

### 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*

#### What About the Workers? Sleep and Ourselves

The practice of medicine has always included an element of self-sacrifice. Self-flagellation even. The Knights of St. John promised to be “servants and slaves to our Lords, the sick” and many of their waking moments were devoted to their care.

A potential problem with this otherwise admirable dedication is in the proportion of every 24 hours occupied by waking moments and how much time this leaves for sleep. Average normal daily adult sleep required is estimated to be between 7.5 and 8 hours. Many do not consistently achieve this because of the competing demands of their busy work, family and social activities [1]. Unfortunately the basic requirement for sleep is immutable, although a significant proportion of us choose to restrict

our sleep to below optimal in a search for time to deal with these other demands. Ironically the state of sleep restriction decreases our effectiveness in undertaking the wakeful activities for which our sleep is sacrificed.

Apart from tiredness and the desire for sleep, sleep restriction is accompanied by a decrease in cognitive ability and psychomotor performance. Thinking, decision-making, vigilance, reaction times and coordination suffer [2]. The frontal lobes, including centres responsible for emotional modulation, seem particularly affected and increased irritability is a common accompaniment. These effects are seen remarkably quickly and with relatively little sleep loss in experimental sleep restriction. Two nights of 5 hours sleep is enough to

treble the error rate on psychomotor vigilance task testing in young adult volunteers with normal sleep requirements [3]. Seventeen hours of wakefulness is accompanied by an equivalent psychomotor performance to an alcohol level of 0.05g/dl and 24 hours to 0.1g/dl [4]. Legally drunk. Besides their effects on health, wellbeing and social interaction, these consequences

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## 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*

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### Perioperative Care of Obstructive Sleep Apnea Patients: An Urgent Call For Properly Designed Studies

In recent years, the publications assessing the influence of OSA on perioperative morbidity and mortality have increased exponentially. However, almost all the studies have significant methodological limitations, including inadequate sample size. Obviously, the information from these underpowered studies may result in incorrect conclusions. In addition, most of the studies are not well controlled. Another major limitation of available studies is the use of surrogate outcomes (e.g., incidence of desaturation, need for supplemental oxygen) to define the incidence of complications. It is clear that these surrogate outcomes may not influence clinically relevant outcomes such as mortality, hypoxic brain death, significant non-fatal complications (e.g., myocardial infarction), length of hospital stay, and need for readmission.

Several members of SASM have the talent and expertise to help develop guidelines for an optimal study design assessing perioperative complications and management of patients with OSA. SASM can play a major role in this.

The articles included in this issue of the Newsletter discuss the limitations and controversies regarding

perioperative outcome and care of OSA patients. Stavros Memtsoudis provides us with a broad overview of the knowledge base regarding perioperative care of OSA patients, and calls for increased research in this area similar to that for cardiac disease and diabetes mellitus. Although preoperative identification of OSA should lead to modification of perioperative care including choice of anesthetic and postoperative monitoring, large trials report contradictory evidence. Satya Ramchandran further emphasizes the controversies surrounding recent studies assessing perioperative outcome in OSA patients. Interestingly, in contrast to previous reports of increased perioperative morbidity and mortality in OSA patients, recent analysis of large databases show lower resource utilization in this patient population. This is not surprising as previous studies used surrogate outcome measures to define perioperative complications, while recent studies assessed mortality and duration of hospital stay.

The primary therapy for OSA remains positive airway pressure. However, its benefits in the perioperative period remains controversial. Dennis Aukley provides us with a detailed assessment of a recent study evaluating the use

of postoperative positive airway pressure therapy in patients at high risk of OSA. Such critical analysis of published literature is necessary in establishing optimal perioperative care of OSA patients, as well as design future studies.

It is clear that the major cause of postoperative morbidity in the OSA population is opioid-related airway compromise. Anthony Doufas discusses the current understanding of effects of opioids on pain and inflammatory response. The initial observations in children suggesting high pain threshold in OSA patients and reduced need for opioids have now been reported in adults with OSA. These studies allow us to optimize opioid use and prevent opioid-related respiratory complications.

I would like you to know that Satya Krishna Ramchandran will be assuming the role of Editor starting October 2013. I would like to thank the SASM members that have taken time from their busy life to contribute to our Newsletter. Although such activities can be time consuming, they are gratifying and allow personal growth. Therefore, I believe that whenever possible it is important to participate in commit-

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**Stavros G. Memtsoudis MD, PhD,  
FCCP**

*Clinical Associate Professor of Anesthesiology  
Hospital for Special Surgery  
Weill Cornell Medical College  
New York, New York, USA*

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## Perioperative Obstructive Sleep Apnea: A Major Public Health Problem!

The topic of perioperative management of patients with obstructive sleep apnea (OSA) and its related challenges has received little attention outside of specialty societies as evidenced by the lack of discussions in mainstream health media and the virtual absence of funding opportunities for research. However, this apathy may be waning, as an increasing number of publications have pointed towards the importance of related issues not only regarding medical outcomes, but also, health care economics and public health in general [1]. The prevalence of OSA has undoubtedly reached epidemic proportions, with up to one fourth of men and one tenth of women affected [2]. While this disease complex continues to put a burden on our health care systems, the economic implications of OSA are increasingly coming to the forefront for hospitals and health care facilities performing tens of millions of surgical procedures annually in the US alone. The reasons for this trend are multifactorial and include 1) the fact that the prevalence of OSA among surgical candidates is even higher than among the general population, 2) the possibility that patients suffering from OSA are at increased risk for perioperative complications, and 3) the reality that hospitals and physicians feel compelled to employ

expensive but non-proven interventions in an attempt to reduce the risk for adverse events.

While the long-term adverse consequences of OSA are well known, the mal effects of the disease on perioperative risks have only recently gained attention via population-based and institutional investigations. In this context, some studies report that patients with OSA undergoing surgery may be at increased risk for adverse outcomes and need for advanced health care services leading to increasing healthcare expenditures [3, 4].

Further, the prevalence of OSA among elective surgical candidates has been estimated to significantly outpace that of the general population thus adding the burden of the unknown effects of untreated disease on outcomes. With the availability and increasing use of OSA screening tools [5], patients may now more frequently receive a likely diagnosis of OSA just before surgery. While this trend may be beneficial for the overall health care of a patient who now can be referred for further work up, the perioperative physician may find him/herself in a difficult situation with little guidance on what to do next. On one hand the decision to postpone the case and send the

patient for evaluation and possible initiation of therapy may be viewed as the conservative approach. On the other side he/she may feel pressured to proceed given the lack of firm data and high social and economic cost of a cancellation while knowing that the risk for complications may be increased. As neither approach is based on any evidence to help guidance, the decision made will have to be based on many individual assumptions, including in the case of cancellation that the patient will adhere to proposed treatment, as high non-compliance rates with PAP therapy have been reported.

Finally, further economic and logistical burden is placed on health care providers and institutions when patients with OSA do undergo surgery. Although the American Society of Anesthesiologists has published recommendations for the perioperative care of patients with OSA [6], these are based on very little evidence. The suggested use of increased levels of postoperative monitoring, routine PAP therapy and regional anesthesia in an attempt to decrease the risk of complications has been largely based on expert opinion. In fact, only recently a study evaluating the outcomes of OSA patients undergoing ortho-

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pedic surgery under regional versus general anesthesia has substantiated a portion of these guidelines. Specifically, better outcomes were associated with the use of regional anesthetic techniques [7].

Although patient safety concerns represent the motivation for the creation of guidelines, the economic burden of universal implementation, especially increased levels of observation and use of PAP therapy, seems prohibitive in the face of unknown effectiveness of these interventions and the overwhelming number of patients that they would have to be applied to. Therefore, it is not surprising that recent data suggest a low penetration of these guidelines into every day practice.

In conclusion, the mismatch of the increasing disease prevalence and diagnosis thereof, lack of scientific evidence for proposed treatments in the perioperative setting and high associated health care costs, as well as the fear of litigation, has created the perfect storm in respect to the care and its economic impact of patients with OSA undergoing surgery. Given the ongoing focus of health care agencies on addressing rising health care costs, the OSA epidemic and its impact on perioperative medicine represent a prime target for large-scale investments in research and policy endeavors. Learning from the impact that widespread support for the perioperative management of cardiac disease and diabetes has had on outcomes and evidence based care, it seems

obvious that OSA as a disease complex can no longer be ignored as a major public health problem. ❖

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of sleep loss have negative impacts on productivity and safety. Presenteeism – attending work in a suboptimal state – abounds but is difficult to measure. The safety implications involve ourselves (for example, driving home after a lengthy time on duty) and our patients (as, for example, our vigilance suffers and decision making capacities diminish). Anesthesia under such circumstances has been characterized as “the half asleep looking after the half awake”.

Sleep has essential restorative powers but is not a passive state and important activities occur during it. Memory consolidation is one of these [5]. What irony there is in undertaking late night study and cramming for

exams at the expense of the sleep required to consolidate that knowledge the sleep-deprived student desires.

The implications for ourselves are obvious, but the challenges of dealing with them difficult. Should we make more time for sleep? How do we balance this against our work? How do we squeeze in social and family life? How do trainees get enough clinical experience if work schedules are made “even” more sleep friendly? Who will do the work if you are not there? But... are you overdoing it? Are you as effective as you think you are? Are you as vigilant as you ought to be? Even... do you leave enough time for self-reflection, including about your sleep needs?

In thinking about the various aspects of sleep and anesthesia, we need to think about these matters too. As the knights of St. John might have said, in citing Luke 4:23, “physician heal thyself”. ❖

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**Satya Krishna Ramachandran MD FRCA**

*Assistant Professor,*

*University of Michigan Medical School*

*Ann Arbor, MI*



## Is There a Real reason to Study OSA? Recent Trends in Literature

For every clinician who has encountered challenging OSA cases and wondered about the best way to manage them, there are probably some who think that OSA is a modern-day creation that has little independent effect on outcomes. It is also postulated that the coexisting conditions of OSA are primarily responsible for the increased risk of postoperative respiratory failure. Certainly, this notion is not without merit. There are several interesting aspects of early postoperative respiratory failure that highlight the value of further study into OSA. However, recent large retrospective studies have shown that there are clinically relevant relationships between sleep disordered breathing, high risk of OSA and postoperative outcomes including respiratory failure, ICU stay, treatment costs and postoperative mortality.

Mokhlesi and colleagues [1] show that despite the increased independent association of SDB with postoperative cardiopulmonary complications, ***the diagnosis of SDB was not independently associated with increased in-hospital death.*** This finding may have implications for current concepts on risk of sudden postoperative death. Indeed in one previous study, the frequency of sudden unanticipated death was

less than 1:2,000 to 1:12,500 [2]. Mokhlesi's study was performed on over 1 million patients over several 100 hospitals. The lack of increased mortality in the SDB group may suggest increased levels of monitoring in high-risk patients, earlier treatment secondary to more significant hypoxemia and greater response to early treatment. The effectiveness of early CPAP seen in Squadrone's study [3] could very well reflect greater responsiveness to CPAP secondary to upper airway obstruction. Perhaps more interestingly, SDB was associated with earlier postoperative respiratory failure when compared to patients without SDB and had lower length of stay, total charges, pneumonias and in-hospital death compared to patients without SDB [1]. This again points to two important factors: SDB in general, and OSA in particular, is responsive to early airway interventions. Second, the risk period of intubation far preceded previously appreciated increases in postoperative SDB. So why is there this dichotomy between research findings of timing of early postoperative intubation (peaks on day 1) and increased apneas (peaks on days 3-5)? Are the patients with SDB who develop early postoperative respiratory failure different from others who don't? If so, can we predict this preoperatively?

Lockhart and colleagues [4] used a prospective cohort design to evaluate the influence of high risk of OSA or prior diagnosis of OSA on postoperative mortality. The study sample included 14,962 patients, of whom 1939 (12.9%) reported a history of OSA. The four screening tools that were evaluated identified a high prevalence of undiagnosed patients at risk for OSA (9.5%-41.6%). There was no significant difference in 30-day postoperative mortality between patients with possible OSA and the rest of the surgical population. On adjusted analyses, preoperative OSA screening failed to predict mortality rate up to one year postoperatively. This study highlights challenges with screening of large populations for rare outcomes (30-day postoperative mortality is still relatively rare) and the differences in accurate screening of disease conditions vs. screening of outcome states. On the other side, this study was conducted in a single center that was focused on outcome modification with OSA, and that inherent bias may have impacted outcomes.

These two large studies have ignited the debate in the scientific community about mechanisms of OSA and postoperative respiratory failure and overall impact of

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outcomes modification with additional monitoring, therapy and hospitalization. In summary, ***the first 72 hours following surgery have direct implications for respiratory morbidity.*** Respiratory failure caused by opioid use in the postoperative period peaks in the first 24 hours after surgery, whereas the incidence of significant postoperative hypoxemia and sleep-related breathing abnormalities continues through and peaks on the third postoperative night [5-7]. Concomitant with these physiological changes, the risk of unanticipated respiratory failure increases during the first three postoperative days. ***Early postoperative respiratory failure occurs in 0.2-3.8% of cases and is associated with an independent 9-fold increase in mortality.*** Although postoperative respiratory complications are associated with a 12-fold increase in patient care costs, in addition to the significant impact on mortality, ***similar costs are not seen with increased respiratory failure associated with OSA*** in retrospective analyses of large databases. The existing “phenotypic” prediction model for early postoperative respiratory failure shows modest discrimination and limited

modifiability. The early postoperative period also captures the most intense clinical interaction between patients and healthcare providers, and carries a high potential for innovation to influence risk-benefit relationships.

***Based on higher success and lower costs with early airway intervention, OSA still presents an excellent, minimally explored opportunity for studying prediction and mechanisms of early postoperative respiratory failure.*** First, OSA is a highly prevalent condition that affects ~ 25% of middle-aged men and ~ 10% of middle-aged women, with significant impact on quality of life, life expectancy, cardiovascular disease, respiratory disease and several other end-organ abnormalities, in common with the metabolic syndrome. Second, emerging evidence suggests an independent 3-fold increase in the rate of early postoperative respiratory failure in patients with high risk of OSA. Previous diagnosis of OSA is associated with a 2-fold increase in respiratory complications after surgery. Third, the metabolic syndrome “complex” of obesity, diabetes mellitus and hypertension encapsulates

independent risk features of OSA, and has been shown to independently increase perioperative morbidity. This provides additional scientific rationale for choosing OSA as a target clinical model to study early postoperative respiratory complications. Further, it is unknown if these individual components of OSA improve prediction of perioperative respiratory failure better than the simple knowledge of diagnosis or risk features of OSA. This major knowledge gap significantly impacts perioperative care of patients with OSA due to inadequate rigorous outcomes data in literature. ❖

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tee activities. If you are interested in contributing to SASM activities, please review the SASM organizational structure on the SASM website to determine the committee of your interest, and email your request to Francis Chung, President Elect of SASM. ❖



**Dennis Auckley MD**

*Director, Center for Sleep Medicine*

*Division of Pulmonary, Critical Care and Sleep Medicine*

*MetroHealth Medical Center*

*Associate Professor of Medicine*

*Case Western Reserve University*



## Is There a Role For Positive Airway Pressure Therapy For OSA Patients in the Perioperative Setting?

There is growing consensus that patients with known or suspected obstructive sleep apnea (OSA) are at increased risk for cardiorespiratory complications following surgery [1, 2]. A number of protocols have been published suggesting interventions that might be expected to reduce the risk of postoperative complications in these patients [1, 3, 4]. Common to all of these protocols is the recommendation that positive airway pressure therapy (PAP = CPAP or Bilevel PAP) be utilized postoperatively for patients with known OSA on PAP therapy at home and be considered for those at high risk for OSA but not yet diagnosed or on therapy. However, to date there is limited data regarding the effectiveness of PAP therapy in the postoperative setting in patients with known or suspected OSA. The recently published study by O’Gorman et al [5] is the first controlled trial attempting to better understand the role of PAP therapy for surgical patients with OSA, though it raises more questions than answers.

PAP therapy remains the first-line treatment for OSA [6]. It improves daytime sleepiness, quality of life, metabolic parameters, left ventricular function, blood pressure control

and may reduce the rates of nonfatal and fatal cardiovascular events [7-11]. PAP devices accomplish these outcomes by stabilizing the upper airway, decreasing the number of apneic episodes, decreasing the duration of apneic episodes and improving oxygenation. All of these effects seem to be beneficial in the postoperative period, as the residual effects of anesthetics and the use of opioids for pain control may aggravate OSA.

It seems intuitive that patients with known OSA already on PAP therapy utilize their prescribed therapy up until the day of surgery, and then be placed back on it as soon as possible postoperatively. Surprisingly, there are no adequately controlled studies to verify that this is of benefit. Case series data from the 1990s suggested that the use of PAP therapy could reduce postoperative complications in OSA patients [12]. Another retrospective study of OSA patients undergoing lower extremity joint replacement surgery reported improved outcomes in patients utilizing CPAP perioperatively, although the subset of patients was too small to statistically analyze and the study was not controlled [13]. Aside from the limited data, concerns have also

been raised about the effectiveness of previously determined fixed PAP pressure settings in controlling OSA and preventing hypoxia in the postoperative setting [14]. In addition, a recent retrospective study found that OSA patients receiving autotitrating PAP (APAP) therapy within a week of their surgery tended to be poorly compliant in the first 30 days of therapy [15]. While clearly more and better quality data is needed, there may be benefit and likely little risk, to keeping patients already on PAP therapy at home for OSA on treatment postoperatively.

Perhaps the more difficult clinical situation is what to do with patients who are identified as being at high risk for OSA, but are not yet diagnosed, and have surgery planned in the very near future. Should these patients have their surgery delayed in order to undergo diagnostic testing and therapeutic intervention prior to surgery? This approach would be quite labor intensive and certainly would wreak havoc on surgery schedules. Should these patients identified as being at high risk for OSA be monitored more closely postoperatively? This seems to make sense though again would likely require increased resource

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utilization, depending on the intensity of monitoring. Or, should these patients be empirically placed on PAP therapy postoperatively? While the first 2 approaches lack data to aid in clinical decision-making, the last approach has now been evaluated in the recent study by O’Gorman et al [5].

In this prospective, randomized and controlled study, 138 patients undergoing elective total knee or hip arthroplasty were screened for OSA risk by the Flemons sleep apnea clinical score. Those considered at low risk for OSA (52 patients) received standard perioperative care. Those scoring as high risk for OSA (86 patients) were randomized to either standard perioperative care or standard perioperative care plus postoperative APAP (n=43 in each group). Overnight oximetry was performed on the first postoperative night and a limited channel cardiorespiratory sleep study (Embletta) was performed off therapy on the night before discharge. The authors hypothesized that patients identified as being high risk for OSA and randomized to APAP therapy would experience a shorter hospital length of stay (primary outcome) and less postoperative complications than those managed with standard care alone. An intention to treat analysis was used for analyzing the primary outcome.

There were no significant differences in length of stay or postoperative complication rates between the two groups, suggesting no benefit to the use of APAP in the postoperative setting. In fact, on subgroup analysis of the patients with an AHI  $\geq 15$  by the inpatient Embletta study, the

group placed on APAP had a 1 day longer length of stay (p=0.02) than the standard care group. While these results might be considered disappointing, several factors from the study warrant discussion before we completely dismiss the idea of empiric PAP therapy in high-risk patients.

In the group randomized to APAP (n=43), 38 patients utilized the device postoperatively whereas 5 either refused (n=1), were not placed on APAP due to a process failure (n=2) or had loss of data from the device (n=2). In those who did use APAP therapy, the median time on APAP the first night was 373 minutes (just over 6 hours), but decreased significantly to a median of only 184.5 minutes per night (just over 3 hours) for their postoperative hospital stay. Unlike the earlier mentioned retrospective study that also found limited perioperative compliance with newly initiated PAP therapy [15], patients in this study received intensive PAP education, including verbal and video instruction, mask fitting and a PAP trial postoperatively. Interventions to improve PAP compliance in the perioperative setting should be evaluated, as it’s conceivable that improved compliance could improve outcomes.

Aside from the overall poor compliance with PAP therapy, the machine estimated median AHI was 13.5 with only 14 of the 38 patients achieving “effective control” of their sleep apnea postoperatively (defined as an AHI <10). While there appeared to be no significant issues with mask leak, the APAP devices used in the study were not

able to differentiate central from obstructive events and thus did not increase the pressure in response to apneas if the machine had already reached 10 cm H<sub>2</sub>O. Therefore, additional work is needed to determine how to achieve more effective PAP therapy in the postoperative period. Taken together, the suboptimal control of the OSA coupled with limited time on PAP therapy raises concerns that under treatment may mask any potential benefits that PAP therapy might offer in this setting.

Of interest, the patients randomized to APAP postoperatively experienced significantly more nocturnal hypoxia on the first postoperative night as compared to the high-risk group treated with standard care alone. Per their institution’s protocol, all patients received oxygen at 2 L/min on the first postoperative night and thus the authors’ postulate that this difference may have been the result of a reduction in the fractional oxygen concentration inspired in the APAP group due to the oxygen being diluted during bleed-in to the APAP devices. Another consideration is that the APAP devices may have led to worsening hypoventilation and one wonders if an autoadjusting bilevel pressure device would be more effective postoperatively.

And finally, it is important to note that, despite the rigorous and excellent methodology of the study, it was underpowered to detect a difference in the primary outcome (length of stay). In addition, the overall rate of complications (16%) was much lower than anticipated

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**Anthony G. Doufas, MD, PhD**

*Associate Professor*

*Department of Anesthesiology, Perioperative and Pain Medicine*

*Stanford University School of Medicine*

*Stanford, CA*



## Experimental Pain and Opioid Analgesia in Volunteers at High Risk for Obstructive Sleep Apnea

A large fraction of surgical patients suffer from obstructive sleep apnea (OSA) [1, 2], which might increase their risk for postoperative respiratory complications mainly due to drug-induced airway compromise [3-5], especially when  $\mu$ -opioid receptor agonists are administered to treat pain [6, 7]. In that context, the objective of this investigation was to characterize pain processing and opioid analgesia in male volunteers who were recruited based on a formal diagnosis or their risk for OSA [8]. Using experimental models for heat- and cold-induced pain, the study examined the effect of sleep disruption on pain processing and opioid analgesia. Based on prior evidence from pediatric surgical patients with OSA [9, 10], the authors hypothesized that preoperative nocturnal hypoxemia would be associated with a decreased sensitivity to painful stimuli and increased potency to opioid analgesia, while they also investigated the role of specific inflammatory and hypoxia markers in predicting sensitivity to pain and opioid analgesic effect.

Thresholds and tolerances for experimentally induced heat- and cold- related pain, were assessed in volunteers at risk for OSA, at

baseline, placebo, and during an opioid (remifentanyl) infusion targeting at two different effect site concentrations (1 and 2 mcg/mL, presented in random order). Volunteers also underwent over-night polysomnography and blood draw for determination of inflammatory (interleukin [IL]-6, IL-1 $\beta$ ; tumor necrosis factor- $\alpha$  [TNF- $\alpha$ ]) and hypoxia (erythropoietin [EPO], insulin growth factor binding protein-1 [IGFBP-1], vascular endothelial growth factor [VEGF]) markers. Mixed linear regression analysis was employed to assess in 43 volunteers the effect of sleep fragmentation (indicated by time spent awake after sleep onset, and the number of sleep stage shifts in nocturnal polysomnography), nocturnal hypoxemia (indicated by the nadir nocturnal arterial saturation [SaO<sub>2</sub>]), and the examined biomarkers on heat- and cold-pain parameters, as well as their change under remifentanyl infusion.

Lower nadir SaO<sub>2</sub> and increased serum levels of IGFBP-1, TNF- $\alpha$ , and IL-1 $\beta$  were associated with enhanced analgesic potency for remifentanyl, while increased IGFBP-1 was also associated with higher heat and cold pain threshold at baseline. Statistical model adjustment for the

presence of inadequate/fragmented sleep in these subjects supported an independent association of hypoxia and inflammation with opioid analgesic pharmacology.

These findings support previous clinical evidence in children where recurrent nocturnal hypoxemia increased sensitivity to the analgesic effect of morphine (i.e., children with a nadir arterial SaO<sub>2</sub> <85% required half the dose of morphine to treat post-adenotonsillectomy pain) compared with those with nadir SaO<sub>2</sub>  $\geq$  85% [9, 10]. Furthermore, this effect is also supported by experimental evidence where the application of intermittent hypoxia up-regulated  $\mu$ -opioid receptors in developing rats [11, 12]. In contrast, a different clinical investigation has recently demonstrated that African American children with OSA required more opioids for postoperative pain management and experienced longer post-anesthetic recovery due to inadequate pain control, compared with Caucasian children suffering from OSA [13]. The presence of such contradictory findings in the literature is characteristic of the highly complex physiology underlying the examined outcomes and

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the need to control for multiple confounders. Since demonstrating an independent effect of nocturnal recurrent hypoxemia on pain and/or opioid analgesic sensitivity may be confounded by several other parameters including sleep quality (adequate vs. short, continuous vs. disrupted, light vs. deep sleep) and genetic predisposition, much larger studies are required to demonstrate such an effect, especially if the latter is of medium-to-small size. Although the relatively small size, as well as the experimental character of the present investigation necessitates its replication in larger clinical populations [8], this study is the first to link intermittent hypoxia and serum markers of systemic

inflammation to pain and opioid analgesic sensitivity phenotypes. ❖

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### *“Is There a Role For Positive Airway Pressure Therapy For OSA Patients in the Perioperative Setting?” continued from page 8*

and so it is very likely that the number of subjects enrolled was far too few to detect any clinically meaningful difference in complication rates.

At this point, the role of empiric APAP therapy in the postoperative setting for patients at high risk for OSA clearly warrants further study. Large, well-designed, controlled multicenter trials will be required to determine if and how this therapy may optimally benefit our patients and, hopefully, led to improved patient outcomes. ❖

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# SASM



3<sup>rd</sup> Annual Meeting  
October 10-11, 2013  
Le Méridien San Francisco



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

*Program Chair:*

Babak Mokhlesi, MD, MSc  
University of Chicago

*Program Co-Chair:*

Frances Chung, MBBS  
University of Toronto  
*SASM President-Elect*

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.

October 10, 2013 will include workshops on “Protocol Development for the Perioperative Management of Patients with SDB” and “Basic Science of Sleep.”

We hope you join us in San Francisco.

**Visit [www.SASMhq.org](http://www.SASMhq.org) for more Information!**





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## 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**

