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EDITORIAL

Gestational sleep apnea: have we been caught napping?

Obstructive sleep apnea (OSA) is a modern epidemic whose historical chronology has paralleled the development of the obesity epidemic.[1] Despite the publication of guidelines regarding the perioperative management of patients with OSA, the formation of protocols by hospitals and the adoption of these practices by individual practitioners are typically inadequate.[2,3] This may be even more manifest for OSA in pregnancy. In this issue of the *International Journal of Obstetric Anesthesia*, Dominguez et al. present a survey of members of the Society for Obstetric Anesthesia and Perinatology (SOAP) which focused on OSA-awareness in pregnancy.[4] While most respondents accepted that OSA in pregnancy is a potential problem that can affect maternal and fetal well-being, the vast majority never screen their patients for OSA, and work in hospitals where there is no departmental policy for screening or management of OSA during pregnancy. In the few (<10%) respondents who did screen for OSA, most used the STOP-Bang screening tool, which may have limited utility during pregnancy.[5] It is likely that this study was in a non-representative sample, as reflected in a very low response rate with a high predisposition towards academic practice. Nevertheless, it is unlikely that the situation among non-responders or in private practice is better and may well be worse.

In this editorial, we briefly review what we know about OSA in pregnancy, as compared to the non-pregnant population, and comment on the disparities between what we *can* do and what we *actually* do in this cohort. We ask why we are not doing more and conclude with the suggestion that there is need for a new diagnosis, "gestational sleep apnea", to parallel other established transient diagnoses of pregnancy, like gestational hypertension and gestational diabetes mellitus.

Obstructive sleep apnea is defined as recurrent episodic cessation or limitation of normal breathing during sleep mainly due to exaggerated depression of pharyngeal muscle tone. Sleep-disordered breathing (SDB) is the term used to capture the spectrum of the condition whereby OSA is a manifestation of its severity. The syndrome of OSA arises from increased sympathetic output from intermittent asphyxia and repeated nocturnal arousal.[6] This results in increased arterial stiffness and endothelial dysfunction, which may lead to pulmonary hypertension and cor pulmonale. The etiology of pregnancy-related SDB is multifactorial[7] due in part to generalized hyperemia (with nasal congestion and airway edema) along with decreased functional residual capacity. This may be partially offset by a progesterone-mediated ventilatory stimulation and a preference for lateral sleep posture in pregnancy.[8,9]

Recently, OSA has been more widely appreciated as endemic in the general female population,[10,11] particularly during pregnancy[12] and especially in the presence of comorbidities.[13] Surveys and home monitoring devices have documented the prevalence of OSA during pregnancy[12] but few studies have quantified pregnancy-associated OSA with overnight in-lab polysomnography (PSG), which is the gold standard of diagnosis.[14,15] Based on currently available evidence, SDB seems to occur with a prevalence of 15% in healthy obese pregnant women[16] and 50% in high-risk pregnancies.[17]

Pregnancy-related morbidity related to OSA has been well established.[18] Maternal adverse effects include a more than five-fold increase in in-hospital mortality due to multiple diagnoses including cardiomyopathy and pulmonary embolism.[16] The occurrence of SDB during pregnancy has been shown to be an independent risk factor for gestational hypertension and diabetes, even after adjusting for multiple confounding variables.[17] Indeed, some have suggested PSG referral for all pregnant women with fetal growth restriction and preeclampsia with snoring or obesity.[8] Certainly, poor fetal outcomes including fetal growth restriction and preterm delivery have been reported to be associated with SDB during pregnancy.[18–20]

Although there is evidence of OSA abatement after pregnancy,[21] the immediate postpartum period still presents a risk for increased respiratory-related maternal morbidity. A state-wide study of anesthesia-related maternal deaths over a 15-year period demonstrated that unrecognized hypoventilation or obstruction was associated with both OSA/SDB and obesity.[22] In addition, the administration of postpartum opioids has been shown to be significantly correlated with increased apnea-hypopnea index (AHI) in the supine position.[23] Nevertheless, it has not been possible to find an association between the use of neuraxial opioids in surgical patients with OSA and adverse respiratory outcomes[24] and neuraxial opioids are probably safer than systemic opioids in these patients.[24]

In the non-pregnant adult population with OSA, local and national protocols have been proposed for

screening, diagnosis, therapy and general peri-anesthetic care.[25–27] These include identification of patients for whom continuous positive airway pressure (CPAP) is indicated and recommendations for the use of home CPAP in the hospital setting. However, recent studies report poor knowledge of and poor compliance with these protocols.[2,3] There seems to be a disparity between what we *can* do about OSA and what we *actually* do. Dominquez et al. alert us to an even greater disparity in the pregnant population.[4] For example, physicians and patients may attribute daytime somnolence to physiological changes of pregnancy rather than to gestational sleep apnea. Others might regard gestational sleep apnea as being too transient an entity to warrant referral to a sleep center, particularly as PSG studies and the initiation of CPAP therapy is time consuming, labor-intensive and expensive.

It is important to establish effectively the true prevalence of OSA across the spectrum of pregnancy; clearly not every tired or obese pregnant woman will have gestational sleep apnea. Improving the screening tool for gestational sleep apnea is imperative. Half of the eight questions in the most commonly used tool, the STOP-Bang questionnaire, are of questionable relevance to the pregnant woman (age >50 years, gender, day-time somnolence and neck circumference). While the Berlin questionnaire may possibly have more validity in this cohort, objective measures such as serum bicarbonate[28] or the difference between wakeful and sleep carbon dioxide may improve disease identification. Other improvements in screening, diagnosis and management may include the use of home sleep studies to improve access to definitive diagnostic tests, the standardization of recommendations for sleep position in pregnancy, and the implementation of criteria for CPAP administration during pregnancy. All of these guidelines need to be better researched and implemented.

Increasing diagnosis and therapy for any disease is invariably associated with escalating costs in both manpower and resources. The justification must be based on evidence. Clinical research studies in non-pregnant human subjects with sleep apnea requires a huge effort; this is even more challenging during pregnancy as there is a vulnerable cohort with a limited time window for study. Population studies in pregnancy are hampered by the fact that gestational sleep apnea has no diagnostic code, disease classification or severity index. Like the other established transient diagnoses of pregnancy, gestational diabetes and gestational hypertension, the establishment of a diagnostic code of gestational sleep apnea will 1) require diagnostic criteria, 2) stimulate specific therapies to improve maternal and neonatal outcome, 3) facilitate surveillance in the peripartum and postnatal period, and 4) allow researchers to trace the epidemiologic course of the condition, both its genesis and its path into chronicity. We believe that the time has come for our profession to wake up to this diagnosis.

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