

Table of Contents

Message from the President 1-2

Editor's File 2

Perioperative PAP Therapy: Nuts and Bolts 3-5

Treatment of Hypercapnic COPD With Noninvasive Ventilation 6-7

Rethinking Gabapentinoids and Respiratory Depression 8-9

Screening for Pediatric Obstructive Sleep Apnea 10-11

The Weight of Obstructive Sleep Apnea 12-13

Monitoring in Sedation: Looking (Twice) into the Future? 14-15

SASM Member Benefits 16

SASM newsletter

Message from the President

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In my last message as the SASM President, I would like to express my deepest gratitude for the great honor of having been nominated by the Board and elected by the membership to serve as your fifth president. I am also very grateful to our Past Presidents (Drs. David Hillman, Frances Chung, Peter Gay and Girish Joshi) for their counsel. My work would have not been possible without the efforts of our tireless Executive Board, Committee Chairs, and Committee members for their help, support and encouragement throughout the year. It is due to their dedication and hard work that SASM remains a preeminent society. Last, but not least, I am extremely grateful to our Executive Director, Marie Odden and her staff who have worked diligently on behalf of SASM. I would like to remind our membership that although our society is a young one, we are now close to completing our eighth year as a society. This is an achievement that we should all be proud of. To be frank, the responsibility of serving as SASM President was quite intimidating and daunting. Nonetheless, it has been an extremely gratifying experience for me and I learned a lot from our Board and members.

I have developed a deep conviction that SASM is an important and vibrant society that has much to offer to the fields of Anesthesia and Sleep Medicine. Our ultimate goal remains the betterment of patients' perioperative healthcare. Over the past 8 years, we have successfully brought together an outstanding group of clinicians and researchers in the field of Anesthesia and Sleep Medicine to foster education, scientific progress, and research in the two fields. Our society continues to work diligently with the International Anesthesia Research Society (IARS). We are grateful to Dr. David Hillman for serving as the Executive Section Editor for Sleep and Respiration for *Anesthesia & Analgesia*, the flagship journal of IARS and the official journal of SASM. Both IARS and SASM share the goal of increasing the amount of published literature related to respiration and sleep medicine. This relationship has been most critical in helping disseminate knowledge of sleep medicine in the fields of anesthesiology. Under Dr. Hillman's leadership there has been a significant growth in the number of submitted and published manuscripts related to the fields of Anesthesia and Sleep Medicine. We must continue to foster this relationship with IARS because publication of important research, clinical reviews and guidelines will enhance SASM's influence and footprint.

During my term we decided to explore a new structure for our flagship annual scientific meeting. This year in San Francisco, the SASM 2018 Annual Meeting will be limited to a full day of scientific activities. The Conference Committee decided to introduce workshops during the meeting. We plan to have two concomitant workshops: 1) Positive Airway Pressure and Noninvasive Ventilation workshop led by Drs. Peter Gay and David Hillman, and 2) Point of Care Ultrasound workshop focused on assessing cardiac function and hemodynamic status, led by Dr. Stephen Haskins. We hope these workshops will be well-received by SASM members. Dr. Krish Ramachandran and Dr. Tom Cloward have done a fantastic job putting together the program for our annual meeting on Friday October 12, 2018 in San Francisco. This year, the theme of the meeting will be "Perioperative Care and Sleep Medicine: Controversies,

>> President's Message continued on next page

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Editor's File

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Welcome to the October edition of the SASM newsletter. The SASM Annual Meeting in San Francisco is quickly approaching. This issue of the newsletter features up-to-date and important topics we hope SASM members caring for patients with sleep disordered breathing during the perioperative period will find relevant to their practice. I would like to thank all of the contributors to this issue of the newsletter. These articles highlight areas for further research to improve perioperative management of patients with sleep disordered breathing.

Patients with OSA are at increased risk for complications during the intraoperative and postoperative period. Positive airway pressure (PAP) therapy is considered to be the most effective treatment for OSA, therefore it is vital for perioperative physicians to understand the different modalities of PAP therapy that are available, and the indications for these modalities. In this issue, Dennis Auckley, MD provides a comprehensive summary of his recent article on perioperative PAP therapy that was published in the journal *Respiratory Care*.

The effectiveness of noninvasive ventilation (NIV) for treating hypercapnic respiratory failure has been controversial. Peter Gay, MD reviews the evidence for and against this technique beginning in the 1970's and 1980's to more recent trials including a trial published in 2017 that showed benefits of home NIV with oxygen versus oxygen alone on hospital readmission or death after an acute COPD exacerbation.

The opioid epidemic has prompted many clinicians to use multi-modal non-opioid analgesics including gabapentinoids to try to reduce the risk for opioid-induced respiratory depression. However, as Toby Weingarten, MD outlines, there is increasing evidence that gabapentinoids when used with opioids, may be associated with increased risk for postoperative respiratory depression. This is an unexpected finding that clinicians should consider when prescribing opioids for surgical patients who are already on chronic gabapentinoids and for patients who are newly prescribed gabapentinoids after surgery.

Although children with sleep disordered breathing (SDB) are at higher risk for perioperative respiratory complications,

there is currently a lack of a universal and validated preoperative screening tool for children with SDB. Heather McClung, MD, Denis Jablonka MD, and Rajeev Subramanyam MD, MS, critically examine some of the tools available for screening SDB in children.

In this issue, Sarah McConville, MD addresses a common question that perioperative physicians caring for patients who are preparing to have bariatric surgery may ask. She reviews the relation between weight and OSA and the effect of weight loss in obese individuals and improvement in OSA and co-morbid conditions.

Finally, Pedro Gambus, MD discusses the challenges of monitoring adequate depth of sedation and future possibilities for monitoring technology that can be developed to monitor sedation.

I look forward to seeing many of you at the upcoming SASM Annual Meeting in San Francisco on October 12, 2018.

If you are interested in contributing an article or joining the Newsletter Subcommittee, please contact me as we welcome contributions from all SASM members.

>> President's Message continued from previous page

Challenges and Special Populations". I am confident the program will be well-received by attendees.

I also like to express my gratitude to Dr. Jean Wong who continues to chair our Newsletter Subcommittee and Dr. Susana Vacas who continues to lead the Scientific Update Subcommittee. Both subcommittees and their members develop important summaries three times a year in order to keep our membership informed about new developments and controversies in the field.

From the financial standpoint, SASM remains in good health. One of our main challenges will continue to be securing more corporate and industry support in order to enable us to pursue various projects that expand membership benefits such as reintroduction of research grants. Another challenge for any young society like ours is to continue to increase membership and compete with much larger societies. I am grateful for the efforts of the Membership Committee, under the leadership of

Drs. Meltem Yilmaz and Ellen Soffin who have been developing creative approaches towards increasing our membership. I am confident that with the hard work and the dedication of our Board and members, we will overcome these challenges.

It has been a great honor to serve as SASM President and I wish you all a nice end of the summer and I am looking forward to seeing you in San Francisco!

Perioperative PAP Therapy: Nuts and Bolts

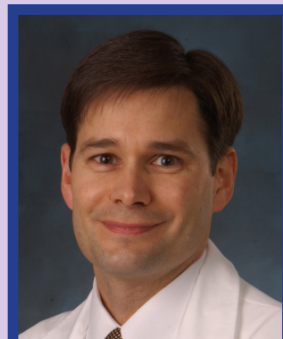
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Disclosures: Research Funding from Medtronics. Site PI for PRODIGY study monitoring CO2 in inpatients on opioids.

Growing evidence suggests that patients with obstructive sleep apnea (OSA) are at increased risk of postoperative cardiopulmonary complications.^{1,2,3} Positive airway pressure (PAP) therapy is considered the first-line treatment for OSA.⁴ Epidemiologic data suggests that the use of PAP therapy in the perioperative setting may reduce the incidence of adverse cardiopulmonary events postoperatively in patients with OSA,^{5,6} though randomized controlled trials of sufficient size and power are lacking at present. Regardless, most if not all perioperative providers will be confronted with caring for patients already on PAP therapy, or perhaps in urgent need of PAP therapy following surgery, and thus having a working understanding of PAP therapy is relevant to practice. Important aspects of this treatment include the different modalities, indications for use, interface choices, monitoring therapy, and trouble shooting. A recently published article in the journal *Respiratory Care* sought to review these issues for those involved in perioperative care.⁷ This newsletter brief will summarize some of the highlights from that review.

Modalities of PAP Therapy and Indications

The primary modalities of PAP therapy include Continuous PAP (CPAP), bilevel PAP (BPAP) and adaptive servo ventilation (ASV). There are variations of each modality, which will be briefly discussed, though CPAP and BPAP are the primary therapies most likely to be encountered by the perioperative provider.

CPAP provides a fixed continuous pressure throughout the respiratory cycle to “splint” open the upper airway during sleep. CPAP is delivered as a fixed pres-

sure within a range from 5-20 cm H₂O, though settings in the 6-16 cm H₂O range are typically used in clinical practice. The optimal pressure setting for a given patient is determined by an in-lab polysomnogram during which a sleep technician manually adjusts the pressure to eliminate respiratory disturbances and snoring, ideally in all stages of sleep, and including supine sleep. Alternatively, CPAP can be used in an auto-titrating mode (often referred to as AutoCPAP or APAP) wherein the device continually monitors flow and algorithmically adjusts the pressure setting on an ongoing basis (over minutes) to eliminate flow limitation. APAP pressures are chosen by the prescriber to cover a range of pressures (e.g. 6-16 cm H₂O) likely to be needed to control the OSA. The main advantages of APAP over CPAP are twofold: an in-lab titration study can often be avoided, and the pressure varies over the course of the night to accommodate for different clinical situations where OSA may worsen (e.g. more supine sleep, addition of opioids or sedatives). As such, it can be particularly useful for empiric treatment of OSA in outpatients but may also play a role in the inpatient setting. CPAP and APAP are used in the treatment of OSA. APAP should not be used in patients with significant co-morbid hypoventilation or at risk for central apneas.⁸

BPAP, also known as bi-level ventilatory assistance, noninvasive pressure-support ventilation, and the proprietary names of BiPAP (Phillips Respironics) and VPAP (ResMed), differs from CPAP by providing separate and independently adjustable inspiratory pressure (IPAP) and expiratory pressure (EPAP). EPAP functions like CPAP to prevent upper airway collapse, while IPAP provides pressure support to assist inspiratory effort, and may be useful to reduce the work of breathing and prevent or treat hypoventilation. IPAP

also has stabilizing effects on the upper airway independent of the EPAP setting, thus allowing the EPAP setting to be lower than conventional CPAP.⁹ This may lead to better tolerance and adherence to PAP therapy in some patients. Like CPAP, BPAP settings are usually determined by an in-lab titration sleep study with manual adjustment of the settings (typical IPAP range 12 to 25 cm H₂O, EPAP range 6- to 20 cm H₂O). Unlike CPAP, BPAP can be used with back up rate to help with hypoventilation or central apneas. There are also autoadjusting BPAP devices which utilize separate pressure ranges for IPAP and EPAP in order to empirically treat OSA and maintain adequate ventilation. BPAP and its autoadjusting variation are usually used for OSA, often in cases where hypoventilation co-exists. Volume Assured Pressure Support (VAPS) is a relatively newer mode of BPAP that includes an EPAP setting to control upper airway collapse but employs a floating pressure support that targets a predetermined set minute or alveolar ventilation to better control ventilation during sleep. VAPS is primarily being used in patients with neuromuscular disease-associated sleep-related hypoventilation, though some consider its use in other hypoventilation syndromes.

ASV is a mode of PAP therapy designed to treat periodic breathing in sleep with or without accompanying OSA. Similar to BPAP, ASV delivers EPAP (fixed or autoadjusting) and pressure support but in contrast to BPAP the pressure support level varies on a breath to breath basis with a goal of eliminating periodic breathing.¹⁰ With ASV, the pressure support increases as spontaneous breathing effort decreases, and then decreases as spontaneous breathing effort increases. This anticyclical or “antidromic” support is designed to stabilize periodic breathing during sleep. ASV is indicated to treat periodic

>> Postoperative PAP Therapy continues on next page

>> Postoperative PAP Therapy continued from previous page

breathing associated with preserved ejection fraction heart failure, opioid use and treatment-emergent central apneas,⁷ but is currently contraindicated in patients with low ejection fraction heart failure.¹¹

When is PAP Therapy Indicated?

In the perioperative setting, one of the main uses of PAP therapy is to treat uncontrolled upper airway obstruction (OSA) and/or hypoventilation associated with, or worsened by, recovery from anesthesia, ongoing opioid use, and/or sedative drug use.⁷

Patients with known sleep disordered breathing and sleep-related hypoventilation syndromes are at increased risk of

breathing. In these situations, APAP (OSA) or BPAP (OSA and/or hypoventilation) could be considered with empiric settings chosen (e.g. APAP 6-16 cm H₂O, BPAP with an IPAP 14 cm H₂O and EPAP 6 cm H₂O) with adjustments made based upon clinical response. Considerations when adjusting therapy will include clinical observation of the patient on therapy, assessment of oximetry, possibly assessment of carbon dioxide (CO₂) monitoring, and downloads from the PAP devices.

Interface Choices

In general, there are 3 primary interfaces for use with PAP therapy; an oronasal mask that covers the nose and mouth (also called a full-face mask), a nasal mask that

a nasal interface, particularly in those who may be sedated from anesthesia and/or medications.

Monitoring and Trouble Shooting Therapy

Patients placed on PAP therapy in the perioperative setting should be monitored to assess the efficacy of therapy. This is particularly important when central nervous system depressing medications are used, if upper airway edema is a concern (e.g. difficult intubations, upper airway surgery, excessive fluid requirements) and in those with marginal oxygenation postoperatively. Ideally, the patient should be assessed during sleep, day or nighttime, while on PAP therapy. The most immediate feedback can come from observing

Table 1 – Trouble Shooting PAP Therapy	
Problem	Intervention options
Interface-related	
Claustrophobia	Change to nasal pillow interface. Trial desensitization exercises (daytime practice with the device).
Discomfort	Interface assessment and change to a different type or brand
Skin breakdown	Avoid excessive tightening. Interface change (consider nasal pillows). Protective barrier for skin.
Eye irritation	Usually from leaking and requires an interface assessment. In some cases, a lower pressure or trial APAP or BPAP may be warranted.
Pressure-related	
Nasal congestion / sinus pain	Heated humidification. Change to oronasal interface. Nasal topical sprays (i.e. steroids, antihistamines). Short-term use of nasal decongestants (avoid long-term use).
Nasal /oral dryness	Heated humidification. Saline nasal spray/gel. Interface change. Lower pressure or change to APAP or BPAP.
Rhinorrhea	Nasal ipratropium
Pressure intolerance	Lower the pressure or change to APAP, CPAPexp or BPAP.
Aerophagia	Lower pressure or change to APAP or BPAP. Avoid a oronasal mask. Assess for gastroesophageal reflux and treat if present.

APAP = autoadjusting CPAP, BPAP = bilevel positive airway pressure, CPAPexp = expiratory relief CPAP

adverse cardiopulmonary events postoperatively. PAP therapy should be utilized for such patients during sleep or when sedated. In patients who are already on PAP therapy at home, it is reasonable to continue with their home PAP mode and settings perioperatively, recognizing adjustments may need to be made to accommodate for opioid / sedative induced worsening.¹²

PAP therapy may also be considered in postoperative patients who are observed to obstruct their airway or experience hypoventilation but who have not yet been formally diagnosed with sleep disordered

covers the nose, and nasal pillows that fit snugly inside the nostrils. In patients new to PAP therapy, particularly in the inpatient setting, an oronasal mask may initially be tried. This type of interface is particularly helpful for those with nasal obstruction and/or those who are mouth breathers. However, the bulkiness of oronasal masks relative to nasal interfaces may be uncomfortable for some patients, and these interfaces can be difficult to tolerate if underlying claustrophobia exists. In such cases, one of the nasal interfaces may be preferable. Close monitoring for oral leaking is warranted in patients using

the patient at the bedside, assessing oxygenation and, if applicable, assessing CO₂. In some cases, arterial blood gas analysis may be a useful adjunct, especially where hypoventilation is a possibility. Based on these assessments, a determination can be made if adjustments in the pressure settings are needed, and if so, if a higher level of care may be warranted to ensure adequate monitoring and adjustments of therapy.

For patients with OSA on CPAP or APAP, if there is evidence of ongoing obstruction or hypoxemia, increasing the PAP setting

>> Postoperative PAP Therapy continued from previous page

by 1-2 cm H₂O (or increasing the lower setting on APAP) may be all that is needed. In patients with hypoventilation, adjustment of the IPAP setting to increase the IPAP to EPAP gradient will improve tidal volume and minute ventilation to correct or lessen hypoventilation. It's important to recognize that if the EPAP is increased (for airway obstruction or hypoxemia) while on BPAP, then the IPAP should be raised by the same amount to ensure adequate ventilatory support. Adding in a back-up rate to BPAP may be helpful for persistent hypoventilation and/or observed central apneas, though monitoring for dyssynchrony will become important.

Obtaining a download from the PAP device the day after use can provide a more global impression of how effective the therapy is and may also supply information regarding usage of the device as well as the presence of significant leaks. Typically, a Respiratory Therapist can help with procuring the download.

Additional monitoring for side effects from PAP therapy is key to helping patients use the device following surgery. A number of issues can limit compliance, and these are detailed, along with options for managing these problems, in Table 1.

Summary

Noninvasive PAP therapy options continue to evolve. Their role within the hospital setting is evolving, and its likely PAP therapies have been under-utilized in the perioperative setting. Improvements in the equipment have led to more versatility and increased ease of use for patients. In addition, the understanding of pathophysiology and optimal use of these devices has advanced, allowing providers to better tailor therapy to individual needs and circumstances. One of the main barriers to use of these therapies has been provider lack of familiarity with their application in the hospital setting, and it is hoped that educational efforts such as this will lessen the threshold for use.



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Treatment of Hypercapnic COPD With Noninvasive Ventilation

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Noninvasive ventilation or NIV includes any form of ventilatory assistance delivered without the aid of an endotracheal tube. This technique goes back many decades to the years of the polio epidemic in the 1950s when negative pressure ventilation in the form of the “iron lung” was a popular form of prolonged support for ventilatory failure. Interest grew in the 1970s and 80s with the concept of respiratory muscle fatigue and the potential benefits of targeted treatment in patients with chronic ventilatory failure especially those with chronic obstructive pulmonary (COPD) disease and daytime hypercapnia. A randomized controlled trial utilizing nocturnal NIV in hypercapnic COPD patients over many weeks demonstrated a reduction in daytime PaCO₂ levels and an improvement in quality of life (3). The study led to an early conclusion that by permitting recovery of muscle function and strength by reducing a tendency to fatigue, there would be a reduction in daytime CO₂ levels and perhaps even pulmonary function. Other studies failed to confirm the consistent benefit of nocturnal NIV in hypercapnic COPD patients (4,5) but once NIV was approved for this therapy and later shown to be a benefit for acute hypercapnic respiratory failure and hospitalized COPD patients, it quickly spread into wide acceptance and use.

A reasonable question arose as to whether or not the use of NIV could reduce the likelihood of hospital readmission when used routinely upon dismissal. Furthermore, could a more specific phenotype or factors in specific patients be used to identify patients expected to benefit most from post-hospital NIV therapy? A systematic review published in 2014 (7) was undertaken in 2 separate meta analyses at 3 and 12 months after hospitalization for COPD exacerbation to examine whether NIV could reduce readmissions. The

study also included a sub-group analysis to comparing IPAP level, compliance, and levels of hypercapnia in the control and actively treated groups. Overall, there was no significant difference in the readmission rate between the 2 groups and although in some studies the hypercapnia was less at 3 months, there was no difference at 12 months in gas exchange, 6 min walking distance, health-related quality of life, pulmonary function tests, or sleep efficiency. An interesting observation that emerged however was the fact that significant reductions in PaCO₂ were found and patient is ventilated with the highest IPAP levels of at least 18 cm H₂O for 5 hr a night especially in those patients who had a baseline PaCO₂ of dismissal of 55 mm Hg when compared to those patients with lower IPAP levels, poor compliance, and lower levels of hypercapnia at dismissal.

The Germans decided to pursue the question of whether specific benefit is related to what they called ‘high-intensity NIV’ (8) and had followed a large group of similar patients prospectively but in an uncontrolled fashion. A further difficulty was incurred by the fact that the introduction to this therapy required several days of hospitalization to establish tolerance and to insure the targeted maximum decrease in hypercapnia. They used an average rate of 21 ± 3 breaths per minute and a mean IPAP/EPAP of 28 ± 5/5 ± 1 cmH₂O. They eventually established tolerance in 73 patients (mean FEV₁ 30 ± 12 %predicted) between 1997 and 2006. After one year of the NIV therapy, they showed a sustained PaCO₂ decrease from 51.7 ± 6.6 to 44.9 ± 12.7 mmHg.

During this decade, it became necessary to prove this hypothesis in a prospective RCT and was undertaken in a sentinel prospective multicenter RCT of NPPV in

patients with chronic stable hypercapnic COPD compared to optimized standard therapy (9). The NPPV therapy was targeted to reduce baseline PaCO₂ by at least 20%, or to achieve PaCO₂ < 48 mmHg. The results were similar to the previous prospective trial and revealed a mean IPAP of 21.6 cmH₂O; mean EPAP was 4.8 cm H₂O and the backup rate was 16.1 bpm (range 2-24). They were also able to accomplish a mean NPPV usage of 5.6 hours/day. The primary outcome was the 1 year all cause mortality which showed a dramatic difference at 33% fatality in a control group compared to only 12% in the NPPV group.

The latest trial on the effect of home NIV with oxygen versus oxygen alone on hospital readmission or death after an acute COPD exacerbation has recently been published from this group (11). This was a randomized trial of 116 patients followed for a year using either a mean IPAP of 24 cm H₂O with a backup rate of 14 breaths/minute or 1 liter of supplemental oxygen. There were 28 patients on home oxygen alone and 36 patients with NIV and oxygen that completed the trial with a median time for readmission or death of 4.3 months in the home oxygen plus NIV group versus only 1.4 months in the home oxygen alone group. This was a high risk group overall as demonstrated by the 1 year risk of readmission or death of 63% in the NIV and oxygen group versus 80% in the oxygen alone group. Furthermore, there were 25 patients that died in this study with about equal numbers in each group.

With the striking evidence that has emerged over the last few years of not only the reduction in hospital readmission but now mortality in hypercapnic COPD patients using oxygen and NIV, it would seem obvious that insurance

>> Hypercapnic COPD continues on next page

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>> Hypercapnic COPD continued from previous page

coverage for this therapy would quickly come into alignment. The current criteria for reimbursement remains completely at odds making it very difficult for these patients to receive appropriate therapy that includes a backup rate. At this time, it is possible to employ the high intensity pressure levels but there is no currently published successful protocol in the USA. There are several efforts underway by multiple medical societies urging all insurers to consider coverage for this type of NIV therapy. On a more encouraging note, there are many open and

ongoing discussions about re-designing many aspects of reimbursement for all types of durable medical equipment in patients with chronic respiratory diseases and I remain cautiously optimistic about the future.

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Rethinking Gabapentinoids and Respiratory Depression

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The medical community has become aware that the current state of high-dose perioperative opioid practice has many detriments, including a gate way to chronic opioid use, respiratory depression and death, delayed recovery of bowel function, and immunosuppression with potentially worsening oncological surgical outcomes. Perioperative multi-modal analgesia has garnered much interest for potential opioid sparing effects, and has become the cornerstone of many Enhanced Recover After Surgery (ERAS) protocols.[1] The aim of multi-modal analgesia pathways is to leverage the synergistic effects of multiple agents in order to reduce the dose of individual agents while enhancing analgesia and reducing side-effects, and increasingly to reduce or eliminate perioperative opioid doses. Often the cornerstone of these analgesic protocols include medications devoid of central nervous system activity such as acetaminophen, nonsteroidal anti-inflammatory drugs, and local anesthetics, all of which should not have any effects on postoperative respiratory drive. Some protocols also incorporate a gabapentinoid, either gabapentin or pregabalin. Meta-analyses of the perioperative administration of either medication have shown that they have analgesic and opioid sparing properties, but increase the risk for postoperative sedation.[2, 3] Despite their proclivity for inducing sedation, based on toxicology literature these medications are widely believed to not suppress respiratory drive.[4, 5]

However, some observations from the Mayo Clinic raise questions about this assumption. The Mayo Clinic has a unique practice where the respiratory status during anesthesia recovery is systematically monitored by nurses for 'respiratory specific events' (episodes of apnea, hypoventilation, desaturations, and/or 'pain-sedation mismatch' – a report of a

high pain score despite residual sedation). [6] Patients who have these events have higher rates of postoperative pulmonary complications [6] and are at five times more risk of requiring emergent naloxone on postoperative wards following discharge from anesthesia recovery.[7, 8] In the two large retrospective studies we found associations between preoperative gabapentin administration and respiratory specific events during anesthesia recovery.[9, 10] When used as part of a multi-modal analgesic protocol for total joint arthroplasties performed under general anesthesia supplemented by peripheral nerve block, preoperative administration of 600 mg gabapentin was associated with increased risk for respiratory events (odds ratio, OR, 1.47, 95% CI 1.26 – 1.70, $P < 0.001$).[10] Similarly, when used as part of multi-modal analgesia for laparoscopic surgery, gabapentin was associated with respiratory events during anesthesia recovery (OR 1.47, 95% CI, 1.22-1.76; $P < .001$).

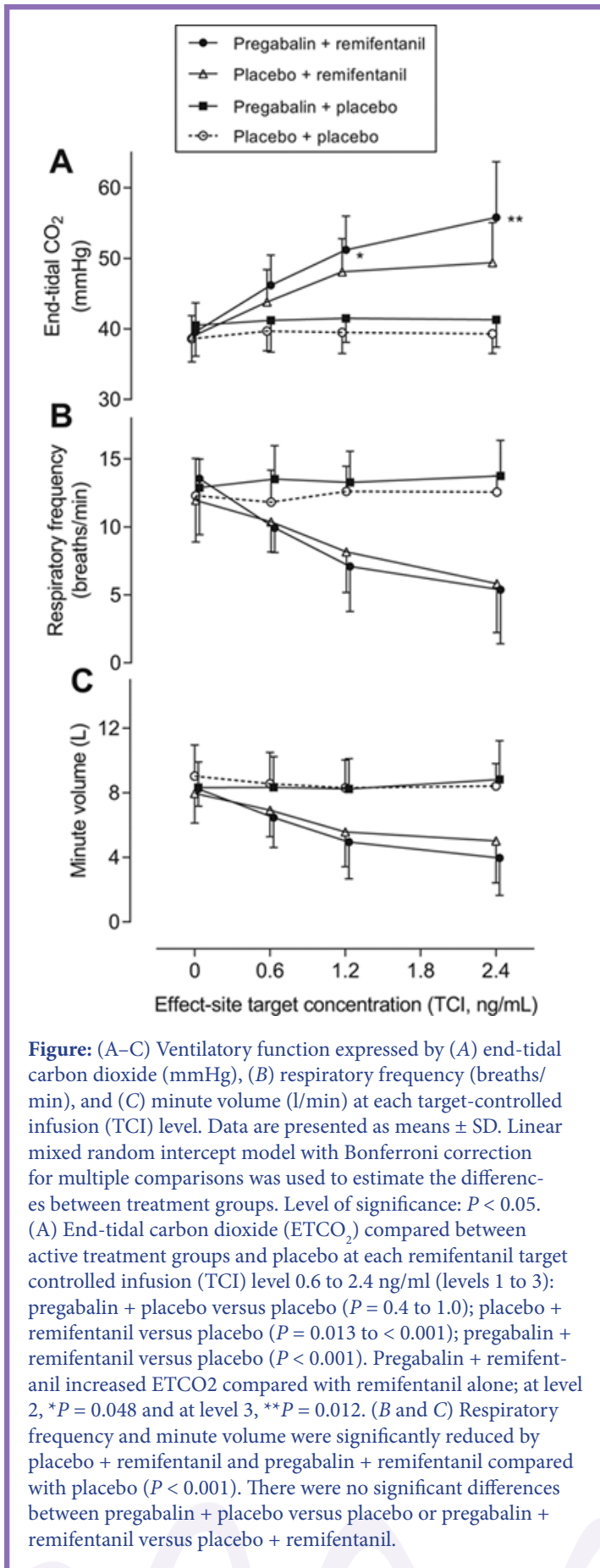
These observations are in line with a double blind crossover study by Myhre et al[11] which observed the effects of pregabalin, remifentanyl, and their combination on healthy volunteers on ventilatory effort as measured by end-tidal carbon dioxide, respiratory frequency, and minute volume. Pregabalin alone had no effect on respiratory drive. However, when co-administered with remifentanyl, pregabalin potentiated the respiratory depressive effects of the opioid as assessed by increasing levels of end-tidal carbon dioxide (Figure). At higher dose of remifentanyl, pregabalin potentiated respiratory depression by 62%. These findings suggest that when gabapentinoids are administered in opioid-naïve patients together with opioid analgesics, patients may be more prone to developing respiratory depression. One could speculate that the increased seda-

tion with the perioperative use of these medications could be a component of respiratory depression. Considering serious opioid respiratory depressive events has an approximate incidence of 0.1%, prospective randomized control trials of perioperative gabapentinoids may have been inadequately designed and powered to detect this increased risk.[12]

Patients on chronic gabapentinoids may also be at increased risk for postoperative respiratory depression. We recently analyzed patients who were administered naloxone within 48 hours of surgery for opioid induced respiratory depression or over-sedation.[8] Patients who were on chronic gabapentinoids and had these medications continued postoperatively were at increased risk of requiring naloxone reversal of opioid medications (OR 6.30, 95% CI 2.38, 16.66, $P = 0.001$). This increased risk may also be present in the ambulatory setting. A large population based study from Ontario examined risk factors for opioid-related deaths among patients on chronic opioid therapy and found that co-administration of gabapentin and opioids increased the risk of death (OR 1.49, 95% CI 1.18 to 1.88, $P < 0.001$) compared to opioid therapy alone.[13] Another recent study examined postmortem toxicology reports of patients who died from drug overdoses and found that 22% of decedents tested positive for gabapentin, and among those who tested positive for opioids 26% also tested positive for gabapentin.[14]

These observations suggest that both *de novo* and chronic gabapentinoid use may be associated with increased risk for postoperative respiratory depression. This risk should be accounted in the calculus of the relative merits of the use of these medications as part of multimodal analgesic pathways. Regardless, when encountering

>> Rethinking Gabapentinoids continued from previous page



patients administered these medications, healthcare providers should be aware of this risk and institute appropriate postoperative monitoring.

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Screening for Pediatric Obstructive Sleep Apnea

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Pediatric Sleep Disordered Breathing (SDB) and Obstructive Sleep Apnea (OSA) are attracting increasing attention in the perioperative arena as research highlights the significant morbidity and mortality these children can suffer after their surgical or non-operating room procedures. While it is known that surgical patients with known OSA are more likely to experience respiratory complications during their recovery, it remains unclear how best to assess risk and severity of SDB in patients not formally diagnosed with polysomnography (PSG). Our experience suggests that only 10-20% have a preoperative PSG. The 2014 American Society of Anesthesiologists practice guidelines for the perioperative management of patients with OSA recommends screening all patients over the age of 1 year for SDB using medical record review, interview, and physical exam.¹ However, no particular screening modality is endorsed. Therefore, the quest for a simple, accurate, and validated screening process for SDB in the pediatric population continues. In this newsletter, we will discuss the most recent questionnaires specifically designed to assess the risk for SDB in children who present for procedures requiring general anesthesia.

The Pediatric Sleep Questionnaire was one of the first pediatric screening tools for SDB. It was developed by Chervin et al in order to evaluate OSA presence and severity in the absence of PSG for clinical research purposes focusing on neurobehavioral outcomes. The portion that involves 22 parent-reported, closed question items is called the Sleep Related Breathing Disorder Scale (SRBD) and explores symptoms such as snoring,

daytime sleepiness, behavior, obesity, and nocturnal enuresis. Every yes is one point and the total score is divided by the number of questions answered. Scores ≥ 8 (out of 22) are considered suggestive of high risk SDB. This scale has been validated as an instrument to identify SDB and associate with PSG results to an extent valuable in research.² However, its application in clinical, and more specifically, perioperative care remains untested.

In the operating room, children with SDB are more likely to have airway obstruction, laryngospasm, and other adverse respiratory events. In order to develop a screening questionnaire with more perioperative relevance, Tait et al looked at the SRBD questions in relation to perioperative respiratory adverse events (PRAE) in children ages 2-17 years old.³ Using a factor analysis, they identified 5 symptoms that were most predictive of PRAE and created the STBUR (Snoring, Trouble Breathing, Un-Refreshed) tool. This study found that a PRAE was 3 times more likely with 3 STBUR symptoms, and 10 times more likely with all 5 symptoms reported. This tool was compared to a positive SRBD scale and PSG, which indicated a 1.9 and 2.6 likelihood of PRAE respectively. A follow up study by the same group revealed that patients with >3 STBUR symptoms had significantly more oxygenation desaturations and experienced more escalation in care during recovery in the setting of receiving equal opioid dosing.⁴ These findings were consistent across all ages and ASA groups. The incidence of desaturation was similar

between those with SDB by STBUR screening and those with positive PSG. Although not validated as a tool diagnostic of OSA, the STBUR questionnaire seems to identify children at risk for PRAE due to SDB in an efficient and cost-effective manner.

The SRBD scale was adapted in another study by Raman et al in to order develop a questionnaire predictive of OSA diagnosed on PSG in children ages 6-18 years old.⁵ This study identified questionnaire answers that correlated with the apnea hypopnea index in patients undergoing PSG. Six questions were identified for a short scale and found to correlate with moderate to severe OSA on PSG. Because questionnaires are subjective and can vary in reliability, this study also explored whether the neck circumference or body mass index consistent with childhood obesity would increase the predictive ability of the questionnaire. These objective measures did not improve upon the questionnaire's ability to identify at-risk patients. Although this approach is consistent with the ASA recommenda-

>> Obstructive Sleep Apnea continues on next page

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Table 1: STBUR and Predictive Short Scale questionnaire to screen pediatric OSA

STBUR Questionnaire ³	Predictive Short Scale Questionnaire ⁵
Snore more than half the time	Snore more than half the time
Loud snoring	Always snoring
Trouble/Struggle to breath	
Stop breathing	Stop breathing
Waking up unrefreshed	
	Bed wetting
	Growth retardation
	Overweight

tion to incorporate physical exam in the screening process and is part of an adult assessment for OSA, these findings highlight the difference between adult and pediatric SDB.

There are several other questionnaires for SDB that are less commonly used for perioperative screening and are not discussed in this article. The OSA-18 questionnaire was developed to document quality of life with SDB but has poorly correlated with PSG.⁶ In addition, the CAS-15 is a standardized history and

physical exam that has been useful in diagnosing SDB in healthy children presenting for possible adenotonsillectomy.⁷

Continued work in this area of pediatric perioperative medicine is needed in order to find a universal, accessible, and validated screening tool that will aid in both the diagnosis of SDB and the prediction of perioperative adverse events in these at-risk patients. A child with SDB may require modification of the anesthetic plan, particularly with regards to opioid management and post-operative monitoring.

Many institutions have adopted some type of questionnaire for SDB and incorporated it into the electronic health record and standard preoperative evaluation in order to increase screening compliance by the anesthesia providers. In addition, there are

ongoing quality improvement projects across the nation looking at how screening for SDB is affecting patient safety and outcomes in our children.

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The Weight of Obstructive Sleep Apnea

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“Will my sleep apnea go away once I lose weight?” is a question frequently asked by patients in the sleep clinic who are undergoing preparation for bariatric surgery. My answer is often, “I hope so, but it depends.” Obesity is a known risk factor for obstructive sleep apnea (OSA). In the United States, approximately forty percent of adults are obese (body mass index, BMI, ≥ 30 kg/m²) and nearly one third are overweight (BMI 25-29.9 kg/m²).¹

Among individuals with OSA, as many as 90% may be overweight, and the prevalence of OSA increases with the severity of obesity.² Peppard and colleagues modeled the prevalence of OSA based on age, gender, and BMI, using data from the Wisconsin Sleep Cohort and the National Health and Nutrition Examination Survey (NHANES).³ Moderate-to-severe OSA is relatively uncommon among individuals with a BMI under 25 kg/m². When one’s BMI is 40 kg/m² or higher, moderate-to-severe OSA occurs in approximately half of adult males and nearly one third of females over age 50 (table 1). In the bariatric surgery population, studies show prevalence rates near 70% and some as high as 90%.⁴⁻⁶

What are the effects of weight that increase the risk obstructive sleep apnea (Figure 1)? Tissue deposition in the pharyngeal fat pads, tongue and neck can narrow the upper airway. While the tone of airway dilator muscles may be able to compensate for this narrowing during wakefulness, attenuation of this muscle activity during sleep can predispose the airway to collapse.⁷ Central obesity can result in cephalad displacement of the diaphragm and reduction in lung volume, with resultant loss of the “tracheal tug,” a caudally-directed traction force that contributes to airway stability.⁸

In addition to the physical effects of adipose tissue, a “chemical effect” may also

Table 1. Model-based Prevalence Estimates of Moderate-to-Severe Sleep-Disordered Breathing, Wisconsin Sleep Cohort Study, 1988-2011

BMI (kg/m ²)	Male (30-49 years)	Male (50-70 years)	Female (30-49 years)	Female (50-70 years)
<25	0.93%	3.6%	0.18%	1.44%
25-29.9	3.8%	10.6%	0.73%	4.5%
30-39.9	16.6%	29%	3.6%	13.9%
≥ 40	55%	56%	18.6%	33.5%

Peppard, P.E., et al., *Am J Epidemiol.*, 2013;177

contribute to airway collapse. Obesity induces an inflammatory state, and elevated levels of pro-inflammatory cytokines such as TNF- α may affect the central nervous system response to airway obstruction.⁹

For many people trying to lose weight, reducing their burden of medical comorbidities is often a priority. Patients may say, “I want to lose weight so that I don’t need to use insulin” or “my sister’s sleep apnea went away after surgery and I hope mine will too.” Given that obesity is a modifiable risk factor for OSA, encouraging words are often reasonable during these visits.

Over three decades ago, Smith and colleagues reported that weight loss in obese individuals with severe OSA (mean AHI 55/hr) resulted in a significant improvement in their AHI (mean AHI 29/hr).¹⁰ Subjects had a mean weight of 150% their ideal body weight at baseline and were re-evaluated once they had lost at least 5% of their starting weight. More recently, a Finnish study evaluating weight loss via diet and a cognitive behavioral program showed that the ODI₄ (4% desaturation criteria) was reduced by 50% or more in over 61% of

subjects who had lost weight at 6 months (mean weight loss 14% body weight).¹¹ The degree of weight loss correlated with the reduction in ODI. A review of medical therapy for treatment of OSA conducted by the OSA task force (AASM) concluded that dietary modification can result in significant weight loss and that this will likely result in significant improvement in OSA.¹²

Bariatric surgery is becoming an increasingly popular method of weight loss, with nearly 500,000 surgeries performed an-

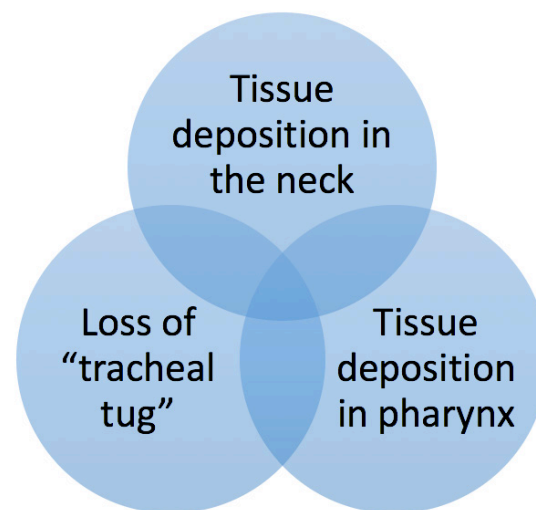


Figure 1. Mechanical factors contributing to OSA in obesity

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nally, worldwide. Approximately half of these occur in Canada and the United States. Some individuals will lose as much as 80% of their excess body weight. Both obesity and obstructive sleep apnea are risk factors for post-operative complications. Consensus guidelines regarding the perioperative management of OSA in bariatric surgery were recently published, and offer recommendations regarding testing, treatment, post-operative monitoring, anesthetic management, and follow up.¹³ These guidelines also include a recommendation regarding screening for obesity hypoventilation syndrome (OHS), a comorbidity which further increases the risk of post-operative complications.^{13,14}

An updated Cochrane review comparing surgical versus non-surgical methods of weight loss indicates that weight loss was higher in patients undergoing surgery and that the burden of type 2 diabetes was reduced to a greater extent in this group.¹⁵ A systematic review and meta-analysis showed a weighted mean decrease (WMD) in BMI of 13 kg/m² and a WMD in AHI of 26 events/hr among high quality studies (Newcastle-Ottawa scale score 7 or greater) evaluating surgical methods for weight loss.¹⁶ Subjects undergoing non-surgical weight loss had a WMD in AHI of 6.2/hr and WMD in BMI of 2.9 kg/m². Most of the individual studies had follow up 1-2 years after the initial intervention and thus more data is needed to evaluate the long-term effects of weight loss and specific weight loss methods on OSA.

To the question of “how much weight matters?” we gain insight from the Wisconsin Sleep Cohort Study. A 10% weight loss predicted a 26% reduction in the baseline AHI, and even a 5% weight loss predicted a 15% drop in AHI as compared to baseline.¹⁷ As one would expect, weight gain

was predictive of increases in AHI and increased the odds of developing moderate-to-severe OSA.

While the likelihood of OSA resolving completely with weight loss depends on a number of factors, for any individual we can try to offer more specific answers. A patient with moderate OSA and few risk factors aside from obesity may have a high likelihood of their OSA resolving if they lose 20% of their baseline weight. On the other hand, older individuals with severe OSA and multiple risk factors may not achieve full resolution. However, weight loss may allow for reduction of PAP pressure settings, or may obviate the need for supplemental oxygen in addition to PAP therapy. Patients should be informed that weight loss is not a guarantee their sleep apnea will resolve, and the recommendation should be made that they follow up with a sleep provider for evaluation and not simply discontinue PAP therapy on their own.¹³

As perioperative providers we can offer guidance to our patients regarding testing and treatment for sleep-disordered breathing, and education to our colleagues regarding the medical and post-operative implications of OSA and its management. Although both obesity and OSA are prevalent, we can be encouraged by the positive effects of weight loss, and can pass some of that encouragement on to our patients.

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Predictive Monitoring in Sedation: Looking (twice) into the Future



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The title has a double meaning because there are improvements to be made in overall patient monitoring during surgery, specifically for monitoring sedation procedures performed outside the operating room. The future has arrived, now requiring new specific predictive sedation monitoring systems that objectively assess, in real-time, if a patient is sedated adequately. Specifically, is the patient unconscious but easily arousable, pain and stress free during the procedure, not over or under sedated as measured by vital signs, and oxygen saturation. Adequately sedated also means that the procedure achieves the desired outcome by the procedural doctor, while avoiding the risks of under-sedation (patient recall or movement) or over-sedation (respiratory depression, airway obstruction, hemodynamic instability, or delayed awakening. Such risks could result in hospital or ICU admission (See Table 1).

The purpose of this article is to discuss future possibilities of predictive monitoring technology in the area of sedation.

How monitoring during sedation procedures currently works

After examination of the patient, monitoring begins by checking baseline conditions. Initial drug dosing is adjusted according to the specifics of the patient: age, weight, height, and gender. These are the factors that determine the amount of propofol or remifentanyl started, the two most common iv drugs used. Based on clinical experience, once an “adequate” level of sedation has been achieved, the procedure starts. Again, based on the

anesthesiologist’s clinical experience with the procedure, drug doses are changed to anticipate potential changes in intensity of noxious stimulation, or increased or decreased, depending on the vital signs.

The same process is repeated many times a day, in many different areas of the hospital, wherever sedation is required. In fact, the same process is repeated in different hospitals or outpatient facilities, in this country, and around the world.

Looking Twice Into The Future

All the information from each patient’s infusion pump and the clinical monitors, describes how the patient responded during the procedure? What do we do with all that patient-specific information? That information might help us predict how fast the patient was discharged to a ward or from the hospital? What side effects, if any, do we analyze against all this patient specific data? Essentially, we do nothing. Even

if it stays in some hard drive or on some “cloud of data” as part of the patient’s Electronic Health Record, at best, it remains in our memory, part of our personal experience. It is not used objectively for future care, should the same patient need another procedure.

Future- First Look- What if we could use real-time information for ongoing patient care?

What if continuous real-time information about the patient and the procedure could be available to clinicians and researchers? What if it could be stored and ready to use immediately? For both population studies, and individual patients, we could identify which are the main factors affecting the relationship between, drugs, drug effects, and noxious stimulation during the procedure. Maybe factors other than age or weight could be important, and the specific contribution of each identified. This objective information would come from

<i>Table 1. Optimal sedation vs under or over sedation. Characteristics, clinical signs, and consequences</i>		
UNDER-SEDATION	OPTIMAL SEDATION	OVER-SEDATION
Awareness	Unconscious AROUSABLE	Unconscious NO AROUSABLE
Stress	No stress	No stress
Pain	No response to painful stimulation	No response to painful stimulation
GAG or Movement	No movement	No movement
Adequate SpO2	Adequate SpO2	Hypoxia
Hyperventilation	Normal Breathing	Hypopnea or Apnea
Hypocarbica	Normocarbica	Hypercarbica
Hypertension	Normotensive	Hypotension
Tachycardia	Control of pain (PACU)	General anesthesia
Side effects in PACU	Discharged home	Delayed awakening
Prolonged recovery		Prolonged recovery
Possible lesions derived		Admission to Hospital (ward, ICU)
Incomplete diagnosis		Procedure non completed

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hundreds to thousands of different subjects undergoing similar procedures under sedation. With millions of data points, we could faster and more objectively condense our models of dose, response to intravenous drugs under different levels of sedation, intensity of noxious stimulation, different patient characteristics (age, weight, gender, even different acute or chronic diseases), and more.

Dosing guidelines for anesthesiologists for propofol, remifentanyl, rocuronium, just to name a representative drug from each family, have been designed based on pharmacokinetic-pharmacodynamic (PKPD) models. Given the current advances in data storage and computing power, infusion rates can now be changed in real time using data from the patient monitors. For those doing research in sedation, the amount of real time data available to them increases dramatically.

[Future- Second Look- Advancing the mathematical models used to predict drug effects](#)

Outside of medicine we are used to seeing predictive information from mathematical

models based on previously collected data. Think on the weather forecast, it not only tells us about what is the weather right now but, based on different factors, it predicts the weather we can expect tomorrow. Should we bring an umbrella or wear a short sleeve shirt.

The same analogy can now, and should be, applied in clinical monitoring. The clinician can now see the current state of the patient, but the current state of predictive monitoring systems can and should also be displayed, predicting what is likely to happen to the patient ahead of time, in the very near future. Like the weather, it may not be perfect, but we now have the capacity to do it better than ever before. It should be possible to anticipate the level of sedation required in a predefined time window. Wouldn't it be clinically interesting to know what the rate of infusion of propofol needs to be, for instance, in 2.6 minutes, so there is a 90% probability of avoiding severe respiratory depression? 2.6 minutes could be enough time to adjust the propofol infusion to avoid clinically significant respiratory depression.

Almost any signal coming from the body can be measured. Our ability to collect and store large amounts of data from patients is now possible. We are now in a "big data" revolution. The methods to extract potentially useful information from vast amounts of clinical data have dramatically evolved thanks to sophisticated modeling techniques. Not every answer comes from a bell-shaped curve. There is a vast world of mathematical analytical methods using artificial intelligence derived methods that can be applied in the clinical setting to improve patient care. These possibilities to improve clinical care will change thanks to technology looking towards horizons that we did not even considered some years ago.

The future is closer every day. Some parts are already here. One aspect of this "upcoming future" is that we can now improve patient care by using real-time predictive analytics for our patients under sedation. By using new monitoring systems, with new predictive dimensions in monitoring, we are looking at a better future for our patients?

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- 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

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 - Showing special support for SASM. This donation is inclusive of annual membership and available for all classes of membership.
- ▶ **Active Member - \$115**
 - Physician and scientist members with the ability to vote, hold office and serve as Directors on the Board.
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