

### Message from the President



**David Hillman, MBBS, FANZCA, FRCP (Edin), FRACP (Hon)**  
*President, SASM  
 Head, Dept of Pulmonary Physiology and Sleep Medicine Director, West Australian Sleep Disorders Research Institute, Sir Charles Gairdner Hospital, Perth, Australia*

### Pain, Sleep and the Society of Anesthesia and Sleep Medicine

“State” refers to a mode or condition of being. Anesthesia and sleep are examples. As state-based (rather than organ-based) medical specialities, the considerations of anesthesiology and sleep medicine are broad, encompassing all organ systems. While there is much other common ground between them, some of the most important concerns shared by the two specialties relate to a third state – the state of pain.

Perioperative pain disturbs sleep and ventilation. Controlling it requires strategies that either circumvent the need for opioids and other sedating drugs or, where they are used, ensure that analgesia is achieved without

undue depression of respiration or arousal responses.

The relationship between pain and disturbed sleep is bi-directional. Pain disturbs sleep and disturbed sleep appears to exacerbate pain, probably through central sensitization [1]. Fibromyalgia is a condition that appears to involve such a mechanism, with neuropathic pain a common feature and sleep disturbance ubiquitous [2,3]. Significant insomnia is seen in over half of patients with chronic back pain and almost 90% of pain patients have at least one sleep complaint [4,5]. Pain complaints and sensitivity to heat stimuli increase in sleep-restricted volunteers, effects reversed by catch up sleep [6,7]. Self-reported short sleepers have reduced

thresholds to noxious stimuli [8].

Interestingly, melatonin - the hormone of sleep – appears to have a positive impact on pain. It may help reduce pain and sleep disturbance in fibromyalgia and other chronic pain syndromes [9]. There is some evidence to suggest management of sleep disorders may improve pain symptoms [10].

Almost 80% of patients undergoing chronic opioid treatment for non-ma-

*President’s Message  
 continued on page 11*

### In This Issue

- President’s Message..... 1, 11
- Editor’s File ..... 2
- Obstructive Sleep Apnea ...3, 5, 7
- SAMBA Patient Selection  
 Consensus..... 4-5
- Perioperative CPAP  
 Adherence..... 6-7
- Post-Adenotonsillectomy Pain  
 Management..... 8-10
- SASM Annual Conference  
 Workshops Agenda ..... 12-13
- SASM Officers & Board of  
 Directors ..... 14
- SASM Member Benefits ..... 14



## Editor's File

**Girish P. Joshi, MBBS, MD, FFARCSI**

*Editor*

*Professor of Anesthesiology and Pain Management*

*University of Texas Southwestern Medical Center*

*Dallas, Texas USA*



### SASM: The Road To Improve Management of Patients With Sleep Disorders

As is obvious from the wide range of topics covered in the SASM Newsletter, sleep disorders influence almost every aspect of our health. David Hillman, MBBS discusses the relationship between sleep and pain. Similarly, it is well recognized that Obstructive Sleep Apnea (OSA) and type 2 diabetes are closely related. Although OSA is implicated in development of type 2 diabetes, there are suggestions that type 2 diabetes might induce OSA. Jason O'Neal, MD and Eswar Sundar, MD provide us with an update on the relation between OSA and type 2 diabetes.

Over the past two decades there is a move of surgical procedures from an inpatient setting to an outpatient setting. Thus, it is not surprising that an increasing number of patients with OSA are being scheduled for ambulatory surgery. However, this remains controversial due to concerns of increased perioperative complications. In this issue, I discuss the recent Society for Ambulatory Anesthesia (SAMBA) consensus statement regarding approach to selection of OSA patients for ambulatory surgery.

As emphasized in the SAMBA recommendations, one of the major factors in improving postoperative outcome in OSA patients is the use of Continuous Positive Airway Pressure (CPAP) therapy, which is an insurance against life threatening apnea in the face of lack of arousal due to residual anesthetics, residual muscle paralysis and opioids. However, as discussed by Babak Mokhlesi, MD, MSc, the adherence to CPAP therapy in the perioperative period is a major concern. Thus, there is an urgent need to identify the barriers to CPAP use. In the mean time, one of the steps that could help is education of the patient and their caregivers.

It is now clear that an optimal analgesic technique would include use of combinations of analgesic with different mechanisms of action. Because the origin and consequences of pain are procedure-specific, the type and number of analgesics used should depend upon the surgical procedure (Joshi GP, Kehlet H: *Anesthesiology* 2013;118:780-2). Therefore, it is necessary to develop evidence-based, procedure-specific protocols. The PROSPECT working group, which consists of anesthesiologists and surgeons, has published web-based, procedure-specific pain management guidelines

for over a decade ([www.postoppain.org](http://www.postoppain.org)). Kimmo Murto, MD, FR-CPC and Christine Lamontagne, MDCM, FRCPC provide a comprehensive review on options for pain management in children undergoing adenotonsillectomy.

In recent years the literature related to sleep disorders is increasing exponentially. However, making some sense of these publications can be challenging. Members of SASM review this literature and provide a summary analysis ([www.SASMHq.org](http://www.SASMHq.org)). In addition, the topics presented during the SASM Annual Meeting cover clinically important and controversial aspects of care of patients with sleep disorders. This meeting also provides an opportunity for researchers to discuss their data with experts from around the world. Furthermore, SASM is offering a research grant geared towards improving our understanding of management of patients with sleep disorders. Overall, SASM plays a significant role in improving care of patients with sleep disorders.

I look forward to seeing you at the SASM Annual Meeting held in San Francisco, CA from October 10-11, 2013. ❖



**Jason O'Neal, MD**  
 Resident in Anesthesiology  
 Beth Israel Deaconess Medical Center  
 Harvard Medical School  
 Boston, USA



**Eswar Sundar, MD**  
 Director of Postanesthesia  
 Care Unit  
 Beth Israel Deaconess  
 Medical Center  
 Harvard Medical School  
 Boston, USA

## Obstructive Sleep Apnea and Diabetes Mellitus: The Perfect Storm?

How often do your patients have the comorbidities of diabetes and Obstructive Sleep Apnea (OSA)? Or better yet, how often do they have uncontrolled diabetes and are noncompliant with treatment for their OSA? If you care for patients in a major medical center, you may have several of these patients daily. Now, what if you were told that treatment of OSA might prevent and even treat type 2 diabetes (DM2)? A few studies have started to look for a link and are suggesting this statement may have some truth to it.

Diabetes and OSA are associated with high rates of morbidity, mortality and increased health care costs. Almost 26 million Americans suffer from DM2, and 1.9 million are newly diagnosed every year. These numbers continue to increase with an estimated 79 million classified as pre-diabetics in the United States today. Many people also suffer from OSA, with the prevalence in the obese ranging from 11-44% in women and 33-77% in men, while the prevalence of OSA in diabetics is estimated at

71%. This suggests that up to 19 million diabetics may have undiagnosed and untreated OSA [1]. The numbers affected by these disease processes are staggering and cannot be ignored.

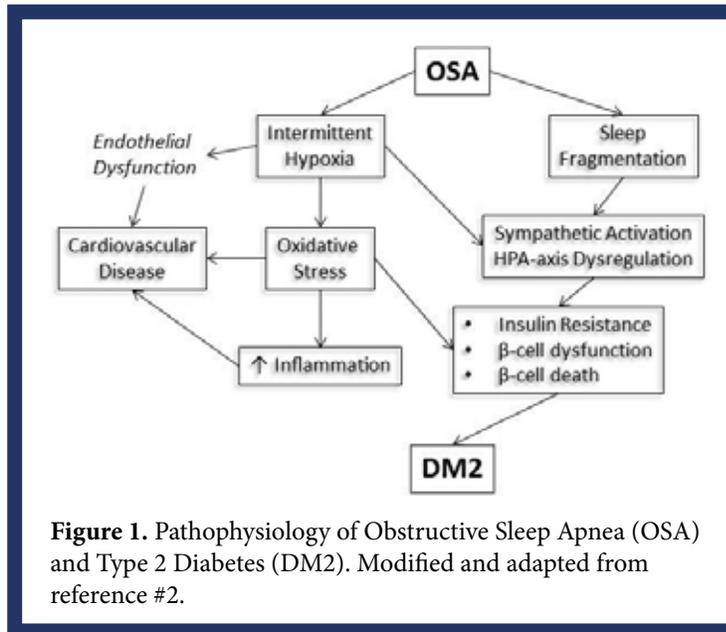
Multiple studies have shown OSA to be related to insulin resistance, glucose intolerance and DM2, al-

changes in inflammatory pathways, hypoxic injury to the pancreas and alteration in central pathways for glucose control causing insulin resistance and pancreatic  $\beta$ -cell destruction and death (Figure 1) [2]. Future studies are needed to confirm these possibilities.

As for clinically relevant studies, the results are varied at this time. A recent meta-analysis found that people with moderate-to-severe OSA were associated with a greater risk of developing DM2 [3]. This meta-analysis included six prospective cohort studies and found a relative risk for developing diabetes of 1.63 (95% confidence interval (CI) 1.09–2.45) in people with moderate-to-severe OSA. A relative risk of 1.22 (95% CI 0.91–1.63) for people with mild OSA was not statistically significant.

Several limitations were noted in the study, including different definitions of OSA, different methods for diabetes diagnosis and confounders differed in each study.

Studies are underway assessing the impact of positive airway pres-



**Figure 1.** Pathophysiology of Obstructive Sleep Apnea (OSA) and Type 2 Diabetes (DM2). Modified and adapted from reference #2.

though the mechanism for this remains unknown. Potential theories include hypoxia and sleep fragmentation from OSA, leading to insults in glucose metabolism by activating the sympathetic nervous system and Hypothalamic-Pituitary-Adrenal (HPA) axis. This may cause

“Obstructive Sleep Apnea and Diabetes” continued on page 5



**Girish P. Joshi, MBBS, MD, FFARCSI**

*Professor of Anesthesiology and Pain Management*

*University of Texas Southwestern Medical Center, Dallas, Texas*

## SAMBA Patient Selection Consensus Statement on Selection of Adult OSA Patients For Ambulatory Surgery

The suitability of ambulatory surgery in patients with Obstructive Sleep Apnea (OSA) remains controversial because of the concerns of increased perioperative complications. In 2006, the American Society of Anesthesiologists (ASA) published practice guidelines for management of surgical patients with OSA, including patient selection for ambulatory surgery [1, 2]. Since the publication of the ASA practice guidelines, several studies have been published assessing perioperative complications after ambulatory surgery in OSA patients, including those undergoing laparoscopic bariatric surgery and upper airway surgery [1]. A systematic review of published literature, evaluating the perioperative complications in OSA patients undergoing ambulatory surgery, assessing preoperative factors that may influence the perioperative outcome (e.g. severity of OSA, co-existing medical

conditions and invasiveness of the surgical procedure) was performed [2]. Compared with non-OSA patients, OSA patients had a higher body mass index (BMI) and more

of factors, particularly co-existing medical conditions and the use of opioids. Patients with non-optimized comorbid medical conditions may not be good candidates for ambulatory surgery [2].

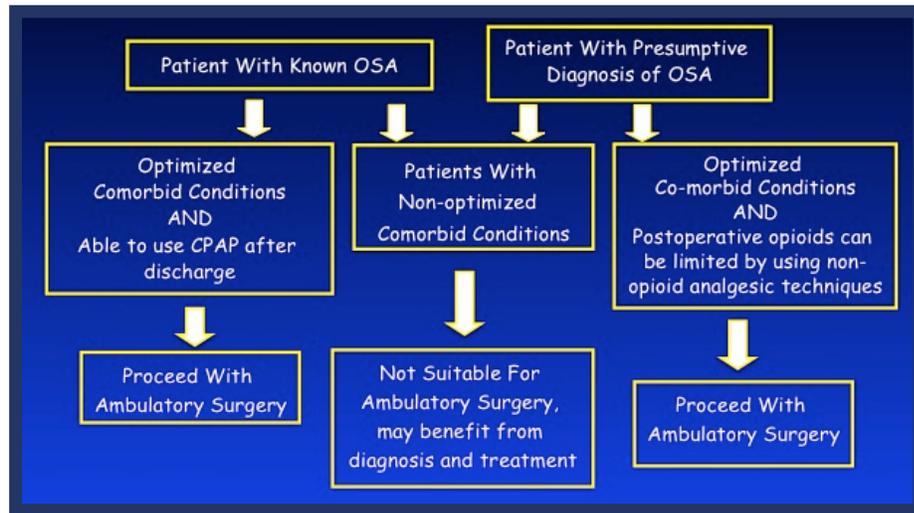
Patients with a known diagnosis of OSA and optimized comorbid medical conditions can be considered for ambulatory surgery if they are able to use a CPAP device in the postoperative period, because in the included studies,

a majority of the OSA patients used CPAP or BiPAP postoperatively, which may have contributed to a safe perioperative course. Patients who are unable or unwilling to use CPAP after discharge may not be appropriate for ambulatory surgery.

co-morbidities including diabetes, hypertension, stroke, myocardial infarction and congestive heart failure). The studies evaluating perioperative outcome in OSA patients undergoing ambulatory surgery are sparse.

In patients with an established diagnosis of OSA (either by a sleep study or presumptive diagnosis), an adverse perioperative outcome is associated with a complex interplay

Patients with a presumed diagnosis of OSA, based on screening tools such as the STOP-Bang questionnaire and optimized comorbid conditions, can be considered for



**Figure 1:** Decision making in preoperative selection of a patient with Obstructive Sleep Apnea scheduled for ambulatory surgery (modified from Joshi GP et al: *Anesth Analg* 2012; 115: 1060-8).

*“SAMBA Patient Selection Consensus Statement” continued on next page*

most types of ambulatory surgery if postoperative pain relief can be provided predominantly with non-opioid analgesic techniques, because opioids have a significant propensity to exacerbate OSA and prevent arousal. No guidance could be provided for OSA patients undergoing upper airway surgery due to limited evidence.

There was significant emphasis on the need for education of surgeons, patients and their family (or caregivers) regarding the need for increased vigilance after discharged home. Patients on preoperative CPAP should be advised to use their CPAP device for several days postoperatively, as the potential risks can last for several days

**Preoperative Considerations:**

- Comorbid medical conditions include hypertension, arrhythmias, heart failure, cerebrovascular disease, and metabolic syndrome.
- If OSA is suspected during the preoperative evaluation, one could proceed with a presumptive diagnosis of OSA albeit with caution.
- Educate surgeon, patient and family.

**Intraoperative Considerations:**

- Use non-opioid analgesic techniques, when possible.

**Postoperative Considerations:**

- Exercise caution in OSA patients who develop prolonged and frequent severe respiratory events (e.g., sedation analgesic mismatch with opioids, desaturation, and apneic episodes) in the postoperative period.

**Figure 2:** Perioperative considerations in patients with Obstructive Sleep Apnea scheduled for ambulatory surgery.

after surgery. In addition to the usual nocturnal CPAP use, patients should be advised to use CPAP whenever sleeping, even during the daytime. Also, patients should be advised against sleeping in the supine position. Patients who are assumed to have OSA based on the

screening questionnaire should be advised to follow-up with their primary physician for possible sleep study.

Finally, the deleterious effects of opioids must be emphasized and patients should be asked to limit opioid use. ❖

**References:**

- 1 Joshi GP, Ankichetty SP, Gan TJ, Chung F. Society for Ambulatory Anesthesia (SAMBA) consensus statement on preoperative selection of adult patients with obstructive sleep apnea scheduled for ambulatory surgery. *Anesth Analg* 2012; 115: 1060-8.
- 2 Gross JB, Bachenberg KL, Benumof JL et al, American Society of Anesthesiologists Task Force on Perioperative Management. Practice guidelines for the perioperative management of patients with obstructive sleep apnea: a report by the American Society of Anesthesiologists Task Force on Perioperative Management of patients with obstructive sleep apnea. *Anesthesiology* 2006; 104: 1081-93.

“Obstructive Sleep Apnea and Diabetes” continued from page 3

sure in OSA on glycemic control. In a case-crossover study examining the use of Continuous Positive Airway Pressure (CPAP) in DM2 patients with OSA, the mean sleeping glucose decreased from 122 to 102 (p=0.03) with CPAP treatment and glucose levels found to be less variable. However, no difference in hemoglobin A1c was seen (7.2% to 7.1%) [4]. Another study investigating positive airway pressure on glycemic control found no difference in either fasting glucose or hemoglobin A1c in DM2 patients [5]. A larger, randomized controlled trial utilizing sham-CPAP and effective-CPAP arms would provide further insight on glycemic control in DM2 patients

with OSA. Are only patients with more advanced stages of OSA at a greater risk for DM2? Should patients with DM2 and OSA be encouraged to use CPAP for a few weeks before surgery to optimize glycemic control? Although studies suggest a link between OSA and DM2 and possible benefit from positive airway pressure, the current evidence is inconsistent and limited. Also, a potential confounder for these studies is obesity [6]. While some studies looking at OSA and DM2 have accounted for obesity, others have not. OSA and DM2 are disorders affecting large numbers of patients.

Evidence is starting to accumulate suggesting associations and interactions between the two conditions that may have the makings of a ‘perfect healthcare storm’. There is more to come, but in the meantime, beware of these comorbidities in your patients. ❖

**References**

- 1 Pamidi S, Tasali E. Obstructive sleep apnea and type 2 diabetes: is there a link? *Front Neurol*. 2012;3:126. Epub 2012.
- 2 Ioja S, Weir ID, Rennert NJ. Relationship between sleep disorders and the risk for developing type 2 diabetes mellitus. *Postgrad Med*. 2012;124:119-29.

“Obstructive Sleep Apnea and Diabetes” continued on page 7



**Babak Mokhlesi, MD, MSc**

*Director, Sleep Disorders Center and Sleep Fellowship Program*

*Section of Pulmonary and Critical Medicine*

*Department of Medicine, University of Chicago, Chicago, IL*

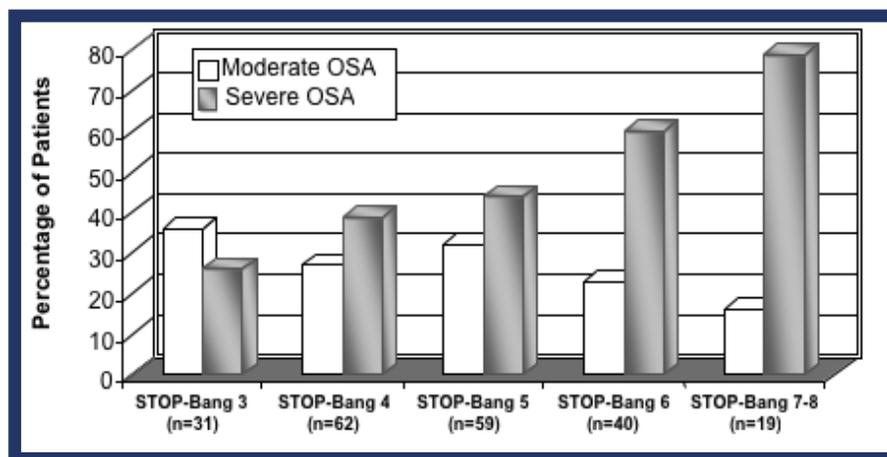
## Perioperative CPAP Adherence in Patients With Newly Diagnosed Obstructive Sleep Apnea

Since its initial description more than 40 years ago, Obstructive Sleep Apnea (OSA) has been gaining more interest among anesthesiologists and there is accumulating evidence that moderate-to-severe OSA can increase perioperative complications [1-3]. Given the important implications of untreated OSA [4-9], the American Society of Anesthesiologists (ASA) and the Society for Ambulatory Anesthesia (SAMBA) recommends screening surgical patients for OSA and implementing treatment if significant OSA is present [10, 11]. Although most studies have focused on screening and diagnosis of OSA, less is known about CPAP therapy and adherence during the perioperative period.

Recent data from our group and the Mayo Clinic underscores the challenges patients and clinicians face in achieving adequate CPAP adherence in the immediate postoperative period [12, 13]. Specifically,

we were interested in objectively quantifying perioperative CPAP adherence and optimal CPAP pressure settings in a cohort of patients diagnosed preoperatively with moderate or severe OSA.

Of the 211 patients who screened high risk for OSA and underwent a diagnostic PSG, 6% did not have OSA, 20% had mild OSA, 28% had moderate OSA and 46% had severe OSA. However, the prevalence of severe OSA increased from 36%



**Figure 1:** Prevalence of moderate or severe OSA across all STOP-Bang scores in 211 patients who underwent in-laboratory polysomnogram. The 5 patients with STOP-Bang score of 8 were combined with the 14 patients with STOP-Bang score of 7.  $p = 0.02$  by  $\chi^2$  comparing OSA severity in various STOP-Bang categories.

Over a 2-year period, 431 patients who screened at high risk for OSA were referred for an in-laboratory polysomnogram (PSG). Of the 431 referred patients, 211 scheduled and completed the in-laboratory PSG, while the remainder of the patients did not show up for the scheduled PSG. None of the patients underwent a sleep consultation prior to the diagnostic PSG.

in patients with STOP-Bang score of 3 to 79% in patients with STOP-Bang scores of 7-8 (Fig 1).

CPAP therapy was required and provided to a total of 138 out of 211 patients (65%) and objective CPAP adherence data during the first 30 days of therapy was available in 104 of the 138 patients who received CPAP therapy (75%). On average, patients were started on CPAP therapy 4 days before the date of surgery. The overall median adherence to CPAP during the first 30 days of therapy was suboptimal at 2.5 h/night (interquartile range of 0.7-4.5 h/night), and there was no significant difference in CPAP adherence between the 2 STOP-Bang

*“Perioperative CPAP Adherence” continued on next page*

categories. Based on this distribution, only 25% of patients prescribed CPAP therapy were using their CPAP devices for  $\geq 4.5$  h/night. As expected, adherence was also poor when examined as a categorical variable since only 33% of patients used CPAP  $\geq 4$  h/night. In a fully adjusted linear regression model, African American race, male gender and the presence of depressive symptomatology were each independently associated with approximately 1 h less of CPAP use per night after adjustment for OSA severity, STOP-Bang category, hypersomnolence and education level. We also found that the majority of patients were optimally treated with CPAP pressure of  $9 \pm 2$  cm H<sub>2</sub>O, and there was no significant difference in the optimal CPAP pressure between the 2 STOP-Bang groups.

Our study demonstrates that in an urban tertiary care academic medical center, overall CPAP adherence during the first 30 days of perioperative period was extremely low compared to known CPAP adherence at our institution, as well as reported national averages of 4.7 hours/night [18]. Our data also demonstrates that the average CPAP level required to optimally treat patients is approximately  $9 \pm 2$  cm H<sub>2</sub>O. Therefore an auto-titrating CPAP device with a minimum pressure setting of 7

cm H<sub>2</sub>O and a maximum pressure setting of 12 cm H<sub>2</sub>O adequately treated the vast majority of our patients.

In summary, the adherence to prescribed CPAP therapy in the perioperative period in our population was extremely low. Further research is needed to identify barriers to CPAP adherence in this patient population, or efforts directed towards diagnosis are likely to be wasted. ❖

### References:

- 1 Kaw R, Pasupuleti V, Walker E, et al. Postoperative complications in patients with obstructive sleep apnea. *Chest* 2012;141:436-41.
- 2 Memtsoudis S, Liu SS, Ma Y, et al. Perioperative pulmonary outcomes in patients with sleep apnea after noncardiac surgery. *Anesth Analg* 2011;112:113-21.
- 3 Flink BJ, Rivelli SK, Cox EA, et al. Obstructive sleep apnea and incidence of postoperative delirium after elective knee replacement in the nondemented elderly. *Anesthesiology* 2012;116:788-96.
- 4 Campos-Rodriguez F, Martinez-Garcia MA, de la Cruz-Moron I, et al. Cardiovascular mortality in women with obstructive sleep apnea with or without continuous positive airway pressure treatment: a cohort study. *Ann Intern Med* 2012;156:115-22.
- 5 Gami AS, Howard DE, Olson EJ, Somers VK. Day-night pattern of sudden death in obstructive sleep apnea. *N Engl J Med* 2005;352:1206-14.
- 6 Punjabi NM, Caffo BS, Goodwin JL, et al. Sleep-disordered breathing and mortality: a prospective cohort study. *PLoS Med* 2009; 6:e1000132.
- 7 Young T, Finn L, Peppard PE, et al. Sleep-disordered breathing and mortality: eighteen-year follow-up of the Wisconsin sleep cohort. *Sleep* 2008;31:1071-8.
- 8 Somers VK, White DP, Amin R, et al. Sleep apnea and cardiovascular disease: an American Heart Association/American College of Cardiology Foundation Scientific Statement from the American Heart Association Council for High Blood

Pressure Research Professional Education Committee, Council on Clinical Cardiology, Stroke Council, and Council on Cardiovascular Nursing. *J Am Coll Cardiol* 2008;52:686-717.

- 9 Marin JM, Carrizo SJ, Vicente E, Agusti AG. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. *Lancet* 2005;365:1046-53.
- 10 Gross JB, Bachenberg KL, Benumof JL, et al. Practice guidelines for the perioperative management of patients with obstructive sleep apnea: a report by the American Society of Anesthesiologists Task Force on Perioperative Management of Patients with Obstructive Sleep Apnea. *Anesthesiology* 2006;104:1081-93.
- 11 Joshi GP, Ankichetty S, Chung F, Gan TJ. Society for Ambulatory Anesthesia (SAMBA) consensus statement on preoperative selection of patients with obstructive sleep apnea scheduled for ambulatory surgery. *Anesth Analg* 2012;115:1060-8.
- 12 Guralnick AS, Pant M, Minhaj M, et al. CPAP Adherence in patients with newly diagnosed obstructive sleep apnea prior to elective surgery. *J Clin Sleep Med* 2012; 8:501-6.
- 13 O’Gorman SM, Morgenthaler TI, Gay PC. Does auto-titrating positive airway pressure therapy improve postoperative outcome in patients at risk for obstructive sleep apnea syndrome? A randomized controlled clinical trial. *Chest* 2012; (In press).
- 14 Pamidi S, Knutson KL, Ghods F, Mokhlesi B. The impact of sleep consultation prior to a diagnostic polysomnogram on continuous positive airway pressure adherence. *Chest* 2012; 141:51-7.
- 15 Kribbs NB, Pack AI, Kline LR, et al. Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. *Am Rev Respir Dis* 1993; 147:887-95.
- 16 Engleman HM, Martin SE, Douglas NJ. Compliance with CPAP therapy in patients with the sleep apnoea/hypopnoea syndrome. *Thorax* 1994;49:263-6.
- 17 Reeves-Hoche MK, Meck R, Zwillich CW. Nasal CPAP: an objective evaluation of patient compliance. *Am J Respir Crit Care Med* 1994;149:149-54.
- 18 Sawyer AM, Gooneratne NS, Marcus CL, et al. A systematic review of CPAP adherence across age groups: clinical and empiric insights for developing CPAP adherence interventions. *Sleep Med Rev* 2011;15:343-56.

### “Obstructive Sleep Apnea and Diabetes” continued from page 5

- 3 Wang X, Bi Y, Zhang Q, et al. Obstructive sleep apnoea and the risk of type 2 diabetes: A meta-analysis of prospective cohort studies. *Respirology*. Epub 2012 Sep 18.
- 4 Dawson A, Abel SL, Loving RT, et al. CPAP therapy of obstructive sleep

apnea in type 2 diabetics improves glycemic control during sleep. *J Clin Sleep Med*. 2008;4:538-42.

- 5 Prasad B, Carley DW, Krishnan JA, et al. Effects of positive airway pressure treatment on clinical measures of hypertension and type 2 diabetes. *J Clin*

*Sleep Med*. 2012;8:481-7.

- 6 Tasali, Mokhlesi B, Van Cauter E, et al. Obstructive sleep apnea and type 2 diabetes: interacting epidemics. *Chest*. 2008. 133:496-506.



**Kimmo Murto MD, FRCPC**  
 Department of Anesthesiology  
 Children's Hospital of Eastern Ontario  
 Director of Research  
 Assistant Professor of Anesthesia  
 University of Ottawa, Canada



**Christine Lamontagne, MDCM, FRCPC**  
 Department of Anesthesiology  
 Children's Hospital of Eastern Ontario  
 Director of Chronic Pain Services  
 Assistant Professor of Anesthesia  
 University of Ottawa, Canada

## Post-Adenotonsillectomy Pain Management in Children: Can We Do it Better?

**A**denotonsillectomy (T&A) is the most common surgical procedure performed in North America [1]. The most common indication for T&A is suspicion for Obstructive Sleep Apnea (OSA). However, less than 10% of these children have a definitive OSA diagnosis [2,3]. Children experience significant pain and severe functional limitation for 7 days after T&A [4,5]. Poor pain control may lead to increased negative behavior and hospital visits, impaired food intake, dehydration, sleep disturbance and risk of secondary hemorrhage [6-8]. Reasons for variable pain control are multiple and include preoperative anxiety, perioperative fasting and hydration status, postoperative emesis, drug pharmacokinetics and dynamics, use of local anesthetic infiltration and surgical technique. A parent's ability to assess pain and their willingness to administer an analgesic are essential for effective pain management [9-17]. The focus of this review will be on current pharmacologic approaches to post T&A pain management in children and how pharmacogenomics provides insight into variable drug efficacy and related adverse events.

### Opioids

Opioids, including codeine, are commonly administered to provide postoperative pain relief. However, codeine has been associated with life-threatening events or death in children less than 6 years of age due to relative overdose and/or CYP2D6 extensive or ultra-rapid metabolizing status [10,18,19]. It is a pro-drug that requires conversion to morphine by the liver enzyme isoform CYP2D6 [20]. Genetic polymorphism of this enzyme can lead to a wide and unreliable spectrum of analgesic effects after appropriate dosing. Ultra-rapid conversion to morphine, secondary to CYP2D6 gene duplication, can lead to dangerously high levels of morphine (up to 10% and 29% of European and North African descendants respectively) or the inability to convert codeine to morphine ("poor metabolizer," ineffective analgesic for 6-10% of Caucasians) [21,22]. There is debate in the literature regarding its role as an analgesic in children [23]. Many hospitals, including our own, have removed codeine from their formulary. It is telling that the World Health Organization has replaced codeine with morphine as part of a two-step strategy to treat moderate to severe pain in children [24].

In children aged 2-18 years, administering an oral morphine elixir 100-300 mcg/kg (maximum 10 mg) every 3-4 hours, as required, has worked well in our own hospital. In the event of morphine allergy or intolerance, hydromorphone syrup 20-60 mcg/kg every 3-4 hours can be substituted. Interestingly, morphine dosage and plasma concentration do not directly correlate with analgesia [25]. Both genetic and non-genetic factors contribute to the unpredictability. Genetic factors associated with morphine resistance include variation in genes for the mu receptor (e.g. gene OPRM-1 polymorphism A118G), metabolizing enzymes (e.g. gene UGT2B7 polymorphism A-842G) and blood brain barrier/gastrointestinal tract drug transporters (e.g. gene ABCB1 polymorphism C3435T) [26,27]. To further complicate matters, the prevalence of OSA in the T&A population can be as high as 32% [28,29]. Recurrent hypoxemia from OSA can lead to up-regulation of central mu receptors and increased sensitivity to the respiratory depressant effects of opiates [30,31]. Limited preoperative identification makes it challenging to appropriately adjust perioperative opioid dosing. Children have likely suffered anoxic events and death as a result of this interac-

*"Post-Adenotonsillectomy Pain Management" continued on next page*

tion [32-34]. Compared to adults, children have a two-fold increase in fatal respiratory events post T&A; these events account for the majority of death and major brain injury [35,36]. Guidelines for morphine administration in the presence of moderate to severe OSA should include appropriate monitoring, a 50% dose reduction, avoid “around-the-clock” dosing and combine with non-opioid adjunctive analgesics.

Alternative opiates, including hydrocodone, oxycodone and tramadol, are metabolized by the CYP2D6 enzyme and subject patients to unpredictable adverse events and variation in analgesic response similar to codeine [23, 37-39]. Tapentadol, a new mu opioid agonist and a norepinephrine reuptake inhibitor is not affected by CYP2D6 enzyme activity and is being evaluated in children.

### **Non-steroidal Anti-inflammatory Drugs (NSAIDs)**

In children, post T&A bleeding is a relatively common complication (3% prevalence) and accounts for approximately one third of T&A mortality [35,36]. NSAIDs effectively treat mild to moderate pain and are used as an adjunct for severe pain. They are more effective when administered as prophylaxis for pain rather than the treatment of established pain. Most NSAIDs non-selectively inhibit both COX-1 and COX-2. It is unknown to what degree the inhibition of one or both of the COX enzymes is required to achieve maximal analgesia [40]. Controversy surrounds NSAIDs

and their association with increased post T&A bleeding and/or need for repeat surgical intervention [41-43]. Interpretation of results requires caution because analysis of multiple small studies does not achieve an overall sample size sufficient for a definitive conclusion [41,43]. However, ketorolac (predominately a COX-1 inhibitor) appears to increase post-operative bleeding and is contraindicated [44]. Diclofenac, considered a weak COX-2 inhibitor, has been shown to be efficacious but its relation to post T&A bleeding is unclear [41]. COX-2 specific inhibitors are devoid of anti-platelet effects [45]. Some advocate starting ibuprofen on postoperative day two while a recent guideline is indifferent to its timing [34,44]. A COX-2 specific inhibitor would appear to be an ideal co-analgesic. Celecoxib is the only true COX-2 specific inhibitor available [46]. Unfortunately, pediatric literature in a perioperative setting does not exist.

### **Acetaminophen**

Acetaminophen alone may not provide adequate analgesia. The combination of acetaminophen with NSAIDs makes for a powerful combination [23,44]. Scheduled dosing of acetaminophen is effective, but the correct mix, dosing and timing of NSAIDs in addition to acetaminophen is unknown. Oral acetaminophen 30 mg/kg followed by a daily maximum of 4 grams or 100 mg/kg/day is effective [50,51]. Administration of intra-operative rectal acetaminophen results in unpredictable bioavailability [52]. The role of intravenous acetaminophen

for pediatric T&A patients has yet to be determined [53,54].

### **Steroids**

A Cochrane review of 19 pediatric studies (1756 children) evaluated a Single Intra-operative Dose (SID) of dexamethasone (0.15-1.0 mg/kg, maximum dose 8-25 mg) within 24 hours post T&A and reported pain reduction of 1.07 cm (from 4.72 to 3.65) on a 10 cm VAS scale [55]. A similar degree of pain reduction was shown with higher doses of dexamethasone (0.4-1.0 mg/kg; maximum 8-50 mg) [56]. In an adult meta-analysis there was no difference in postoperative analgesia or opioid use between intermediate (0.11-0.2 mg/kg) and high dose (> 0.21 mg/kg) dexamethasone [57]. A SID of dexamethasone 0.24 mg/kg (0.17-0.34 mg/kg) combined with a reduced dose of morphine 0.1 mg/kg (0.06-0.12 mg/kg) was found to significantly reduce post T&A respiratory events in children with severe OSA [58]. A SID of steroid has been associated with an increased incidence of post-T&A re-intervention and bleeding events [59,60]. However, inadequate sample size and the presence of confounding variables, including the concomitant use of NSAIDs, repeated doses of steroids and the inclusion of surgical related bleeding casts doubt on this concern. The optimal dose for a SID of dexamethasone appears to be within the 0.11-0.25 mg/kg range. The maximum dose is unknown.

### **Ketamine**

Ketamine has a role in perioperative pain management in children.

When 0.5 mg/kg was administered intravenously or topically at the time of tonsillectomy, a decrease in post-operative pain scores was shown without additional nausea, vomiting or psychomimetic effects [61]. A study of 60 children undergoing tonsillectomy concluded that ketamine, as an adjunct to fentanyl, improved post-operative analgesia without delaying discharge [62].

### **Alpha-2 Agonists**

Dexmedetomidine is not approved by the FDA for pediatric use, but appears to be a useful analgesic adjunct [63]. Attractive qualities include facilitated arousal, maintenance of a patent airway and minimal respiratory depression, ideal for the T&A population. Bradycardia and hypotension occur, but are not clinically relevant at doses < 1 mcg/kg. Higher doses are well tolerated. Intra-operative dexmedetomidine 1 mcg/kg and morphine 100 mcg/kg have comparable postoperative morphine sparing effects and time to discharge readiness [64]. It provides superior postoperative analgesia to fentanyl and reduces the incidence of emergence agitation. However, it may prolong length of stay in the post anesthesia care unit [65,66]. A single dose combined with ketamine and/or a reduced dose of morphine provides safe and effective analgesia for post T&A patients [67].

### **Summary**

Pain management in the post-T&A child needs to be safe, multimodal in approach, patient-specific and involve the parents. Appropriate post-operative monitoring is essential for children with moderate to severe OSA. A SID of steroid com-

bined with morphine and “around-the-clock” acetaminophen seems to be a reasonable analgesic approach. Ketorolac administration is contraindicated. The role of the other NSAIDs, including COX-2 specific inhibitors, is unclear. Pharmacogenomics of analgesics will bring us closer to our goal of personalized pain medicine. However, all is lost if we do not teach and provide support for parents to appropriately administer analgesics to their children. ❖

### **References**

- 1 Erickson BK et al. Otolaryngol Head Neck Surg 2009; 140:894.
- 2 Mitchell R et al. Laryngoscope 2006; 116: 956.
- 3 Murto K. Society for Ambulatory Anesthesia Newsletter 2010; 25:6.
- 4 Stewart D, et al. Pediatr Anaesth 2012; 22: 136.
- 5 Howard K, et al. Pediatr Anaesth 2010; 20: 913.
- 6 Kotiniemi LH, et al. Pediatr Anaesth 1996; 6:45.
- 7 Sommer M et al. Clin J Pain 2010; 26:87.
- 8 Colclasure JB, et al. Ear Nose Throat J 1990; 69:155.
- 9 Johr M. Curr Opinion Anaesth 2006; 19: 260.
- 10 Kelly LE, et al. Pediatrics 2012; 129: e1343.
- 11 Kain ZN, et al. Pediatrics 2006; 118: 651.
- 12 Engelhardt T, et al. Pediatr Anaesth 2011; 21: 964.
- 13 Heshmati F, et al. IJMS 2004;29: 72.
- 14 Blakeslee DB, et al. Laryngoscope 1997; 107: 1332.
- 15 Alexiou VG, et al. Arch Otolaryngol Head Neck Surg 2011; 137: 558.
- 16 Fortier MA, et al. Pediatrics 2009; 124: 588.
- 17 Grainger J, et al. Clin Otolaryngol 2008; 33: 411.
- 18 Voronov P, et al. Pediatr Anaesth 2007; 17: 684.
- 19 Ciszkowski C, et al. N Engl J Med 2009; 361: 827.
- 20 Caraco Y. New Engl J Med 2004; 351: 2867.
- 21 Williams DG, et al. Br J Anaesth 2002; 89: 839.
- 22 Palmer SN, et al. Anesthesiology 2005; 102:663.
- 23 Tremlett M, et al. Pediatr Anaesth 2010; 20: 183.
- 24 [http://www.who.int/medicines/areas/quality\\_safety/guide\\_perspainchild/en/index.html](http://www.who.int/medicines/areas/quality_safety/guide_perspainchild/en/index.html)
- 25 Duedahl TH, et al. Pediatr Anaesth 2007; 17: 756.
- 26 Ventura MA, et al. Pharm Wellness Rev 2011; 2: 29.
- 27 Sia AT, et al. Anesthesiology 2008; 109: 520.
- 28 van Someren V et al. Clin Otolaryngol 1990; 15: 263.
- 29 Constantin E et al. Pediatrics 2010; 125: e162.
- 30 Brown KA, et al. Anesthesiology 2004; 100:806
- 31 Brown KA, et al. Anesthesiology 2006; 105:665.
- 32 Stevenson AN, et al. Laryngoscope 2012; 122: 71.
- 33 Simonsen AR, et al. Int J Pediatric Otolaryngol 2010; 74: 977.
- 34 Sadhasivam S. Pain Medicine 2012; 13: 982.
- 35 Morris LGT, et al. Otolaryngol Head Neck Surg 2008; 138: 315.
- 36 Windfuhr JP et al. Eur Arch Otorhinolaryngol 2010; 265: 1527.
- 37 Samer CF, et al. Br J Pharmacol 2010; 160: 919.
- 38 Bozkurt P. Pediatr Anaesth 2005; 15: 1041.
- 39 Gan SH, et al. Mol Diagn Ther. 2007;11:171.
- 40 Kokki H. Pediatric Drugs 2003; 5:103.
- 41 Moniche S et al. Anesth Analg 2003; 96:68.
- 42 Marret E et al. Anesthesiology 2003; 98:1497.
- 43 Cardwell M, et al. Cochrane Database Sys Rev 2005; (2): CD003591.
- 44 Baugh RF, et al. Otolaryngol Head Neck Surg 2011; 144 (IS): S1.
- 45 Leese PT, et al. J Clin Pharmacol 2000; 40: 124.
- 46 Gajraj N, et al. Anesth Clin N Am 2005; 23:49.
- 47 Bean Lijewski JD et al. Otolaryng Head Neck Surg 2007; 137:545.
- 48 Joshi W et al. Anesth and Analg 2003; 97:35.
- 49 Vallee E et al. J Otolaryngol 2007; 36:264.
- 50 Anderson BJ, et al. Anesthesiology 1999; 90: 411.
- 51 Berde CB, et al. N Engl J Med 2002; 347:1094.
- 52 Korpela R, et al. Anesthesiology 1999; 91: 442.
- 53 Buck ML. Pediatr Pharmacotherapy 2011; 17(4).
- 54 Capici F et al. Br J Anaesth 2008; 100: 251.
- 55 Steward DL, et al. Cochrane Database of Systematic Reviews 2011, Issue 8, Art. No.: CD003997
- 56 Afman CE, et al. Otolaryngol Head Neck Surg 2006: 134: 181.
- 57 De Oliveira GS Jr, et al. Anesthesiology. 2011; 115: 575.
- 58 Raghavendran S, et al. Anesth Anal 2010; 110: 1093.
- 59 Plante J, et al. Br Medical J 2012; 345: e5389 doi: 10.1136/bmj.e5389.
- 60 Czarnetzki C, et al. JAMA 2008; 300: 2621.
- 61 Dahmani S, et al. Pediatr Anaesth 2011;21: 636.
- 62 Elshammaa N, et al. Paediatr Anesth 2011;21: 1009.
- 63 Mason K, et al. Anesth Analg 2011; 113: 1129.
- 64 Olutoye OA, et al. Anesth Analg. 2010; 111: 490.
- 65 Pestieau SR, et al. Can J Anesth 2011; 58: 540.
- 66 Patel A, et al. Anesth Analg 2010; 111: 1004.
- 67 Yuen VM. Pediatr Anaesth 2010; 20: 256.

lignant pain have sleep disordered breathing, severely so in half of these [11,12]. It can be obstructive and/or central in nature. The central component may involve hypoventilation or breathing periodicity. Irregular (ataxic) breathing patterns are commonly seen during sleep in patients taking opioids. Opioid-related suppression of arousal responses increases vulnerability of such patients to prolonged events during sleep or sedation.

Non invasive positive airway pressure therapies have much to offer these patients during sleep: continuous positive airway pressure for Obstructive Sleep Apnea, bi-level ventilatory support for sleep hypoventilation and a newer mode – Adaptive Servo Ventilation (ASV) – for breathing periodicity. ASV increases pressure support during the waning/hypoventilatory phase of periodic breathing and decreases it during the waxing/hyperventilatory phase. This varying pressure support reduces the variability in ventilation which counteracts the self perpetuating source of the instability itself – the changing carbon dioxide set point for ventilation between the higher thresholds of sleep (which encourage hypoventilation) and the lower thresholds that accompany the arousals that are often seen during the hyperventilatory phase.

This is a rich mix of considerations which are of the first importance to anesthesiologists and to sleep physi-

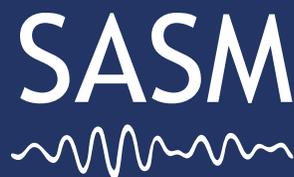
cians. Needless to say that makes them core business for the Society of Anesthesiology and Sleep Medicine. Acute aspects are going to receive close attention at our 2013 Annual Scientific Meeting on October 10th and 11th in San Francisco, immediately before the ASA Annual Meeting. Our meeting theme is “Opioids, Respiratory Depression and Sleep-Disordered Breathing (SDB): Peri-operative Implications.” We have assembled an expert faculty and the meeting will present a stimulating and relevant examination of these inter-related problems. Details are available on our website. Please note that Thursday will be devoted to workshops and Friday to the main body of the presentations so, for those of you who get in late, Friday is complete in itself. Remember also that we have our popular dinner on Thursday evening. All members are invited to attend this. This year it features Ralph Lydic, PhD as guest speaker, with Jane C.K. Fitch, MD, the ASA President-Elect, joining us as a special guest. She will also speak to us and it will be a great opportunity to meet and discuss our interests with her. We hope you will join us for this important meeting.



#### References:

- 1 Okifuji A, Hare BD. Do sleep disorders contribute to pain sensitivity? *Curr Rheumatol Rep.* 2011;13:528-34.
- 2 Theadom A, Cropley M, Humphrey KL. Exploring the role of sleep and coping in quality of life in fibromyalgia. *J Psychosom Res.* 2007;62:145-51.

- 3 Spaeth M, Rizzi M, Sarzi-Puttini P. Fibromyalgia and sleep. *Best Pract Res Clin Rheumatol.* 2011;25:227-39.
- 4 Tang NK, Wright KJ, Salkovskis PM. Prevalence and correlates of clinical insomnia co-occurring with chronic back pain. *J Sleep Res.* 2007;16:85-95.
- 5 McCracken LM, Iverson GL. Disrupted sleep patterns and daily functioning in patients with chronic pain. *Pain Res Manag.* 2002;7:75-9.
- 6 Haack M, Mullington JM. Sustained sleep restriction reduces emotional and physical well-being. *Pain.* 2005;119:56-64.
- 7 Tiede W, Magerl W, Baumgärtner U, Durrer B, Ehlert U, Treede RD. Sleep restriction attenuates amplitudes and attentional modulation of pain-related evoked potentials, but augments pain ratings in healthy volunteers. *Pain.* 2010;148:36-42.
- 8 Campbell CM, Bounds SC, Simango MB, Witmer KR, Campbell JN, Edwards RR, Haythornthwaite JA, Smith MT. Self-reported sleep duration associated with distraction analgesia, hyperemia, and secondary hyperalgesia in the heat-capsaicin nociceptive model. *Eur J Pain.* 2011;15:561-7.
- 9 Reiter RJ, Acuna-Castroviejo D, Tan DX. Melatonin therapy in fibromyalgia. *Curr Pain Headache Rep.* 2007;11:339-42.
- 10 Roehrs TA; Workshop Participants. Does effective management of sleep disorders improve pain symptoms? *Drugs.* 2009;69 Suppl 2:5-11.
- 11 Webster LR, Choi Y, Desai H, Webster L, Grant BJ. Sleep-disordered breathing and chronic opioid therapy. *Pain Med.* 2008;9:425-32.
- 12 Mogri M, Desai H, Webster L, Grant BJ, Mador MJ. Hypoxemia in patients on chronic opiate therapy with and without sleep apnea. *Sleep Breath.* 2009;13:49-57.



# Society of Anesthesia and Sleep Medicine 2013

## October 10-11, 2013 • San Francisco, CA

### Opioids, Respiratory Depression and Sleep-Disordered Breathing (SDB): Perioperative Implications

### *Please Join Us for the SASM 3rd Annual Meeting!*

On behalf of the Society of Anesthesia and Sleep Medicine (SASM), we invite you to attend the SASM 3rd Annual Meeting: Opioids, Respiratory Depression, and Sleep-Disordered Breathing (SDB): Perioperative Implications, to be held October 10–11, 2013 in San Francisco, CA.

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

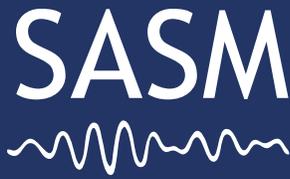
We hope you will join us in San Francisco. Visit [www.SASMHq.org](http://www.SASMHq.org) for More Information!

### Thursday, October 10, 2013

3:00-3:05pm	Introduction to Workshops	Babak Mokhlesi, MD, MSc
3:05-6:00pm	<b>Moderators:</b> Peter Gay, MD and Roop Kaw, MD	
3:05-6:00pm	<b>Workshop 1: Protocol development for the perioperative management of patients with SDB</b> <ul style="list-style-type: none"> <li>Review challenges in implementing a perioperative OSA program (Intermountain Experience: Tom Cloward; Toronto and Singapore Experience: Edwin Seet, MD, Vanderbilt Experience: Michael Pilla, MD)</li> <li>Discussion/Q&amp;A</li> <li>How to do PAP therapy: MetroHealth Experience (Dennis Auckley, MD)</li> <li>Clinical trial design and outcomes to be measured (David Hillman, MBBS)</li> <li>Discussion/Q&amp;A</li> </ul>	
3:05-6:00pm	<b>Moderators:</b> Mervyn Maze, MD, Mark Opp, PhD, Richard Horner, PhD	
3:05-6:00pm	<b>Workshop 2: Basic Science of Sleep</b> <ul style="list-style-type: none"> <li>Upper airway tone and patency in the postoperative period (Atul Malhotra, MD)</li> <li>Interactive effects of anesthesia, opioids, and sleep on breathing and arousal in the perioperative period (Matthias Eikermann, MD)</li> <li>Sleep as an immune system regulator during the postoperative period (Mark Opp, PhD)</li> <li>Discussion/Q&amp;A</li> <li>Sedation and natural sleep (Mervyn Maze, MB, ChB)</li> <li>Impact of anesthesia on circadian rhythms (Max Kelz, MD, PhD)</li> <li>Emergence from General Anesthesia: The Role of Dopamine (Ken Solt, MD)</li> <li>Discussion/Q&amp;A</li> </ul>	
6:00-6:45pm	Welcome Reception	
6:45-8:15pm	Dinner with Invited Speakers:	
	Introduction of the Incoming ASA President: Jane C.K. Fitch, MD	David Hillman, MBBS
	Shared Circuits of Anesthesia and Sleep: Basic Mechanisms and Clinical Relevance	Ralph Lydic, PhD

### Friday, October 11, 2013

7:15-7:55am	Annual General Meeting	
7:00-7:55am	Registration and Continental Breakfast	
7:55-8:00am	Introduction	David Hillman, MBBS
8:00-10:45am	<b>Moderator:</b> Babak Mokhlesi, MD, MSc	



**Society of Anesthesia and Sleep Medicine 2013**  
**October 10-11, 2013 • San Francisco, CA**  
**Opioids, Respiratory Depression and Sleep-Disordered Breathing (SDB): Perioperative Implications**

**Friday, October 11, 2013 continued**

8:00-8:50am	<b>Keynote:</b> Neural Mechanisms of Sleep and Sedation-Induced Respiratory Depression	Richard Horner, PhD
8:50-9:30am	<b>Keynote:</b> Opioids and Central Sleep Apnea	Shahrokh Javaheri, MD
9:30-10:00am	Delirium in Hospitalized Patients, Role of Sleep Disruption, Opioids and Pain	Jacqueline Leung, MD
10:00-10:15am	Discussion/Q&A	
10:15-10:45am	Refreshment Break and Poster Viewing	
10:45am-12:15pm	<b>Moderator:</b> Frances Chung, MBBS	
10:45-11:10am	Should All Postoperative Patients be Monitored on the Wards? Pro	Andreas Taenzer, MD
11:10-11:35am	Should All Postoperative Patients be Monitored on the Wards? Con	Satya Krishna Ramachandran, MD
11:35am-12:00pm	Impact of SDB on Postoperative Outcomes	Babak Mokhlesi, MD, MSc
12:00-12:15pm	Discussion/Q&A	
12:15-1:15pm	Lunch Break and Poster Viewing	
1:15-1:30pm	Awards to Scientific Abstracts Winners	David Hillman, MBBS
1:30-3:15pm	<b>Moderator:</b> Roop Kaw, MD	
1:30-2:05pm	Non-PAP and PAP Treatment of OSA Postoperatively	Sairam Parthasarathy, MD
2:05-2:30pm	Sedation and Anesthesia as a Surrogate for Sleep in Clinical Investigation of the Upper Airway	Eric Kezirian, MD
2:30-2:45pm	Discussion/Q&A	
2:45-3:15pm	Refreshment Break and Poster Viewing	
3:15-5:00pm	<b>Moderator:</b> Norman Bolden MD	
3:15-3:40pm	Anesthesia, SDB and Bariatric Surgery	Roman Schumann, MD
3:40-4:05pm	Suboxone (buprenorphine/naloxone) and Sleep Disordered Breathing, or Look Out for that Low Ceiling!	Robert Farney, MD
4:05-4:25pm	Pain Management with Opioids in the Postoperative Period and Impact on SDB	Anthony Doufas, MD
4:25-4:45pm	Analgesic Management in Children After Tonsillectomy & Adenoidectomy	Kimmo Murto, MD
4:45-5:00pm	Discussion/Q&A	
5:00pm	i-Pad® Giveaway and Closing Remarks	Babak Mokhlesi, MD

*\*Tentative Schedule: Program agenda and speaker selection subject to change.*



## Officers

### **President**

David Hillman, MBBS  
Sir Charles Gairdner Hospital  
Perth, Western Australia

### **President-Elect**

Frances Chung, MBBS  
University of Toronto  
University Health Network  
Toronto, ON Canada

### **Secretary**

Norman Bolden, MD  
Case Western Reserve University  
MetroHealth Medical Center  
Cleveland, OH USA

### **Treasurer**

Peter Gay, MD  
Mayo Clinic  
Rochester, MN USA

## Board of Directors

Babak Mokhlesi, MD  
University of Chicago Pritzker  
School of Medicine  
Chicago, IL USA

Girish Joshi, MB, BS,  
MD, FFARCSI  
University of Texas  
Southwestern Medical School  
Dallas, Texas USA

Roop Kaw, MD  
Cleveland Clinic Lerner  
College of Medicine  
Cleveland, OH USA

## Committee Chairs

### **Newsletter Chair**

Girish Joshi, MB, BS,  
MD, FFARCSI  
University of Texas  
Southwestern Medical School  
Dallas, Texas USA

### **Abstract Chair**

Yandong Jiang, MD, PhD  
Harvard University and  
Massachusetts  
General Hospital  
Cambridge, MA USA

### **Discussion Group Moderator**

John Loadsman, MBBS,  
PhD, FANZCA  
Royal Prince Alfred Hospital and  
University of Sydney  
Camperdown, Australia

### **Teaching & Training Co-Chairs**

Dennis Auckley, MD  
MetroHealth Medical Center  
Bay Village, OH USA

Bhargavi Gali, MD  
Mayo Clinic  
Rochester, MN USA

### **Clinical and Research Chair**

Roop Kaw, MD  
Cleveland Clinic  
Solon, OH USA

## SASM Membership Benefits at a Glance...

These are exciting times for SASM. While we are a new and growing organization, we feel our collaborative efforts will give rise to unlimited opportunities. You have the ability to make an impact from the very start. **Please consider joining SASM today!**

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

### **Benefits of SASM Membership include:**

- Significantly Reduced Registration Fees at SASM Sponsored Scientific Meetings
- SASM Newsletter
- \*Full Voting Rights in Electing SASM Board of Directors and SASM Officers (\*Dependent on membership category)
- Regular Receipt of "Literature Updates" and "Featured Articles," Allowing All Members to Stay Current on New Developments in the Area
- Enhances Your Network of Regional, National and International Colleagues
- Learn of Collaborative Research Projects
- Educational Material Posted on SASM Website for Members
- Access to a "Discussion 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

The easiest and quickest route to join as a member of SASM is to visit our website, [www.SASMhq.org](http://www.SASMhq.org), and pay by credit card by clicking on the Membership Information tab. You can also mail check payment to our office at the address provided below.

### SASM Classes of Membership:

#### **Gold Patron Member - \$250**

- Showing special support for SASM
- This donation is inclusive of annual membership and available for all classes of membership.

#### **Active Member - \$100**

- Physicians and Scientists. Active Members have voting rights, can hold office and serve on the Board of Directors.

#### **Associate Member - \$50**

- Non-Physicians and Non-Scientists. Associate Members do NOT have voting rights.

#### **Educational Member - \$50**

- Fellows, Residents, Medical Students or other undergraduates. Educational Members do NOT have voting rights.

### **Please consider joining as a "Gold Patron" for 2013**

The additional donation beyond general membership will be used to promote scholarly activity in the area of anesthesia and sleep medicine and promote patient care programs in areas common to anesthesia and sleep medicine. Gold Patrons will be recognized on our website for their extraordinary support of SASM efforts and will be invited to special events highlighting the programs made possible with their donations, including a keynote speaker dinner at the Annual Meeting.

### **SASM - NEW OFFICE LOCATION!**

6737 W Washington Street, Suite 1300  
Milwaukee, Wisconsin 53214

SASM is a 501(C)(3) non-profit organization. Membership dues may be deductible as a business expense.  
**SASM Tax ID number is 27-4613034**

