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COMMENTARY

Timing is everything: snoring onset and blood pressure trajectories in pregnancy

Commentary on Dunietz GL, Hao W, Shedden K, et al. Maternal habitual snoring and blood pressure trajectories in pregnancy. *J Clin Sleep Med*. 2022;18(1):31–38. doi:10.5664/jcsm.9474

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Sleep-disordered breathing (SDB) is a highly prevalent condition in pregnancy and increases risk for pregnancy complications including hypertensive disorders of pregnancy (HDP).[1–3] HDP affect 1 out of 10 pregnancies and are associated with significant maternal morbidity and mortality.[4,5] Despite the association between SDB and HDP across study designs, many questions remain, including how SDB is related to when, and how, blood pressure (BP) changes occur across gestation. Such knowledge can advance the science by shedding light on monitoring and therapeutic strategies in women with chronic habitual snoring or pregnancy-onset snoring. In this issue of the *Journal of Clinical Sleep Medicine*, a paper by Dunietz et al[6] surveyed 1,305 pregnant women in the third trimester investigating the presence of frequent snoring (≥ 3 nights/week, a characteristic symptom of SDB) prior to and during pregnancy and extracted BP measurements collected throughout pregnancy from medical records. The vast majority of participants had at least 10 measurements during the course of pregnancy. Results from this study showed that women with pregnancy-onset snoring and women with chronic habitual snoring had higher maternal BP across gestation when compared with women without habitual snoring. Importantly, patterns of BP trajectories differed between women with pregnancy-onset vs chronic habitual snoring, such that women with pregnancy-onset snoring had higher systolic BP at 18 weeks' gestation and in the third trimester relative to women with chronic snoring. The authors conclude that divergent BP trajectories between these groups may provide an opportunity for early identification and possible interventions for pregnant women at risk to develop HDP.

Results from this study have important implications for prenatal care. First, the study emphasizes the importance of screening for chronic and pregnancy-onset habitual snoring early in pregnancy, as early identification may signal risk for women at risk to develop HDP. Unfortunately, historically, SDB is underdetected and undertreated in pregnancy,[7,8] representing a missed opportunity to improve maternal and neonatal health. For those women who endorse pregnancy-onset habitual snoring, prenatal care planning could mirror protocols in place for women at risk for HDP, including frequent BP surveillance, prophylactic low-dose aspirin administration, and fetal growth monitoring. This approach to clinical care may be particularly helpful given results that women with pregnancy-onset snoring displayed BP trajectories that diverged at 18 weeks' gestation, weeks prior to a typical diagnosis of gestational hypertension. Last, results from this study present interesting data on BP trajectories rather than the conventional BP cutoff criteria ≥ 140/90 mmHg.[9] This cutoff has been recently revisited with the suggestion of an alternative threshold following the new definition of hypertension in the general population of 130/80 mmHg, although no change in thresholds has made it into any hypertension-in-pregnancy practice guidelines. It is unclear at this point whether trajectories of BP changes over gestation are a more sensitive indicator of risk for HDP or its associated pathology. In addition, the significance of the cutoff of 1 mm for divergence of BP trajectories is debated. While some data in the general population suggested that a reduction of 1 mmHg in systolic BP reduced the risk of stroke by 5%,[10] other data failed to show a similar benefit. In addition, the Control of Hypertension In Pregnancy Study (CHIPS) trial that demonstrated a difference of 5.8 mmHg systolic and 4.6 mmHg diastolic pressures between the tight and less tight BP target groups in pregnancy did not show any difference in perinatal outcomes, except for differences in the frequency of severe maternal hypertension.[11] Further, ambulatory monitoring data for 48 consecutive hours every 4 weeks in women with normotensive and complicated pregnancies demonstrated an average 8% BP increase starting in midgestation in normotensive women and a 9–13% increase in complicated pregnancies,[12] many-fold higher than the cutoff proposed in the present study.

As the authors have acknowledged, the study is limited by self-report of snoring and recall bias. Pregnant women were recruited in late pregnancy and asked to recall the onset of their snoring and estimate the trimester of pregnancy at which snoring had started. It is possible that this recall bias may have impacted the classification of pregnant women by snoring status. Further, the recall design limited the ability to examine the temporal relationship between the onset of snoring and the

development of HDP. Such a relationship would have been important in assessing causality in the association of SDB and HDP. It has been argued that the association may be bidirectional, based on biological plausibility for either pathway.[13] It is possible that pregnancy-onset snoring may occur as a downstream phenomenon of HDP, rather than a causal phenomenon, whereas snoring predating pregnancy is a potential causal factor in the association. However, these questions need to be better explored in longitudinal studies that screen for SDB and BP measurements at various time points in pregnancy. Future studies also need to investigate nocturnal measurements and measurements outside of the clinical practice. Nocturnal measurements obtained with ambulatory BP monitoring, such as the nocturnal dip in BP, have a predictive ability for HDP.[14,15] Nevertheless, conventional ambulatory BP monitoring may be difficult to implement at intervals as frequent as those in this study due to the inconvenience and potential discomfort of repeated measures during the day and at night.

In summary, this study brings us closer to understanding differences between gestational-onset SDB and SDB predating pregnancy as they relate to hypertensive disorders of pregnancy, and the importance of frequent screening for these disorders. These data may also be important in the identification of a window in gestation for the implementation of therapeutic strategies aimed at the prevention of cardiovascular outcomes such as HDP. Periodic screening for these disorders during gestation, rather than at a single time point in pregnancy, may impact clinical monitoring protocols for the development of HDP. Future studies need to focus on a better understanding of dynamic mechanisms that link pregnancy-onset SDB and SDB that predates pregnancy to HDP, as the 2 disorders appear to diverge in terms of cardiovascular measures and perinatal outcomes.

CITATION

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Both authors have seen and approved the final manuscript. The authors report no conflicts of interest.