemia during sleep (33).

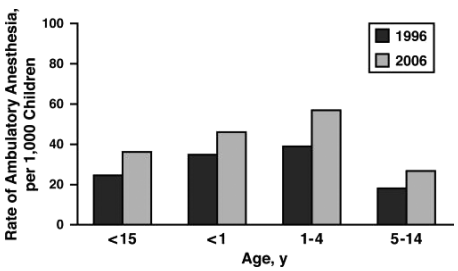

Figure 8 Rate of Ambulatory Anesthesia for children in the United States of America in 1996 and 2006. Rate increased from 26 per 1000 children younger than 15 years in 1996 to 38 per 1000 children of this age group in 2006. Reproduced with permission from Rabbits *et al.* (11).

For children aged 1–4 years, the rate of ambulatory anesthesia has increased from 6.2 per 1000 in 1996 to 13.2 per 1000 in 2006 (Figure 8) (11). In this age group, the majority of surgical indications for T&A is likely to have been obstructive breathing, and children in this age group have a high incidence of severe OSAS associated with hypoxemia during sleep (16,33). The average postoperative time prior to discharge

from in the recovery room is reported to be 71 min (11), a postoperative observation period which is too short to detect the delayed onset of respiratory compromise reported in children with severe OSAS following T&A (15,16,48). The onus then falls on the anesthesiologist to identify the child with severe OSAS by an unusual response to anesthesia. Premedication may cause excessive snoring, desaturation, and obstructive apnea (49). A difficult mask induction may suggest a high pharyngeal closing pressure (50,51). Excessive respiratory sensitivity to opioids and anesthesia resulting in hypoventilation and apnea may be present (24,52). Children with severe OSAS demonstrate a delayed emergence from anesthesia (18). A persistent oxygen requirement especially during sleep is common in children with severe OSAS following T&A (17–19,42,46).

A system that relies on the behavior during anesthesia and recovery to identify the child with OSAS who should be admitted to hospital is subject to error. Herein lies the potential for nocturnal oximetry as a tool to assess eligibility for ambulatory T&A programs in the preoperative period. An abnormal oximetry trend graph, in otherwise healthy children with adenotonsillar hypertrophy as the explanation for their sleep-disordered breathing, has a very high likelihood ratio for predicting OSAS. Even in populations with a low pretest probability of having OSAS, the posttest probability of having OSAS is high (Figure 3). Furthermore, an abnormal oximetry trend graph stratifies the severity of nocturnal hypoxemia allowing exclusion or the high risk child from ambulatory T&A programs. Widespread implementation of nocturnal home oximetry evaluation has been slow. For the better part of the twentieth century, the medical elite has questioned the utility of T&A (53) and today, most health care systems define and remunerate specific indications for T&A. This may be a deterrent to preoperative assessment with noctural home oximetry, as surgeons may fear that a negative result would not support the clinical diagnosis of OSAS. However, the likelihood ratio for a negative oximetry trend graph is only 0.6, a value which is not small enough to rule out a diagnosis of OSAS (22).

Conclusion

Data from the Medical Liability Mutual Insurance Company in New York State suggest that compared with adults, children are experiencing a higher incidence of lethal respiratory events in the postoperative period following T&A (13). Overall, the mortality rate for T&A is low, estimated at 0.6 per 10 000 (12), and as the caseload is scattered across hundreds of ambulatory programs, local departmental reviews of morbidity and mortality may not detect trends in respiratory morbidity. Reliable centralized reporting of morbidity and mortality following T&A would be helpful in this regard. With the escalating frequency of OSAS in children undergoing T&A and the obesity epidemic in children worldwide, perioperative respiratory complications following T&A pose a serious risk to children. Simple non-invasive preoperative diagnostic evaluations are needed to identify at-risk children. Polysomnography cannot fulfill these requirements, and alternative strategies including nocturnal oximetry need to be examined. In addition, further studies are required to develop affordable, accessible, and costeffective monitoring to identify those children with OSAS who will experience respiratory depression following adenotonsillectomy.

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IS IT REALISTIC TO SCREEN PATIENTS FOR SLEEP APNEA AND OBTAIN POLYSOMNOGRAPHY PREOPERATIVELY? A CASE DISCUSSION HIGHLIGHTING THE UNIVERSITY OF CHICAGO EXPERIENCE

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A 69 year old man with osteoarthritis is evaluated for a total knee replacement, and scheduled for one week from today. He has several risk factors for obstructive sleep apnea (OSA) but has never had a sleep study.

Although many major academic medical centers and other large anesthesia practices have preoperative clinics to evaluate patients before the day of surgery, many smaller centers do not have this luxury. In clinics like ours, patients can be screened for OSA and undergo polysomnography (PSG) preoperatively. But is this realistic? The University of Chicago Anesthesia Perioperative Medicine Clinic (APMC) has collaborated with the Department of Medicine and the Sleep Medicine Center to obtain preoperative PSG in all patients deemed at risk for OSA, to examine both its feasibility and possible benefit.

The surgeon's office makes an appointment for our patient with the APMC that afternoon. An anesthesia resident evaluates the patient in clinic and collects his medical data. On the STOP-Bang screening survey, the patient scores a 5, indicating that he is at high risk for OSA.

OSA remains undiagnosed in the majority of patients, and rates tend to be higher in surgical populations. Not all cases of OSA are clinically obvious; some cases of OSA may be found only by specifically screening pre-surgical patients. The cost of preoperative screenings resulting in false positives, unnecessary polysomnograms, admissions, and monitoring, also must be considered. The gold standard test for OSA diagnosis is the PSG, which is often conceptualized as expensive and impractical, particularly in the preoperative period.

The resident discusses the patient with the clinic anesthesiologist, and preoperative planning includes a referral to the Sleep Medicine Center for a preoperative PSG. The importance of an OSA diagnosis is discussed with the patient, who agrees to make an appointment with some reluctance, saying "I have survived other surgeries just fine without this sleep study."

In 2009, our preoperative clinic began screening patients for OSA with the STOP-Bang questionnaire. In collaboration with the Sleep Center a requisition is faxed from the clinic and the patient is contacted by phone to arrange an appointment. The Sleep Center guarantees completion of a preoperative PSG for patients with a STOP-Bang score ≥ 3, and initiation of continuous positive airway pressure (CPAP) therapy if indicated, with an appointment made available within 3 days of referral if patients agree.

When the Sleep Center contacts the patient he refuses a PSG. The next day he calls the APMC and says he does not need "all of this unnecessary testing" before surgery. The short- and long-term risks of undiagnosed OSA are explained to him in detail, and after discussion with his surgeon, he is notified that anesthesia services will not be provided unless he has the appropriate testing before his elective procedure. The patient schedules his sleep study the next day.

Several logistical problems were identified while implementing and improving our preoperative OSA screening program. The most common is that patients simply do not comply with recommendation for polysomnography, despite open availability and ease of scheduling provided by our Sleep Center. Opinions also differed amongst clinic attendings as to when, and on whom, PSG should be performed. A key component involved sharing PSG data on our patient referrals with our providers. Once they saw that the majority of patients referred from the APMC met criteria for moderate-severe OSA and over 50% obtained CPAP devices preoperatively, the providers realized the feasibility of this program. Education of attendings, residents, and physician assistants has led to greater compliance with screening recommendations. Frank discussions with patients about the rationale for, and importance of, diagnosis and treatment of OSA can improve compliance as seen in our case.

After a PSG, our patient is diagnosed with severe OSA, which results in CPAP therapy initiation before an uneventful surgery and recovery. He expresses his appreciation for the care provided by, and the persistence of, his perioperative team.

As of March 2011, 432 patients have been referred to the Sleep Center after being identified in the APMC as high risk for OSA. Of

these, only 213 (49%) completed a PSG, in spite of unlimited access. Over half of patients with STOP-Bang scores ≥ 3, and the great majority of those with STOP-Bang score ≥ 5 had moderate-severe OSA, diagnosed by an apnea-hypopnea index greater than 15 during PSG (64% and 81% respectively, $p < 0.005$). Of all patients who completed polysomnograms, CPAP therapy was initiated during the initial sleep study for 54% with STOP-Bang score ≥ 3, and 71% with score ≥ 5. In addition patients with STOP-Bang score ≥ 5 had higher body mass indices and apnea-hypopnea indices, and spent significantly more total sleep time with oxygen saturations < 90% (Table 1). As our process has evolved, and based on data obtained from sleep studies over the past 2 years, only patients with STOP-Bang score ≥ 5 are now routinely referred for PSG.

In areas where PSG is not readily available, and patients are not routinely seen by an anesthesiologist before surgery, screening could be initiated in the surgeon's or the primary physician's office. Even screening on the day of surgery can identify those at high risk based on the STOP-Bang score alone. Then an anesthetic can be tailored to a patient with a high likelihood of having OSA, and CPAP therapy can be initiated postoperatively. Our data (Table 2) suggest that using pressures of 8-12cmH_2O should be adequate for the majority of patients who require CPAP. Our patient could have been treated with this approach had he been unwilling to obtain the study. However, in-hospital postoperative CPAP does not provide the possible long-term health benefits of continued home CPAP therapy and follow-up with Sleep Medicine specialists.

Our recommendations for preoperative OSA screening implementation include the following:

1. Screening for OSA is recommended with the STOP-Bang questionnaire, either during the preoperative anesthesia, surgery, or primary care physician visit, including on the day of surgery.
2. STOP-Bang is a sensitive and relatively specific test (if a score of ≥ 5 is used as a cutoff for increased risk) that correlates well with PSG results.
3. Timely preoperative PSG and initiation of CPAP is a possibility for patients identified at high risk by screening.
4. Compliance is the main barrier because many patients will not complete PSG, even if recommended.
5. Alternatively, in high-risk patients identified by screening questionnaire but unable or unwilling to undergo preoperative PSG, a presumptive diagnosis can be made, an anesthetic plan can be tailored to the presumed diagnosis, and CPAP therapy can be initiated postoperatively (8-12cm H_2O) if necessary.

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Table 1: PSG results grouped by STOP Bang score

PSG	Score 3-4	Score 5+	p Value
Mod-Severe OSA	60 (64%)	96 (81%)	< 0.005
AHI	25 (8-33)	38 (31-39)	< 0.001
BMI	31 (25-35)	36 (19-53)	< 0.001
%TST < 90%	6 (0-6)	12 (1-20)	< 0.03
Total patients	n = 94	n = 119	

Means and interquartile ranges reported for continuous data, number and % for categorical data.
PSG = polysomnogram; AHI = apnea-hypopnea index; BMI= body mass index; TST = total sleep time.

Table 2: Final CPAP data grouped by STOP Bang score

PSG	Score 3-4	Score 5+	p Value
CPAP Initiation	51 (54%)	85 (71%)	< 0.03
Final CPAP (cm H_2O)	8.6 (8-10)	9.1 (7-10)	0.36
Total patients	n = 94	n = 119	

Means and interquartile ranges are reported for continuous data, number and % for categorical data.
PSG = polysomnogram; CPAP = continuous positive airway pressure

PERIOPERATIVE COMPLICATIONS IN OSA PATIENTS: EVIDENCE-BASED REVIEW OF THE LITERATURE

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Untreated obstructive sleep apnea (OSA) patients results in greater morbidity and mortality. Untreated OSA patients are at a greater risk of developing cardiovascular disease (OR 3.1, 95% CI 1.2-8.3), including heart failure, arrhythmias (2-4 fold increase), hypertension, (10-fold increase) and stroke (OR 4.33, 95%CI 1.32-14.24).[1] In addition, OSA is independently linked to the development of metabolic syndrome (OR 9.1,95% CI2.6-31.2), stroke and death from any cause.[1]

Can OSA affect perioperative outcome and are patients with OSA at an increased risk for perioperative complications? A systematic search of the literature indicates that there are 11 studies in total (Table 1). Eight studies are in non-cardiac surgery, one in cardiac surgery and two in ambulatory surgery. Five of these studies are of prospective cohort designs and 6 are case control (retrospective). In 7 studies, the diagnosis of OSA is by polysomnography. In two studies, the diagnosis is by screening questionnaire, in one study, by oximetry and one from medical history and physical examination by physician.

In 2001, Gupta et al first studied the postoperative complications in 101 OSA patients undergoing hip or knee replacement.[2] Complications were noted in 39% of OSA patients and 18% of control group (P= 0.01). Serious complications occurred in 24 % of OSA patients versus 9% of the control group (P=0.004).

In a prospective cohort study, Chung et al found that the incidence of postoperative complications in 147 OSA patients vs. control were 27.4% vs. 12.3%, P=0.02.3 The most common complication was oxygen desaturation (20.6% vs. 9.2%; P< 0.04).[3] Gali et al also found an increased risk of postoperative complications (odds ratio 2.7 P=0.004) in 221 high risk OSA patients.[4]

In 262 OSA patients, Kaw et al found increased postoperative complications (OR 6.4, P=0.0005).[5] Similarly, in 240 OSA patients, Liao et al found that the incidence of postoperative complication was increased (OSA vs. control 44% vs. 28%, P< 0.05).[6] OSA and pre-existing stroke were the most significant risk factor for developing postoperative complications. The hazard ratio for OSA was 2.0 (1.25-3.19).[6] In addition, Hwang et al found that 98 patients with OSD4% > 5 sign had a significantly higher rate of postoperative complications than those with an ODI4% < 5 (15.3% vs. 2.7%, p<0.01).[7]

To date, the largest study is by Memtsoudis et al.[8] From the National Inpatient Sample, data from 1998 to 2007, 65,774 sleep apnea patients undergoing orthopedic procedures and 51,509 sleep apnea patients undergoing general surgical procedures were examined for perioperative complications. Sleep apnea was associated with a significantly higher adjusted OR of developed pulmonary complications after both orthopedic and general surgical procedures respectively.[8] They are mainly aspiration pneumonia, acute respiratory distress syndrome, and intubation/mechanical ventilation.[8] For the orthopedic OSA patients, pulmonary embolism is also more common.

In cardiac surgery, Kaw et al studied 37 OSA patients and found a higher incidence of encephalopathy and postoperative infection. There are two studies in ambulatory surgery.[9] Both Sabers et al and Stierer did not found an increase of unanticipated admission in 234 and 103 OSA patients respectively.[10,11] With the advancement of anesthesia technique, short acting anesthetic agents and non-invasive surgery, ambulatory surgery is very safe. The incidence of unanticipated admission after ambulatory surgery is very low, normally 1-2 %. Therefore it is difficult to conclude that ambulatory surgery is completely safe for OSA patients with a small number of patients. This is especially true in severe untreated or undiagnosed OSA patients that require postoperative narcotics after ambulatory surgery.

Of the 11 studies, there is only one study by Ahmad et al with negative results.[12] They showed no difference in adverse outcome of oxygen desaturation. They studied 31 OSA patients versus 9 non-OSA patients undergoing laparoscopic bariatric surgery. Postoperatively, they found no difference in median SpO_2 and the mean number of desaturation episodes per hour between OSA and non-OSA group.[12] However this study had methodological issue as patients in both groups received supplemental oxygen and the sample size is small.

For the length of stay in hospital, Gupta et al found that the stay was significantly longer in the OSA patients versus the control (6.8 + 2.8 days vs. 5.1 + 4.1 days, P< 0.007).[2] Kaw et al also found a longer hospital stay (OR 1.7, p=0.04) in the OSA patients undergoing non-cardiac surgery.5 In OSA patients undergoing cardiac surgery, there is also increased length of stay in ICU.[9]

Recently, Kaw et al did a meta-analysis of the perioperative complications.[13] Out of 6247 records, 13 studies were included in the final analysis (n=3942). OSA was associated with significantly higher odds of any postoperative cardiac events (45 /1195 [3.76%] vs. 24 /1420 [1.69%] respectively; OR 2.07; 95% CI 1.23-3.50, p=0.007 & acute respiratory failure (33/1680 [1.96%] vs. 24/3421 [0.70%]; OR 2.43, 95% CI 1.34-4.39, p=0.003. 13 . OSA was also significantly associated with higher odds of ICU transfer (105/2062 [5.09 %] vs. 58/3681 [1.57%] respectively; OR 2.29, 95% CI 1.62-3.24, p<=0.00001.[13]

Another point of interest is whether perioperative CPAP may decrease the risk of the perioperative complications in OSA patients. Gupta et al found that patients who were not using CPAP prior to hospitalization had a significantly higher incidence of serious complications with the implications that perioperative use of CPAP may decrease postoperative complications.[2] Interestingly, Liao et al found that the OSA patients who did not use home CPAP devices prior to surgery, but required the use of a CPAP device after surgery, had the highest rate of complications.[6] This finding is likely due to CPAP having been initiated in response to an adverse event. There is also the possibility that these patients were not compliant with use of CPAP in the preoperative period.

There are two contrary findings in the use of CPAP to prevent postoperative complications. Kaw et al[5] found that use of CPAP at home prior to non-cardiac surgery did not lower the risk of postoperative complications (p=0.8) or hospital (p=0.19). Ahmed et al also found that OSA patients who used their preoperative home CPAP devices during the first 24 hours postoperatively experienced a larger number of hypoxemic episodes and a larger percentage of time that the SpO_2 was below 90% compared with those that did not use CPAP.[12] It is possible that effective CPAP is altered in the postoperative period and the preoperative setting may not be adequate.[12] Alternately, patients who used the CPAP devices may have been a more severe subset of the OSA group.[12]

Incidence of postoperative respiratory desaturation, respiratory failure, postoperative cardiac events and ICU transfers was higher in patients with OSA. From these studies, we can conclude that we have to take extra precautions and care in dealing with OSA patients undergoing surgery in the perioperative period.

WHO ARE THE PATIENTS THAT REQUIRE POSTOPERATIVE MONITORING?

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Literature is insufficient to examine the impact of type, duration and place of postoperative monitoring: ICU vs continuous oximetry in patient's room in patients with obstructive sleep apnea.1 American Society of Anesthesiology recommends a median of 3 hours longer monitoring in patients with OSA after ambulatory surgery and 7 hours of monitoring after the last episode of airway obstruction or hypoxemia while breathing room air in an unstimulated environment prior to discharge.

The prevalence of OSA is rising in the general population and is higher among patients undergoing surgery. It is estimated that between 1990 and 1998, there was a 12 fold increase in the diagnosis of OSA in surgical outpatients.2 Preoperative workup and postoperative monitoring of such large numbers has to be practical and justifiable. One approach to preoperative risk stratification of patients with OSA may be to identify morbidities associated with and related to OSA. Obesity hypoventilation syndrome (OHS) represents an embedded epidemic of highly morbid patients within the obesity and OSA epidemics. Mortality as high as 23% has been reported in untreated OHS compared at 9% in well-matched obese cohorts at 1.5 yrs after hospital discharge, adjusted HR of 4.0(CI: 1.5-10.4).[3] A recent meta-analysis of a cohort of > 4000 patients with OSA reported a 19% prevalence of OHS confirming an overall prevalence of approximately 3 per 1000.4 A recent retrospective study showed that most patients with OHS are unrecognized at the time of elective surgery and among patients with OSA, those having OHS are at the highest risk of respiratory failure after elective surgery (44.4% vs 2.6%).5Although patients with OHS tend to have more severe OSA4, severity of OSA as measured by apnea-hypopnea index has not been shown to correlate with postoperative complications.

Patients with OSA often have pulmonary hypertension. PH is an emerging risk factor for postoperative complications after non-cardiac surgery. Besides potential postoperative mortality (upto7%); right ventricular failure (upto11%) & respiratory failure (upto 28%) are the most common postoperative complications in patients with PH.[6] Epidemiology of PH is changing; both primary and secondary PH are now more common in obese and elderly respectively. According to a recent study 22 of 27 (82 %) OSA patients with prolonged nocturnal desaturation (as defined by SpO_2 <90% for ≥10 %TST) had PH vs. 65% of patients without nocturnal desaturation.[6] Among demographic and polysomnographic variables, BMI (p=0.026), female gender (p=0.01), and duration of nocturnal desaturation (p=0.018) were the strongest correlates of PH among patients with OSA.[7]

The other subset of patients with OSA who can be considered very high risk category for postoperative complications is patients on chronic opioid therapy. Upto 85% of patients on chronic opioid therapy can have sleep apnea.[8] Patients on opiates for chronic pain can develop complex sleep apnea during the CPAP titration leading to

CPAP failures compared to patients with OSA not on opiates.[9] No data regarding perioperative complications is available among this group of patients.

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Table: Postoperative complications in OSA pts: A systematic review

Study	Design	Dx of OSA	No. of pts	Surgery	Postop Cx
Gupta 2001	Case control (retrospective)	PSG	OSA 101 Control 101	Hip or knee replacement	Postop Cx OSA vs. Control: 39% vs 18%, p<0.001 LOS OSA vs. Control: 6.8 vs 5.1 days, p<0.007
Sabers 2003	Case control (retrospective)	PSG	OSA 234 Control 234	Ambulatory surgery	Unanticipated admission OSA vs. control 23.9% vs 18.8% n.s.
Kaw 2006	Case control (retrospective)	PSG	OSA 37 Control 175	Cardiac surgery	↑ Encephalopathy (p<0.008) ↑ Postop. infection (p<0.028) ↑ ICU LOS (p< 0.031)
Chung 2008	Prospective Cohort	PSG	OSA 147 Control 64	Non cardiac surgery	Post op Cx OSA vs. control: 27.4% vs. 12.3% p< 0.016
Hwang 2008	Prospective Cohort	PSG	OD14% ≥5 98 OD14% ≥5 74	Non cardiac surgery	Postop Cx ODI4% ≥ 5 vs. ODI4% < 5 15.3% vs. 2.7% (p< 0.01)
Ahmad 2008	Prospective Cohort	PSG	OSA 31 Non-OSA 9	Laparoscopic bariatric surgery	No difference in median SPO_2 between OSA and non-OSA
Gali 2009	Prospective Cohort	Screening questionnaire	High risk of OSA 221 Low risk of OSA 472	Non cardiac surgery	Increased risk of postop. Cx (Odds ratio 2.7 P= 0.004)
Liao 2009	Case control (retrospective)	Physician diagnosis	OSA 240 Non-OSA 240	Non cardiac surgery	Postop Cx OSA vs. non-OSA: 44% vs. 28% (p<0.05)
Kaw 2009	Case control (retrospective)	PSG	OSA 262 Non-OSA 219	Non cardiac surgery	Increased postop. Cx (OR =6.4 P=0.0005) Longer hospital stay (OR =1.7 p=0.04)
Stierer 2010	Prospective Cohort	Screening questionnaire	OSA 103 Control 2036	Ambulatory surgery	No increased in unanticipated admission. Increased propensity with difficult intubation, intra-operative use of pressors and postop. O_2 desaturation.
Memtsoudis 2010	Case control (retrospective)	PSG	OSA 45547 Control 136541 OSA 58538 Control 175614	General surgery Orthopedic surgery	Increased risk of aspiration pneumonia, acute respiratory distress syndrome, and mechanical ventilation.

LOS = length of stay

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DOES SELF-TITRATING CPAP THERAPY IMPROVE POSTOPERATIVE OUTCOME IN PATIENTS AT RISK FOR OBSTRUCTIVE SLEEP APNEA SYNDROME? A RANDOMIZED CONTROLLED CLINICAL TRIAL

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Background: Obstructive Sleep Apnea (OSA) increases postoperative morbidity and mortality, and several methods of detecting patients at risk for OSA have been recommended.[1-4] Once identified, the efficacy of post-operative intervention with non-invasive positive airway pressure in patients thought to be at high risk has not been extensively studied.

Study Design: Orthopedic patients, scheduled for total hip or knee replacement, were prospectively assessed for risk of OSA using a clinical prediction tool.[5] Patients assigned low risk status received standard post-operative care. The high-risk group was randomized to receive standard post-operative care alone or in addition to post-operative auto-titrating positive airway pressure (APAP) therapy.[6] All patients underwent a cardio-respiratory sleep study prior to discharge to determine the presence of OSA.

Aims: The aims of our study were 1) to evaluate the effect of pre-emptive postoperative APAP on length of stay and post-operative complications in orthopedic patients identified as high-risk for OSA, 2) to assess the accuracy of the OSA risk assessment in an orthopaedic population.

Results: 137 patients were enrolled between 2002 and 2005. 52 were assigned low-risk and 85 high-risk status (42 randomized to standard care and 43 to receive additional post-operative APAP). Those randomized to APAP used the devices daily, but usage dropped from a median of 396 minutes of use on the 1st postoperative night to an overall median of 213 minutes nightly across their stay. The APAP devices detected a median AHI of 13.4. High-risk patients were more likely to have a post-operative complication than those of low-risk status (23.2% vs. 2.0%, p=0.0008), with the main differences manifesting in the increased need to adjust oxygen (20.73% vs. 1.96%, p=0.0014) and new infiltrates or atelectasis (8.5% vs. 0%, p=0.0434) in the high-risk group. The length of stay did not significantly vary between low and high-risk groups (p=0.1273). Post-operative APAP was not associated with significant differences in length of stay or overall rate of post-operative complications, but there was a trend toward higher rates of delirium (4.76% vs. 0%, p=0.4941) and new infiltrates or atelectasis (11.9% vs. 5%, p=0.4332). Narcotic requirement was less in the first 24 hrs. among APAP users (median morphine equiv. dose 82.5 vs. 56.2 mg, p=0.0221). The presence of OSA as determined by the cardio-respiratory sleep study did not predict length of stay or rate of post-operative complications.

Conclusion: In this study, high-risk status for OSA, as determined by a simple four-point questionnaire, identified patients at increased risk for post-operative complications. The risk questionnaire was a better indicator of postoperative risk than the post-operative polygram. Application of post-operative APAP in the high-risk group did not alter post-operative outcome. Further study is warranted to determine the role for positive airway pressure in the management of patients identified to be at high risk for OSA.

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MECHANISMS FOR RECURRENT APNEA

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Sleep apnea syndromes and sleep disordered breathing in general are characterized by the appearance of multiple apneas and hypopneas; and, hence, the problem becomes one of what mechanisms can produce an apnea and its reoccurrence over time. Theoretically, recurrent apneas are the product of a respiratory controller that has a high loop gain, intrinsically determined by both "plant gain" and "controller gain"[1]. Clinically recurrent apneas are observed in obstructive sleep apnea, Cheyne Stokes respiration, and opiod-induced central apnea, as well as Biot's breathing and RETT syndrome. In these conditions interactions occur between both gain elements, and both apnea types are initiated in the context of a reduction in respiratory drive. Recurrent apnea is a complex trait, where no one element is required or sufficient to produce the trait. Pathways involve both physiologic responses (e.g. response to alterations in gas exchange) and genetic predisposition[2]. Yet, characterizing the genetic architecture of respiratory control has been unexpectedly difficult. Some data arises from insights from pediatric disease, such as RETT and PHOX2b.[3-5] Finding a gene subsequently leads to diagnostic tests, identification of sub-clinical disease in relations, and prognostic the identification of gene mutations. Due to the gene homology for critical biologic functions among the mouse, rat and human, rodent studies that dissect lower level phenotypes like ventilatory stability in the response to hypoxia and re-oxygenation can be directly applied to understanding human physiology and disease[6]. Therefore, a phenotype of recurrent apneas, present in the C57BL/6J mouse and absent in the A/J mouse, is a naturally occurring genetic model of abnormal rhythmogenesis, exhibiting spontaneous pauses during resting breathing[7-8]. and recurrent apneas in reoxygenation following brief exposure to hypoxia[9]. These traits are enhanced by an inhibitor of neural nitric oxide synthase[10], and mitigated by acetazolamide[11], buspirone as a 5-HT agonist[12], and inhibitors of hydrogen sulfide production[13]. The traits are also abolished when chromosome 1 from the A/J mouse is introgressed into the B6 genome[7]. Thus, the appearance of recurrent apnea can be linked to naturally occurring variations in regions of the mammalian genome. The importance of this line of work relates to understanding the functional implications of genetic risk and human sleep apnea[14]. This would be a starting point for understanding mechanisms that sensitize (or desensitize) the control system in regard to ventilatory stability, and for drug discovery.

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INFLUENCE OF OPIOIDS AND ANESTHETICS ON THE CONTROL OF BREATHING AND UPPER AIRWAY OBSTRUCTION

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The three main topics that I will discuss are:

(1) Opioid effect on breathing versus analgesia and the development of utility functions. The pharmacodynamics of opioids, as described by C50, ke0 and parameter, differs with respect to different end-points such as miosis, respiratory depression and analgesia. For fentanyl most rapid effects are observed on the changes in pupil diameter followed by changes in respiratory depression and the development of analgesia. Utility functions, describing the probability of one effect relative to the probability of another effect, serves as a marker for behavior of the opioid in either the time or concentration domain. Utility functions of analgesia versus respiratory depression are a marker of the safety of the opioid. Utility functions are context sensitive and one example of such a function is $U = P(A > 50\%) - P(R > 50\%)$, where U is the utility function as function of time or concentration, A is the analgesic effect and R is respiratory depression.[1]

(2) Control of breathing in the postoperative patients after major abdominal surgery. There is ample evidence that opioids and anesthetics (and their combination) depress the control of breathing.[2] Little data is available on the behavior of the ventilatory control system to combined hypercapnic hypoxic stimuli (mimicking the changes in blood gasses upon the occurrence of an upper airway obstruction) in postoperative patients. Here I will discuss the effect of a mild asphyxic stimulus on ventilatory drive in patients following major abdominal surgery. Experiments were repeated 6 weeks after surgery, The data suggest that the asphyxic stimulus is depressed direct postoperatively but remains depressed for weeks. In contrast, the response to just hypercapnia is little affected. These data suggest a predominant effect of anesthesia and surgery (ie., the effect of opioid analgesics, anesthetics, surgery, stress and inflammation) within the peripheral chemoreflex loop that remains affected for some time following surgery.

(3) Upper airway obstruction in (i) volunteers receiving remifentanil and the combination remifentanil/propofol and (ii) OSAS and obsese patients in the first 3 postoperative nights. Some preliminary data will be presented on the effect of remifentanil on the control of breathing and upper airway patency. Next data will be presented on the occurrence of desaturations in OSAS, obese and control patients following major surgery.

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UNIQUE CONSIDERATIONS OF PATIENT WITH OBESITY HYPOVENTILATION SYNDROME

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Obesity Hypoventilation syndrome (OHS) is defined as the triad of obesity, daytime hypoventilation, and sleep-disordered breathing in the absence of an alternative neuromuscular, mechanical or metabolic explanation for hypoventilation. During the last three decades the prevalence of extreme obesity has markedly increased in the United States and other countries. With such global epidemic of obesity the prevalence of OHS is bound to increase. Patients with OHS have a lower quality of life with increased healthcare expenses and are at higher risk of developing pulmonary hypertension and early mortality compared to eucapnic patients with sleep-disordered breathing. OHS often remains undiagnosed until late in the course of the disease. Early recognition is important, as these patients have significant morbidity and mortality. Effective treatment can lead to significant improvement in patient outcomes, underscoring the importance of early diagnosis.

Given the typical morbid obesity and ventilatory derangements associated with this syndrome, patients with OHS could be at increased risk of postoperative respiratory failure.

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POSTER ABSTRACTS

POSTER VIEWING SCHEDULE

7:30 – 8:00am Poster Session Facilitators:

Group I: Dennis Auckley, in charge of posters 1-5,and 7.

Group II: Yandong Jiang, in charge of posters 8-14

Group III: Satya Krishna Ramachandran, in charge of 15-16, 20, 22,25,and 26.

Group IV: Jean Mantz, in charge of 28,30-33,35-36.

Group V: Ralph Lydic, in charge of 17-19,21,24,27,29,and 34.

1:00 – 1:30pm Poster Session Facilitators:

Group I: Yandong Jiang, in charge of posters 1-5,and 7.

Group II: John Loadsman, in charge of posters 8-14

Group III: Shiroh Isono, in charge of 15-16, 20,22,25,and 26.

Group IV: Jeremy Weingarten, in charge of 28,30-33,35-36.

Group V: Max Kelz, in charge of 17-19,21,24,27,29,and 34.

1:30 – 2:00pm Presentation of Poster Awards

Yandong Jiang