Recognition of obstructive sleep apnea in pregnancy survey

The prevalence of obstructive sleep apnea (OSA) in pregnancy in the USA is 11–20%, with the highest prevalence among obese gravidas.[1,2] Diagnosis of OSA can be obscured by pregnancy-related factors. Recently, there has been interest in OSA in pregnancy, particularly as the incidence of obesity has increased. Several studies and a meta-analysis suggest that OSA is associated with adverse perinatal outcomes, particularly when associated with hypertensive disorders of pregnancy which are major causes of maternal and fetal morbidity and mortality and result in significant healthcare expenditure.[1–5] Physicians and other healthcare providers who care for pregnant women should recognize symptoms and risk factors for OSA to enable effective screening. Research is needed to evaluate valid screening tools in the pregnant population.

On behalf of the Society of Anesthesia and Sleep Medicine, we designed an on-line anonymous survey to assess "Recognition of OSA in Pregnancy". After Institutional Review Board exemption, a 12-question, web-based survey was distributed in English to all members of Society for Obstetric Anesthesia and Perinatology (SOAP) via e-mail in January 2015 and again in February 2015. Questions were asked about institutional guidelines and screening tools used to detect and manage OSA. They were in multiple-choice format, and respondents were able to write comments for selected questions. Responses were collected anonymously and analyzed by Survey Monkey® (Appendix A).

The survey was sent to 1038 SOAP members and opened by 51.6% of recipients (n=536). The overall response rate was 285 (27.5%). Respondents were mostly attending/consultant physicians (91.2%) who practise anesthesiology (97.9%) in the USA (83.2%) and Canada (10.5%). Over 91% of respondents believe that OSA in pregnancy is clinically relevant and 86.8% believe that management and treatment improves maternal and/or neonatal outcomes. However, most respondents (82.7%) said that their departments do not have perinatal OSA management guidelines. Guidelines for pregnancy (7.7%) or during labor (9.5%) exist in few departments. Over 21% of respondents routinely screen for OSA in pregnancy and 35.4% only screen if patients are deemed at-risk. Over 42% of respondents never screen for OSA. Of the 163 respondents who do screen, 154 indicated the method used. The most common were STOP-BANG (77.3%) or STOP questionnaires (12.3%).[6,7] A small number use the Epworth Sleepiness Scale (ESS) (1.9%), Berlin questionnaire (BQ) (0.6%), an in-house screening tool (1.3%) or clinical judgment based on review of systems (6.5%).[8,9] The most common questions asked by respondents regarding OSA symptoms are "prior history of OSA", "night-time snoring" and "observed apneas" (Fig. 1). Respondents were also asked what diagnoses might make them concerned that a pregnant patient was at-risk for OSA (Table 1). The most common responses were obesity, difficult airway on examination and essential hypertension. Interestingly, 89.4% of respondents do not withhold neuraxial opioids for pregnant women with OSA.

To our knowledge, this is the first survey of obstetric anesthesiologists to evaluate recognition of OSA in pregnancy and current use of OSA screening tools and guidelines. The survey reflects the knowledge and opinions of senior obstetric anesthesiologists, located primarily in North America, towards OSA in pregnancy. Importantly, it ascertains that many clinicians agree that OSA in pregnancy is a clinically relevant comorbidity and, if treated, can potentially improve maternal and neonatal outcomes. The survey shows that many obstetric anesthesiologists perceive pregnant patients at highest risk of OSA are likely to be obese with essential hypertension and potentially have a difficult airway. While this is correct, our survey highlights that many respondents do not recognize other relevant comorbidities, including gestational hypertension, preeclampsia, diabetes mellitus and intrauterine growth restriction.[2]

Symptoms of OSA can be difficult to assess in pregnancy and knowledge of relevant comorbidities is important. The optimal screening tool remains unknown. A positive screen on the BQ or ESS is poorly predictive in pregnancy, and is associated with a high false referral rate.[10] Similarly, Lockhart et al. showed that none of the current screening tools accurately detect OSA in the third trimester.[11] The majority of respondents who screen parturients for OSA use tools that have been shown to be ineffective. We believe notable differences exist between the general surgical and obstetric populations, that recognition of OSA in pregnancy is vital, and that a specific screening tool should be validated.

Guidelines and protocols benefit patient care and promote patient safety. Over 80% of respondents claim not to have departmental guidelines for the management of OSA in pregnant women. This may be explained by the current paucity of available data and/or lack of awareness of the condition. One area that requires further consideration for potential guidelines is peripartum opioid administration. Neuraxial opioid administration in obstetric practice is deemed safe;[12] most respondents do not withhold neuraxial opioids for pregnant women with OSA. However, providers may need to review this stance as the parturient demographic becomes older and more obese. Obstructive sleep apnea is a significant risk factor for postoperative anoxic brain injury and death in general surgical patients who receive opioid analgesia.[13]

One limitation of our survey is the response rate of 27.5%, yet on-line surveys do achieve lower response

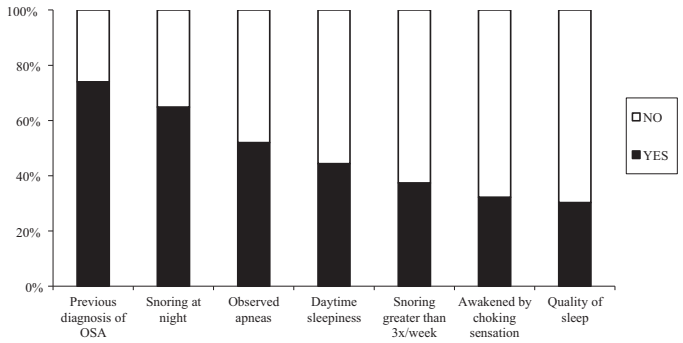

Fig. 1 Questions asked by responders regarding obstructive sleep apnea symptoms

Table 1 Comorbidities in which OSA would be considered

Obesity	98.6%
Essential hypertension	85.2%
Predicted difficult airway	42.4%
Gestational hypertension	29.2%
Preeclampsia	28.1%
Gestational diabetes	22.3%
Intrauterine growth restriction	16.5%

rates than paper surveys.[14] In comparison to other online survey response rates, our e-mail response rate is comparable. Another limitation may be the heterogeneity of the obstetric anesthetic provider respondents' practice (academic vs. private) and locations.

This survey exposes a lack of knowledge regarding OSA in pregnancy among obstetric anesthesiologists. Importantly, it shows that clinicians recognize that OSA is a significant condition whose recognition and management may improve maternal and neonatal outcomes. Anesthesiology providers are key members of a high-risk perinatal care team and must be able to recognize OSA in pregnancy in order to provide appropriate care.

Disclosure

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References

1. Louis J, Auckley D, Miladinovic B, et al. Perinatal outcomes associated with obstructive sleep apnea in obese pregnant women. *Obstet Gynecol* 2012;**120**:1085–92.
2. Louis JM, Mogos MF, Salemi JL, Redline S, Salihu HM. Obstructive sleep apnea and severe maternal-infant morbidity/mortality in the United States, 1998–2009. *Sleep* 2014;**37**:843–9.
3. Liu A, Wen SW, Bottomley J, Walker MC, Smith G. Utilization of health care services of pregnant women complicated by preeclampsia in Ontario. *Hypertens Pregnancy* 2009;**28**:76–84.
4. Facco FL, Ouyang DW, Zee PC, et al. Implications of sleep-disordered breathing in pregnancy. *Am J Obstet Gynecol* 2014;**210**:559. e551–e556.
5. Fung AM, Wilson DL, Lappas M, et al. Effects of maternal obstructive sleep apnoea on fetal growth: a prospective cohort study. *PLoS One* 2013;**8**:e68057.
6. Chung F, Subramanyam R, Liao P, Sasaki E, Shapiro C, Sun Y. High STOP-Bang score indicates a high probability of obstructive sleep apnoea. *Br J Anaesth* 2012;**108**:768–75.
7. Chung F, Yegneswaran B, Liao P, et al. STOP questionnaire: a tool to screen patients for obstructive sleep apnea. *Anesthesiology* 2008;**108**:812–21.
8. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991;**14**:540–5.
9. Netzer NC, Stoohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. *Ann Intern Med* 1999;**131**:485–91.
10. Antony KM, Agrawal A, Arndt ME, et al. Obstructive sleep apnea in pregnancy: reliability of prevalence and prediction estimates. *J Perinatol* 2014;**34**:587–93.
11. Lockhart EM, Ben Abdallah A, Tuuli MG, Leighton BL. Obstructive sleep apnea in pregnancy: assessment of current screening tools. *Obstet Gynecol* 2015;**126**:93–102.

12. Crowgey TR, Dominguez JE, Peterson-Layne C, Allen TK, Muir AS, Habib AS. A retrospective assessment of the incidence of respiratory depression after neuraxial morphine administration for postcesarean delivery analgesia. *Anesth Analg* 2013;**117**:1368–70.
13. Weinger M. Dangers of postoperative opioids. *APSF Newslett*:61–8.
14. Nulty DD. The adequacy of response rates to online and paper surveys: what can be done? *Assess Eval Higher Educ* 2008;**33**:301–14.

Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at http://dx.doi.org/10.1016/j.ijoa.2016.01.003.

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Severe compression of the inferior vena cava during cesarean section

We report a case of extreme fetal metabolic acidosis that was probably caused by severe maternal hypotension secondary to compression of the inferior vena cava (IVC). The mother needed high doses of vasopressors until delivery of the fetus.

A 28-year-old HIV-seropositive patient, weighing 106 kg, with a body mass index of 34.9 kg/m^2 was scheduled for a fourth cesarean section (CS) at term. She had received spinal anesthesia for her previous CS without complication. Long-term medications included atazanavir, ritonavir and emtricitabine/tenofovir. She reported faints when lying supine during the third trimester. No premedication was given. Non-invasive systolic blood pressure (SBP) immediately before induction was 150 mmHg. There were no other features of preeclampsia. Spinal anesthesia was induced in the sitting position using bupivacaine 10 mg, sufentanil 5 μg, morphine 0.1 mg and clonidine 30 μg. An intravenous vasopressor infusion (institutional standard mixture of ephedrine 3 mg/mL and phenylephrine 50 μg/mL) was started at 20 mL/h and a co-load of lactated Ringer's solution 500 mL was commenced when flow of cerebrospinal fluid was obtained through the spinal needle. The patient was then positioned supine with the operating table tilted to the left; blood pressure was measured at 1-min intervals. The upper level of anesthesia was noted to be T4. Within 5 min of spinal injection, SBP decreased to 59 mmHg. This was initially treated by increasing the speed of the vasopressor infusion to 60 mL/h, repeated boluses of ephedrine and infusion of lactated Ringer's solution 1 L. However, SBP remained between 50 and 60 mmHg for 20 min, despite administration of ephedrine 103 mg, phenylephrine 1430 μg and a norepinephrine infusion at 1 mg/h. Surgery was difficult and the baby was delivered 30 min after induction of anesthesia. Apgar scores were 3, 3 and 4 at 1, 5 and 10 min, respectively. Umbilical arterial pH and base excess were 6.67 and −20.3 mmol/L, respectively; umbilical venous pH and base excess were 6.73 and −20.8 mmol/L, respectively. Immediately after delivery the patient became hypertensive with SBP of 180 mmHg for several minutes. The volume of amniotic fluid was estimated at 800 mL. No oxygen desaturation was noted during the perioperative course, there was no loss of consciousness, the electrocardiogram was normal and there were no signs of anaphylaxis. The rest of the procedure was without complication. Postoperatively, maternal blood chemistry and echocardiography were normal. Four hours after delivery the baby's umbilical pH was 7.17, base excess −14 mmol/L and lactate 6.1 mmol/L. The neonate was cooled for four days. On day 5, an electroencephalograph and cerebral magnetic resonance imaging scan were normal and clinical examination was reassuring. At one month, the baby was free of neurologic impairment.

In our case, IVC compression is the presumed mechanism of refractory hypotension as it started soon after moving the patient from the sitting to the supine position, resolved immediately after delivery and no other causes were identified. Obesity and polyhydramnios may have contributed. Similar to a recent report by Murphy et al.[1] delivery of the fetus was the only way to resolve the hemodynamic compromise. If hypotension resistant to high doses of vasopressors occurs, the need for urgent delivery should be communicated to the surgeon. Unfortunately, in our case surgery was difficult because of three previous CS and obesity.

The treatment of hypotension in our case might be questioned as the dose of ephedrine was large and ineffective. The onset of action of ephedrine is slower than that of phenylephrine with its maximum effect taking 2–3 times longer to achieve; thus, the use of ephedrine alone can delay resolution of hypotension. In addition, the large dose of ephedrine may have been a factor contributing to the low umbilical pH at delivery. Previous studies have reported a strong association between high doses of ephedrine and fetal acidosis.[2,3] For this reason, we recommend using phenylephrine when hypotension is persistent or if ephedrine has been given in doses of more than 20–30 mg. Previous studies have reported phenylephrine at total doses close to 3000 μg during CS,[4,5] far greater than the 1430 μg used in this case. Thus, the dose of phenylephrine used may not have been sufficient. Furthermore, our choice of norepinephrine, rather than epinephrine, may be questioned. Onset of action is similar for these two vasopressors but epinephrine is more likely to be rapidly available for bolus