Hypertension, snoring, and obstructive sleep apnoea during pregnancy: a cohort study

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Objective To assess the frequency of obstructive sleep apnoea among women with and without hypertensive disorders of pregnancy.

Design Cohort study.

Setting Obstetric clinics at an academic medical centre.

Population Pregnant women with hypertensive disorders (chronic hypertension, gestational hypertension, or pre-eclampsia) and women who were normotensive.

Methods Women completed a questionnaire about habitual snoring and underwent overnight ambulatory polysomnography.

Main outcome measures The presence and severity of obstructive sleep apnoea.

Results Obstructive sleep apnoea was found among 21 of 51 women with hypertensive disorders (41%), but in only three of 16 women who were normotensive (19%, chi-square test, $P = 0.05$). [Author correction added on 16 June 2014, after first online publication: Results mentioned in the abstract were amended.] Non-snoring women with hypertensive disorders typically had mild obstructive sleep apnoea, but >25% of snoring women with hypertensive disorders had moderate to severe obstructive sleep apnoea. Among women with hypertensive disorders, the mean apnoea/hypopnoea index was substantially higher in snorers than in non-snorers (19.9 ± 34.1 versus 3.4 ± 3.1, $P = 0.013$), and the oxyhaemoglobin saturation nadir was significantly lower (86.4 ± 6.6 versus 90.2 ± 3.5, $P = 0.021$). Among women with hypertensive disorders, after stratification by obesity, the pooled relative risk for obstructive sleep apnoea in snoring women with hypertension compared with non-snoring women with hypertension was 2.0 (95% CI 1.4–2.8).

Conclusions Pregnant women with hypertension are at high risk for unrecognised obstructive sleep apnoea. Although longitudinal and intervention studies are urgently needed, given the known relationship between obstructive sleep apnoea and hypertension in the general population, it would seem pertinent that hypertensive pregnant women who snore should be tested for obstructive sleep apnoea, a condition believed to cause or promote hypertension.

Keywords Hypertension, obstructive sleep apnoea, pregnancy, snoring.

Linked article This article is commented on by Facco F. p. 1694 in this issue. To view this mini commentary visit http://dx.doi.org/10.1111/1471-0528.12888.

Introduction

Hypertensive disorders affect approximately 10% of pregnancies and increase the risk for adverse outcomes.[1] In addition, they substantially increase healthcare costs.[2] Of particular concern is pre-eclampsia, characterised by new-onset hypertension and proteinuria after 20 weeks of gestation.[3] Prompt recognition, evaluation, and management are required to prevent end-organ damage. From a public health perspective, it is alarming that the incidence of pre-eclampsia has increased by almost one-third in the past decade, and is responsible for over 60 000 maternal deaths each year.[4] Moreover, women with pre-eclampsia are at increased risk of cardiovascular disease later in life.[5,6]

In the non-pregnant population, a key contributor to hypertensive disease is obstructive sleep apnoea,[7] a disorder

characterised by nocturnal airway collapse, with the disruption of normal ventilation, hypoxaemia, and sleep fragmentation. The prevalence of obstructive sleep apnoea in women aged 30–39 years is approximately 6.5%, with moderate or severe obstructive sleep apnoea affecting 1–5%.[8–10] The prevalence of obstructive sleep apnoea also increases with increasing body mass index.[11] Nonetheless, it remains highly under-diagnosed: more than 90% of women with obstructive sleep apnoea do not know that they have it.[12] Treatment of obstructive sleep apnoea, using the gold-standard positive airway pressure, reduces cardiovascular morbidity and mortality, with improvement in daytime and nocturnal blood pressure.[13,14]

Accumulating evidence shows that habitual snoring, the hallmark symptom of obstructive sleep apnoea, increases in frequency during pregnancy,[15–18] and affects up to one-third of women by the third trimester.[17,19] Although the prevalence of objectively documented obstructive sleep apnoea in pregnancy remains unknown, a recent study found that 15% of obese pregnant women have obstructive sleep apnoea in the first trimester.[20] Importantly, most studies that have queried pregnant women about snoring or performed overnight polysomnography (sleep study) demonstrate an association with gestational hypertension and pre-eclampsia.[15,17,19–24] In the largest prospective study to date, we have recently shown that snoring, specifically new-onset snoring, during pregnancy is independently associated with gestational hypertension and pre-eclampsia, even after accounting for other contributing factors.[17] Moreover, a recent polysomnographic study of women with and without gestational hypertension found a higher frequency of obstructive sleep apnoea in women with gestational hypertension (53 versus 12%, $P < 0.001$).[24]

As treatment for obstructive sleep apnoea is readily available and can reduce blood pressure,[13] confirmation of its frequency during pregnancy and identification of ways to screen for it should be a high priority. The goal of this study was to investigate the frequency of unrecognised obstructive sleep apnoea and symptoms that may help to identify it among hypertensive pregnant women, as compared with healthy pregnant women who were normotensive.

Methods

These data represent the initial analyses of a longitudinal treatment intervention trial in hypertensive pregnancies. Hypertensive pregnant women were recruited from high-risk prenatal clinics or inpatient units within the University of Michigan between March 2009 and July 2013. Women were eligible if they were ≥14 years of age and had a clinical diagnosis of chronic hypertension, gestational hypertension, or pre-eclampsia, as obtained from obstetric notes in the medical records, and verified by medical coding

using the ninth revision of the International Classification of Diseases (ICD-9).[25] Women were eligible at any gestational age. For comparison, women who were normotensive, without comorbidities, were recruited from prenatal clinics in a parallel study using identical methods. Written informed consent was obtained from all women. Approval was obtained by the University of Michigan Institutional Review Board (IRBMed).

Upon enrollment, women completed a questionnaire about sleep and symptoms of obstructive sleep apnoea. Participants were asked the following questions.

1 'How often do you snore?' The response options offered were: 'Almost every night'; 'Three or four times per week'; 'One or two times per week'; 'One or two times per month'; or 'Never or almost never'. Habitual snoring was considered present if women reported snoring 'Three or four times per week' or 'Almost every night'.

2 'During this pregnancy have you stopped breathing or gasped for air?' The response options offered were: 'Almost every night'; 'Three or four times per week'; 'One or two times per week'; 'One or two times per month'; or 'Never or almost never.'

3 'If you have snored during this pregnancy, when did the snoring begin?', in order to identify the gestational timing of snoring onset to identify women who had chronic symptoms prior to pregnancy. The response options were: 'Already snored before pregnancy'; 'Snoring started in the first trimester'; 'Snoring started in the second trimester'; or 'Snoring started in the third trimester'.

In addition, all women underwent polysomnography either at home or in the hospital (for women who were in-patients) using a portable device (Medipalm, Braebon, Ontario, Canada, or Embletta Gold, Embla, Bromfield, CO, USA). A trained technologist visited all women, initiated the monitoring, and returned on the following morning to retrieve the equipment and download the data. The following channels were recorded: six-channel electroencephalogram (EEG); submental electromyogram; electro-oculogram; electrocardiogram; nasal and oral airflow (thermistor, nasal pressure transducer); chest and abdominal respiratory movement, using respiratory inductance plethysmography; oxygen saturation (SpO_2); snoring microphone; and body position sensor. All of the sleep studies were manually scored by a single board-certified sleep technician blinded to study group (maternal hypertensive status), and were reviewed by a board-certified sleep physician (AVS), who was also blinded to study group. Scoring followed American Academy of Sleep Medicine recommendations.[26] Sleep duration, based upon standard EEG scoring, was available for $n = 50$ women (75%). Women also had a parallel Watch-PAT study (peripheral arterial tonometry, a wrist-worn finger plethysmograph) at the same time as the ambulatory polysomnography. The Watch-PAT

is a well-validated measure of sleep that uses an inbuilt actigraph to measure sleep duration.[27–30] Thus, sleep duration was calculated from the Watch-PAT for those in whom EEG was unavailable, generally because of technical failures, which are common when full polysomnography is attempted outside a laboratory setting.[31,32] We have previously demonstrated the validity of Watch-PAT in pregnancy.[33] Apnoea was defined by a drop in peak thermistor excursion by >90% of the pre-event baseline, where at least 90% of the duration met the amplitude reduction criteria for apnoea, with a duration of ≥10 seconds. Obstructive apnoea was defined as apnoea with continued respiratory effort. Hypopnoeas were scored if the nasal pressure signal excursion dropped by >50% of baseline for ≥10 seconds with ≥3% desaturation or with an arousal. The apnoea hypopnoea index (AHI) was calculated as the number of apnoeas and hypopnoeas per hour of total sleep time. The presence of mild obstructive sleep apnoea was defined as an AHI ≥5 and <15, moderate obstructive sleep apnoea was defined as an AHI ≥15 and <30, and severe obstructive sleep apnoea was defined as an AHI ≥30.[26]

Medical records were accessed to ascertain key variables, including maternal age, gravidity, parity, gestational age at time of study, height, and weight. As body mass index (BMI) is strongly associated with obstructive sleep apnoea, pre-pregnancy or early first-trimester BMI was categorised according to Institute of Medicine recommendations.[34] The women were classified as underweight (BMI <18.5 kg/m^2), normal weight (BMI 18.5–24.9 kg/m^2), overweight (BMI 25.0–29.9 kg/m^2), or obese (BMI ≥30.0 kg/m^2). In addition, clinical diagnoses and medication status were recorded. In reporting this study, guidelines from the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) group were followed.[35]

Statistical analysis

Data were double entered into a database and checked for outliers and normality of the distribution. Analyses were performed using spss 20 (IBM SPSS Statistics, Armonk, NY, USA). Means and standard deviations (SDs) were provided for normally distributed data, and medians and interquartile ranges (IQRs) were provided for non-normally distributed data. For normally distributed data, between-group comparisons of continuous variables were conducted with Student's *t*-tests (hypertension versus no hypertension) and ANOVA (chronic hypertension versus gestational hypertension versus pre-eclampsia), where appropriate. Dichotomised variables were compared with chi-square tests. Non-normally distributed data were analysed using non-parametric statistics. $P < 0.05$ was considered to be statistically significant. Pooled relative risks were calculated using the Mantel Haenszel method and confidence intervals were calculated using the Greenland–Robin variance formula.[36]

Results

A total of 181 hypertensive pregnant women and 70 healthy women who were normotensive were invited to participate; 53 women with hypertensive disorders (29%) and 18 women who were normotensive (26%) agreed. After signing the consent form, but prior to the overnight study, two women withdrew ($n = 1$ hypertensive and $n = 1$ normotensive) and one woman who was hypertensive delivered her infant. The number of women with hypertensive disorders included in the final analysis was therefore 51, and the number of women who were normotensive was 16. There were no demographic differences nor differences in the frequency of snoring between those who declined participation and those who participated (data not shown). Overall, among women with hypertensive disorders at the time of the overnight study, 59% had chronic hypertension (cHTN), 23% had gestational hypertension (GHTN), and 18% had pre-eclampsia (Pre-E). Demographic data are shown in Table 1.

In total, in comparison with women who were normotensive, women with hypertensive disorders were significantly more likely to report snoring: $n = 31$ (61%) versus $n = 3$ (19%); $P = 0.008$. After stratification by obesity, the pooled relative risk for snoring in women with hypertensive disorders was 3.4 (95% CI 2.7–4.3).

Within the hypertensive group, snoring at least three nights per week was reported by $n = 19$ (63%) of the women with chronic hypertension, $n = 7$ (58%) of the women with GHTN, and $n = 5$ (56%) of the women with Pre-E. Notably, the majority of women with cHTN (as well as women who snored but who were normotensive) reported habitual snoring before pregnancy, whereas those with GHTN and Pre-E who snored were more likely to report the onset of snoring during pregnancy rather than before pregnancy (Table 2). Few women reported that they stopped breathing at night ('witnessed apnoea'), with the exception of GHTN where no woman gave a positive answer, and only one woman in each of the other groups answered positively (3% of cHTN, 11% of Pre-E, and 5% of normotensive).

Results from the evaluation of obstructive sleep apnoea are shown in Table 2. Women with pre-E as well as those with cHTN had the highest number of respiratory events compared with other women, but this did not reach statistical significance. The only statistically significant differences between groups were found with mean SpO$_2$ and SpO$_2$ nadir. A subanalysis of just the women with EEG-based sleep duration measures did not change any of the findings, and so all analyses used the total sample.

Figure 1A illustrates the proportion of women in each threshold of apnoea severity. Approximately half of all women with cHTN and pre-E were found to have unrecognised obstructive sleep apnoea. One-quarter of

Table 1. Demographic data for the total sample

	Normotensive controls (n = 16)	Chronic hypertension (n = 30)	Gestational hypertension (n = 12)	Pre-eclampsia (n = 9)
Mean age (years)	28.1 (9.2)	33.3 (4.0)*	31.9 (6.8)	30.4 (7.1)
Mean pre-pregnancy BMI (kg/m^2)	23.7 (4.8)	41.0 (10.4)*,††,‡	34.1 (11.2)**	31.1 (9.6)
Obese pre-pregnancy (%)	2 (13%)	27 (90%)*,‡‡	8 (67%)*	5 (56%)***
BMI at study entry (kg/m^2)	28.1 (4.7)	43.6 (9.3)*,††,‡‡	37.2 (11.3)**	36.0 (8.2)***
Race (%)				
White	9 (56%)	15 (50%)	9 (75%)	5 (56%)
African american	3 (19%)	15 (50%)	3 (25%)	3 (33%)
Asian	3 (19%)	0 (0%)***	0 (0%)***	0 (0%)***
Biracial	1 (6%)	0 (0%)	0 (0%)	1 (11%)
Gestational age (weeks)	33.8 (3.8)	24.6 (8.1)*,†,‡‡	33.0 (2.9)	30.1 (4.2)
Gravidity	2.5 (1.9)	3.4 (2.4)	2.5 (1.3)	4.6 (3.9)***
Parity	0.8 (1.1)	1.5 (1.7)	0.5 (0.7)‡‡	1.9 (1.6)
First pregnancy (%)	6 (38%)	8 (27%)	4 (33%)	3 (33%)
Diabetes mellitus (%)	0 (0%)	5 (17%)	0 (0%)	1 (11%)
Gestational diabetes (%)	0 (0%)	2 (7%)	1 (8%)	0 (0%)
Previous history of GHTN/Pre-E	0 (0%)	8 (27%)***	1 (8%)	1 (11%)
Smoker (%)	2 (12%)	9 (30%)††	0 (0%)	1 (11%)

Mean and standard deviations are shown in the table; all continuous data were normally-distributed.
*P < 0.001 compared with controls; **P < 0.01 compared with controls; ***P < 0.05 compared with controls.
†P < 0.001 compared with GHTN; ††P < 0.05 compared with GHTN.
‡P = 0.01 compared with pre-eclampsia; ‡‡P < 0.05 compared with pre-eclampsia.

Table 2. Sleep variables

	Normotensive controls (n = 16)	Chronic hypertension (n = 30)	Gestational hypertension (n = 12)	Pre-eclampsia (n = 9)	P
Chronic snoring (n; %)	3 (19%)	16 (53%)	1 (8%)	2 (22%)	
Pregnancy-onset snoring (n; %)	0 (0%)	3 (10%)	6 (50%)	3 (33%)	
TST (mins)	378.5 (60.3)	325.0 (134.0)	362.0 (87.0)	342.0 (227.5)	
AHI	2.5 (3.3)	4.0 (23.5)	2.0 (5.0)	4.0 (7.0)	
OAI	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	0.0 (0.0)	
CAI	0.0 (0.0)	0.0 (0.0)	0.0 (0.1)	0.0 (0.0)	
HI	2.5 (3.3)	3.5 (10.3)	1.0 (4.5)	4.0 (6.0)	
AHI ≥5 (n; %)	3 (19%)	13 (43%)	3 (25%)	5 (56%)	
AHI ≥15 (n; %)	1 (6%)	5 (17%)	2 (17%)	1 (11%)	
AHI ≥30 (n; %)	0 (0%)	4 (13%)	1 (8%)	1 (11%)	
Mean SpO$_2$ (%) (n; %)	96.0 (1.3)	96.0 (2.0)	97.0 (2.0)	95.0 (3.0)	0.023
SpO$_2$ nadir (%) (n; %)	92.0 (2.3)	89.0 (5.5)	90.0 (10.0)	90.0 (4.0)	0.035
SpO$_2$ nadir ≤80% (n; %)	0 (0%)	3 (10%)	1 (8%)	0 (0%)	

AHI, apnoea/hypopnoea index; CAI, central apnoea index; HI, hypopnoea index; IQR, interquartile range; OAI, obstructive apnoea index; SpO$_2$, oxygen saturation; TST, total sleep time.
Continuous data shown as median and IQR as non-normal distribution. Dichotomous data shown as raw numbers and percentage.

those with GHTN had obstructive sleep apnoea compared with one in five women who were normotensive. Of note, approximately 10% of women with hypertensive disorders had severe obstructive sleep apnoea (AHI ≥30). Figure 1B illustrates the proportion of women falling at or below the thresholds of minimum nocturnal oxygen saturation for each group. The majority of women with hypertensive disorders had dips in oxygen saturation below 90%; however,
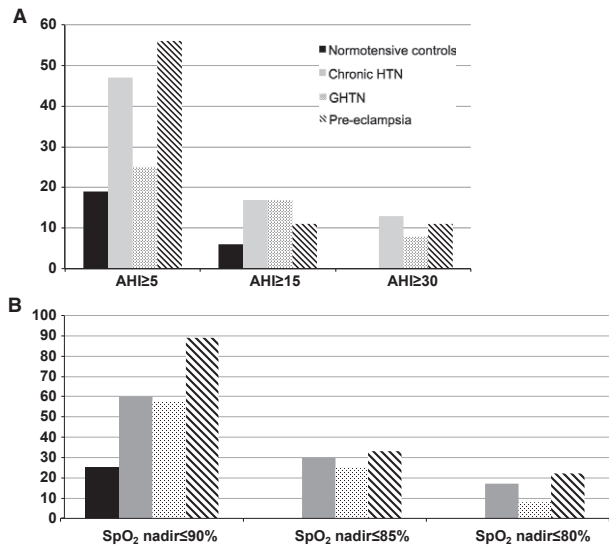
Figure 1. (A) Proportion of women with each threshold of AHI by hypertensive status. (B) Proportion of women falling at or below thresholds of minimum nocturnal oxygen saturation by hypertensive status.

Table 3. Comparison of hypertensive women with and without snoring

	Hypertension and snoring (n = 30)	Hypertension without snoring (n = 21)
AHI ≥5	16 (53%)*	5 (24%)
AHI ≥15	8 (27%)**	0%
AHI ≥30	6 (20%)*	0%
SpO$_2$ nadir ≤80%	6 (20%)*	0%

AHI, apnoea/hypopnoea index; SpO$_2$, oxygen saturation.
*$P = 0.05$; **$P = 0.01$.

almost one in four women with pre-E had nocturnal saturation nadirs below 80%.

Comparisons between hypertensive groups

Analyses were then conducted between snoring and non-snoring women with hypertension. The mean AHI was significantly higher in hypertensive snoring women compared with hypertensive non-snoring women (19.9 ± 34.1 versus 3.4 ± 3.1, $P = 0.013$). Mean SpO$_2$ did not differ between these groups (95.3 ± 1.8 versus 95.7 ± 1.9, $P = 0.40$); however, the SpO$_2$ nadir was significantly lower in women with hypertension and snoring compared with those with hypertension and no snoring (86.4 ± 6.6 versus 90.2 ± 3.5, $P = 0.021$). Notably, women with hypertensive disorders who reported habitual snoring were significantly more likely than non-snoring women with hypertensive disorders to have undiagnosed obstructive sleep apnoea (AHI ≥5; 53 versus 24%, $P = 0.03$; Table 3). After stratification by obesity the pooled relative risk for obstructive sleep apnoea in snoring women with hypertension compared with non-snoring women with hypertension was 2.0 (95% CI 1.4–2.8). Furthermore, approximately one in four hypertensive snoring women had moderate to severe obstructive sleep apnoea, and only the hypertensive, snoring women had oxygen desaturations ≤80%.

Discussion

Main findings

This study demonstrates that a substantial proportion of hypertensive pregnant women have obstructive sleep apnoea,

and that snoring may be an excellent marker in clinical practice for this condition. Although non-snoring women with hypertensive disorders frequently had mild obstructive sleep apnoea, women who self-identified as snorers had moderate to severe obstructive sleep apnoea with clinically significant oxygen desaturation. This suggests that pregnant women with snoring in the setting of hypertension strongly merit evaluation for underlying obstructive sleep apnoea.

Interestingly, in women with hypertension, the timing of snoring onset appeared to be related to hypertension type. Women who reported chronic snoring were most likely to have chronic hypertension, whereas those who reported the onset of snoring during pregnancy were more likely to have gestational hypertension. This finding strengthens similar observations from our prospective study, in which we demonstrated that pregnancy-onset snoring independently predicted gestational hypertension.[17] In contrast, the timing of snoring onset was variable for women with pre-eclampsia.

Strengths and limitations

A major strength of this study is the objective measure of obstructive sleep apnoea via polysomnography in a relatively large sample of women. We chose ambulatory rather than laboratory-based polysomnography because of the inherent difficulties of laboratory-based assessment in pregnancy, particularly for high-risk women. Although laboratory-based monitoring is the 'gold standard', it is not often an option for pregnant women and frequently barriers are encountered in clinical practice. Indeed, previous polysomnographic studies in pregnancy have smaller sample sizes than the current study,[24,37–42] limiting interpretation when results are non-significant. Use of ambulatory polysomnography allowed women to spend the night in their home, minimised potential anxiety and discomfort, and still provided consistent apnoea measures.

In the context of research, home monitoring is widely used, e.g. in National Institutes of Health (NIH) -funded

multicentre epidemiological studies such as the Sleep Heart Health Study and the Study of Women's Health Across the Nation.[43,44] The number of respiratory events obtained from ambulatory monitors is strongly correlated with those obtained from laboratory-based polysomnography, whether or not full polysomnography or cardiopulmonary monitoring is used.[43,45–47] In a meta-analysis home studies were found to provide similar diagnostic information to laboratory polysomnography, but may underestimate the severity of obstructive sleep apnoea.[48] If so, then our findings may have underestimated the severity of obstructive sleep apnoea.

An additional strength was the inclusion of women with chronic hypertension, gestational hypertension, and pre-eclampsia. This allowed the assessment of differential patterns of association with obstructive sleep apnoea, for the first time.

A limitation is that, although we have demonstrated a clear association between hypertension during pregnancy and obstructive sleep apnoea, we were unable to determine causality. We suggest that future studies include a prospective cohort of women that are recruited in early pregnancy and followed through the postnatal period to determine the temporal relationship between hypertension and obstructive sleep apnoea in this population. In addition, large treatment intervention studies in pregnancy using positive airway pressure therapy are also required. Small pilot studies of positive airway pressure in hypertensive pregnancies are encouraging, and suggest that maternal blood pressure can be reduced by treatment of obstructive sleep apnoea;[39,49] however, no large, randomised studies exist.

As discussed below, a wealth of information including randomised clinical trials demonstrates a casual pathway between obstructive sleep apnoea and hypertension in non-pregnant adults,[50] and similar pathways are likely in pregnancy.[51,52] Caution may be warranted in pregnancy, however, because of the occurrence of other physiological changes, as hypertension may promote obstructive sleep apnoea.[53] Secondly, only approximately one-third of women who were invited to take part actually participated. It is possible that those who agreed were more likely to have sleep problems; however, sleep data collected on those who declined participation demonstrated that this was not the case. This suggests that our data are unlikely to have significant bias in this regard.

Interpretation (finding in light of other evidence)
The causal association between snoring/obstructive sleep apnoea has been reported in multiple epidemiological studies of non-pregnant adults.[54–56] It is well established that obstructive sleep apnoea affects autonomic function,[57–59] and subsequently modifies cardiovascular regulation.[60] The cyclic deoxygenation–reoxygenation during repeated apnoeic events, as well as sleep fragmentation from arous-

als, contributes to alterations in the autonomic nervous system. In addition, chronic intermittent hypoxia and exaggerated negative intrathoracic pressure swings can provoke systemic inflammation, oxidative stress, endothelial dysfunction, atherosclerosis, and hypertension.[61,62] In meta-analyses of randomised controlled trials in the non-pregnant population, positive airway pressure, the first-line treatment for obstructive sleep apnoea, has been shown to reduce both daytime and nocturnal blood pressure.[13,14]

Data from pregnant women suggest that snoring/obstructive sleep apnoea is associated with gestational hypertension and pre-eclampsia,[15,17,19–22] as well as fetal growth restriction, operative delivery, prematurity, and admission to the neonatal intensive care unit.[15,20,63–65] We previously demonstrated a clear independent association with pregnancy-onset snoring and maternal hypertension.[17] Moreover, a repeat polysomnographic study of women 1–2 years following gestational hypertension (as well as healthy controls) has shown that women with hypertensive disorders experienced a decrease in the respiratory disturbance index, whereas controls did not.[66] The authors suggested that the physiological effects of pregnancy may have a pathological role in the development of obstructive sleep apnoea during pregnancy in women with gestational hypertension.

The mechanisms of sleep disruption that affect cardiovascular morbidity in non-pregnant individuals are remarkably similar to the biological pathways for pre-eclampsia. Although the pathogenic process for pre-eclampsia is likely to originate in the placenta, the pathways include endothelial dysfunction, oxidative stress, and inflammation.[51] These shared mechanistic pathways have been discussed previously.[52] Nonetheless, although the timing of snoring onset appears to be associated with the timing of hypertension onset, this could also reflect an association in the opposite direction: i.e. snoring could be a manifestation of hypertension or associated oedema. Although this is less likely, the cross-sectional nature of our study precludes a definitive answer.

Our findings are consistent with those of Reid et al.[24] who studied women with gestational hypertension/pre-eclampsia and healthy pregnant controls with laboratory-based polysomnography; this study also found that approximately half of women with hypertensive disorders had undiagnosed obstructive sleep apnoea. No women with chronic hypertension were included.

In the present study, although half of the women with hypertensive disorders had unrecognised obstructive sleep apnoea, those who reported snoring were at particular risk. These women were exclusively found to have moderate to severe obstructive sleep apnoea with significantly lower oxyhaemoglobin nadirs, whereas non-snoring women with hypertensive disorders had only mild obstructive sleep apnoea. This suggests that self-report of habitual snoring in

hypertensive pregnancies may be a useful clinical marker for moderate to severe obstructive sleep apnoea that can be easily ascertained in the clinic. Nonetheless, mild obstructive sleep apnoea may not be benign. In non-pregnant populations mild obstructive sleep apnoea is associated with autonomic alterations and cardiac structural changes.[67–69] Data from pregnant women show that airflow limitation in the absence of obstructive sleep apnoea is frequent in pre-eclampsia,[70] and is reversible with positive airway pressure treatment, with a simultaneous reduction in blood pressure.[49]

A previous study of 24-hour ambulatory blood pressure in 186 hypertensive pregnant women has shown that over half of women also have nocturnal hypertension,[71] and that this was most prevalent in pre-eclampsia (79%) compared with gestational or essential hypertension (45%). Of note, marked augmentation of the haemodynamic response to obstructive apnoeas is observed in pre-eclampsia.[49] In pre-eclampsia, sleep is associated with adverse haemodynamic changes, which can be minimised by positive airway pressure therapy.[40] This suggests that continuous fluctuations of blood pressure caused by nocturnal obstructive events may have relevance to altered blood pressure control, and this has clinical implications for the monitoring and treatment of hypertensive pregnant women.

Conclusion

Approximately half of pregnant women with hypertension may have unrecognised obstructive sleep apnoea. Women presenting with hypertension during pregnancy who also report snoring are at particularly high risk for moderate to severe obstructive sleep apnoea, with clinically significant oxyhaemoglobin desaturation. Although further studies are urgently required, including longitudinal as well as treatment intervention studies, our findings support the need for the obstetric healthcare provider to consider a sleep evaluation in hypertensive pregnancies, especially when snoring is present.

Disclosure of interests

LMO received equipment support from Philips Respironics Inc. and is an advisory board member for the non-profit Star Legacy Foundation. MCC participates in a study that receives equipment from Cura Surgical. RDC receives educational grants from Philips Respironics Inc., Fisher Paykel Inc., receives honoraria as section editor for UpToDate, receives fees for technology licensed by Zansors Inc., is an advisory board member for the non-profit Sweet Dreamzzz Inc., is named in patents owned by the University of Michigan for signal analysis diagnostic algorithms and hardware relevant to the assessment and treatment of sleep disorders, and serves on the Board of Directors of the American Academy of Sleep Medicine, American Board of Sleep Medicine, American Sleep Medicine Foundation, and the International Pediatric Sleep Association. RA received equipment support from Braebon. The other authors have indicated no financial conflicts of interest.

Contribution to authorship

LMO and RDC conceived the study. LMO, RDC, TRBJ, ASB, and MCC designed the study, with input from RA, CG, and CES. LMO, ASB, MCC, and RA contributed to data acquisition. AVS contributed to the analysis of sleep studies and provided all sleep study interpretations. LMO analysed and interpreted the final data, and drafted the article, with all authors providing critical appraisal and final approval of the submitted version.

Details of ethics approval

This study was approved by the University of Michigan Institutional Review Board (IRBMed): HUM#00022596. Initial approval was obtained on 19 May 2009. ClinicalTrials.gov identifier: NCT01029691.

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