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Sleep Disordered Breathing and Gestational Hypertension: Postpartum Follow-up Study

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Background: Gestational hypertension (GH) is a newly recognized risk factor for adverse cardiovascular events later in life. Sleep disordered breathing (SDB) is an established risk factor for adverse cardiovascular events. Recent research has suggested that women with GH may have an increased rate of SDB during pregnancy, but it is not known if this higher rate of SDB persists into the postpartum state.

Objective: To assess whether women with GH continue to have an increased rate of SDB compared to healthy pregnant women, after the physiologic changes of pregnancy resolve.

Methods: We previously studied women with GH and uncomplicated pregnancies with sleep questionnaires and level 1 polysomnography. Participants were invited to participate in repeat testing 1-2 years postpartum. Respiratory disturbance index (RDI) differences were assessed.

Results: Eighteen subjects (11 GH and 7 healthy) had complete follow-up data available for comparison with antepartum data. This group was representative of the initial antepartum cohort. Women with GH experienced a decrease in mean RDI from antepartum to postpartum (12.0 ± 12.3 vs 2.9 ± 2.9; P = 0.02). Healthy women did not experience the same change (2.8 ± 5.3 vs 2.1 ± 3.2; P = 0.81). Postpartum comparisons showed the mean RDI of women with GH had decreased to be similar to that of healthy women (P = 0.75).

Conclusions: SDB in women with gestational hypertension improved in the postpartum state to levels indistinguishable from our healthy subjects. This suggests that the physiologic effects of pregnancy may have had a pathologic role in the development of antepartum SDB in women with GH.

Keywords: Gestational hypertension, postpartum, preeclampsia, sleep disordered breathing

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INTRODUCTION

Gestational hypertension (GH) is a newly recognized risk factor for maternal cardiovascular events later in life.[1] Sleep disordered breathing (SDB) is an established risk factor for adverse cardiovascular events.[2] Compared to women with uncomplicated pregnancies, women with GH complain of lower quality sleep and have increased rates of nasal congestion, snoring, and SDB.[3-5] Pregnancy is known to exacerbate preexisting SDB,[6] but it is not known if the increased rate of SDB in women with GH decreases to baseline after the physiologic changes of pregnancy have resolved. If not, unrecognized and untreated SDB may contribute to the increased cardiovascular risk that is found in women with a history of GH.[7] We had previously reported a higher respiratory disturbance index (RDI) and frequency of SDB (defined as RDI $\geq$ 5) in women with GH compared to women with uncomplicated pregnancies.[4] In this current study we report data from follow-up evaluations of these subjects, performed between one and two years postpartum. The study had two phases: phase one was questionnaire-based, in which we mailed out questionnaires of sleep quality and daytime tiredness to participants in the original study. Along with the questionnaires, we mailed an invitation to return to the sleep laboratory for a full overnight PSG. Results from the questionnaires and PSG were compared to those from the original antepartum study. Our hypothesis was that women with GH would continue to have a higher RDI and higher rate of SDB than women with uncomplicated pregnancies.

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METHODS

Written informed consent was obtained for all patients included in the study protocol, which was approved by the University of Saskatchewan Biomedical Ethics Review Board and registered with the National Institute of Health Clinical Trials Registry (Identifier NCT00259688). Participation was voluntary. For returning mail-in questionnaires, women's names were entered into a draw for one of three iPod Shuffles. An honorarium of $100 was provided for those who agreed to return for a repeat PSG.

Participants

Follow-up questionnaires were mailed to all 60 participants in our original study, between 1-2 years postpartum, with self-addressed, pre-stamped return envelopes. In addition to questionnaires, women were invited to return to the sleep lab for repeat PSG for comparison to their antepartum study. Of the original 60 participants, we were unable to locate 3; 14 never returned questionnaires despite repeated requests; and 8 were excluded because of current pregnancy. The remaining 35 non-pregnant women completed questionnaires, and 19 agreed to return for a repeat PSG. One of these 19 did not have adequate antepartum PSG data, leaving 18 women (11 GH and 7 healthy) for full antepartum to postpartum comparison. This group of 18 women was reasonably representative of the cohort in the original antepartum study (Supplemental Tables S1, S2), the only differences being that healthy women who came in for postpartum assessment had higher ESS (P = 0.01) and snoring prevalence

(P = 0.03) at their antepartum assessment than women without follow-up. One woman in the GH group was still receiving treatment for hypertension. This patient had pre-existing hypertension prior to pregnancy, and her diagnosis of GH was based on pregnancy-related deterioration in hypertensive control. None of the other subjects in either group was being treated for hypertension at the time of follow-up study. All follow-up assessments were completed 10-23 months postpartum, and all women had completed lactating by the time of their follow-up evaluation

Procedures

Baseline data were collected for age, height, weight, body mass index (BMI), and snoring history. Extensive anthropometric data (e.g., neck, chest, and abdominal circumferences as well as Mallampati score, tonsillar size, and maxillary overjet) had been collected for the antepartum study, but none were found to be predictive of SDB in our cohort. We therefore did not collect this data for the follow-up study. Subjects completed a series of short questionnaires, which addressed both sleepiness and common signs or symptoms of SDB. These included the Epworth Sleepiness Score (ESS), the Pittsburgh Sleep Quality Index (PSQI), the Berlin Questionnaire, and a visual analogue scale (VAS) of sleep quality which asked subjects to rate their current sleep on a horizontal scale between "worst" and "best." This was converted to a percentage score, with worst being 0% and best being 100%. For those subjects who consented, the questionnaire assessment was followed by a full-night diagnostic PSG (Sandman 8.0, Tyco Inc., Ottawa, Canada) performed in the Royal University Hospital Sleep Laboratory. PSG channels included electroencephalogram, electrooculogram, submental electromyogram, pulse oximetry, nasal airflow pressure sensor and oronasal thermistor airflow, chest and abdominal excursion by piezoelectric belt, snore vibration sensor, intercostal surface EMG, and anterior tibialis EMG, and electrocardiogram. Studies were scored according the American Academy of Sleep Medicine recommended criteria[8] by a single registered sleep technician who was blinded to clinical data. Accordingly, events were categorized as apneas (a decrease in airflow ≥ 90% from baseline for ≥ 10 sec); hypopneas (decrease in airflow ≥ 30% for ≥ 10 sec and followed by a desaturation of ≥ 4% from the pre-event baseline); and respiratory event-related arousals (RERAs), defined as a sequence of breaths lasting ≥ 10 sec, associated with flattening of the nasal pressure waveform leading to an arousal from sleep, and the sequence does not meet criteria for an apnea or hypopnea. The RDI represented the total number of apneas, hypopneas, and RERAs divided by the hours of sleep.

Data Analysis

Comparisons were conducted between antepartum and postpartum measurements; these comparisons were conducted separately for healthy women and women with GH. Normality was assessed with the Shapiro-Wilk test[9] and QQ plots. Means were compared for normally distributed variables using a repeated measures *t*-test, while the distributions of variables that were not normally distributed were compared with the Wilcoxon signed-rank test. Categorical variables consisting of count data were assessed using the χ^2 test. The analyses presented in the supplemental tables used *t*-tests, Mann-Whitney U tests, and χ^2 tests for normal, non-normal, and categorical data, respectively. A generalized linear model was used to estimate an adjusted mean RDI. Since RDI (dependent variable) was not normally distributed, and the data used to derive RDI is based on counts, Poisson or negative binomial distributions are appropriate for modeling the data. The Poisson distribution makes the restrictive assumption that the mean and variance of RDI are equal,[10] while the negative binomial distribution relaxes this assumption.[11] Mean RDI was 5.5 and variance was 71.3, indicating that a negative binomial distribution was an appropriate choice of distributions. An advantage of the regression model is that mean RDI can be determined while differences in sleep time are explicitly modeled by using TST as the model offset. The regression model is expressed as:

$$\ln\left(\frac{RDI}{TST}\right) = \beta_0 + \beta_1 X_{antepartum}$$

Generalized estimating equations were used to account for repeated measures, using a working correlation matrix to estimate robust standard errors.[12] Model residuals were used to assess fit which confirmed distribution assumptions and variance homogeneity. Data was recorded and stored with Microsoft Excel, and all analysis was conducted with SAS software version 9.2 (SAS Institute Inc., Cary, NC, USA). Statistical significance was assessed using $\alpha = 0.05$.

RESULTS

Women with GH experienced changes in their sleep characteristics between their antepartum and postpartum PSG assessments (Table 1). Notably, the antepartum BMI decreased from 37.6 ± 5.9 kg/m^2 to 32.5 ± 6.8 kg/m^2 (P < 0.01). Sleep efficiency (60.4 ± 16.6 vs. 83.1 ± 16.6 min; P = 0.02), % REM (7.4 ± 5.1 vs. 13.9 ± 6.4; P < 0.01), sleep quality (36.2 ± 14.9 vs. 49.0 ± 23.3; P = 0.01), and TST (235.2 ± 66.7 vs. 363.3 ± 114.1 min; P = 0.01) all increased, while the arousal index decreased (19.5 ± 14.8 vs. 6.0 ± 4.0; P < 0.01). Most importantly, the RDI decreased significantly from antepartum to postpartum (12.0 ± 12.3 vs 2.9 ± 2.9; P = 0.02). Not surprisingly, the healthy women also experienced a decrease in BMI (28.8 ± 4.2 kg/m^2 to 22.8 ± 4.0 kg/m^2; P < 0.01), but otherwise experienced fewer changes in their sleep characteristics than the women with GH (Table 2). Specifically, the change in TST for healthy women was not statistically significant (328.8 + 49.3 to 387.2 + 42.0; P = 0.08), and the RDI decreased non-significantly from 2.8 ± 5.3 to 2.1 ± 3.2 (P = 0.81). Healthy women did spend a significantly higher percentage of sleep in the supine position (22.3 ± 22.6 vs. 45.5 ± 31.6; P = 0.03) compared to antepartum.

The postpartum results for women with GH and healthy women were then compared against each other. Interestingly, despite the women with GH still having a much higher mean BMI (32.5 ± 6.8 vs. 22.8 ± 4.0; P < 0.01), there was no difference in self-reported snoring (45% vs 57%; P = 0.63) or measured RDI (2.9 ± 2.9 vs 2.1 ± 3.2; P = 0.75; Table S3). Antepartum comparisons of women with GH versus healthy women are provided for the interested reader in Table S4.

Having shown that women with GH experience a postpartum decrease in RDI relative to antepartum, while healthy women do not, we sought to obtain an adjusted estimate of RDI. The

adjusted RDI was determined for women with GH and healthy women separately, with a negative binomial regression model. Women with GH demonstrated a decrease in adjusted RDI from 12.7 (95% CI: 10.3-15.5) at antepartum to 3.9 (95% CI: 2.6-6.1) postpartum (Figure 1). In contrast, healthy women had an adjusted antepartum RDI of 3.0 (95% CI: 2.6-3.5) which decreased to 2.3 (2.0-2.5) postpartum (Figure 1).

Table 1—Gestational hypertension antepartum vs. postpartum

Variable	Antepartum (n = 11) Mean ± SD (Median)	Postpartum (n = 11) Mean ± SD (Median)	P-value
Age (year)	27.9 ± 4.7 (27.0)	29.7 ± 4.5 (28.0)	–
Follow-up time (months)	–	17.1 ± 4.4 (16.0)	–
BMI (kg/m^2)	37.6 ± 5.9 (36.2)	32.5 ± 6.8 (32.3)	< 0.01[a]
ESS	6.3 ± 3.2 (5.0)	6.3 ± 3.1 (5.0)	1.00[a]
Sleep quality VAS (%)	36.2 ± 14.9 (30.5)	49.0 ± 23.3 (41.9)	0.01[b]
Snoring (yes/no)	9 (82%)	5 (45%)	0.07[c]
TST (min)	235.2 ± 66.7 (242.0)	363.3 ± 114.1 (408.0)	0.01[a]
Sleep efficiency (%)	60.4 ± 16.6 (61.6)	83.1 ± 16.6 (88.3)	0.02[b]
% Supine (%)	22.2 ± 26.0 (9.9)	36.5 ± 30.0 (35.9)	0.32[b]
% REM (%)	7.4 ± 5.1 (7.3)	13.9 ± 6.4 (13.1)	< 0.01[a]
AHI (events/h)	4.6 ± 9.3 (0.3)	2.6 ± 2.7 (1.3)	0.77[b]
RDI (events/h)	12.0 ± 12.3 (6.4)	2.9 ± 2.9 (1.9)	0.02[b]
Arousal index (events/h)	19.5 ± 14.8 (15.6)	6.0 ± 4.0 (6.1)	< 0.01[b]
Mean SpO_2 (%)	95.7 ± 1.3 (96.0)	96.6 ± 1.5 (96.7)	0.06[a]
Min SpO_2 (%)	90.7 ± 4.0 (93.0)	91.1 ± 5.2 (93.0)	0.63[b]

[a]Paired *t*-test comparison of means. [b]Wilcoxon signed-rank comparison of distribution. [c]Chi-square test comparison of counts. BMI, body mass index; ESS, Epworth Sleepiness Scale; AHI, apnea hypopnea index; RDI, respiratory disturbance index; TST, total sleep time.

Table 2—Healthy subjects antepartum vs. postpartum

Variable	Antepartum (n = 7) Mean ± SD (Median)	Postpartum (n = 7) Mean ± SD (Median)	P-value
Age (year)	29.9 ± 6.9 (32.0)	31.9 ± 6.9 (33.0)	–
Follow-up time (months)	–	17.6 ± 2.8 (18.0)	–
BMI (kg/m^2)	28.8 ± 4.2 (27.2)	22.8 ± 4.0 (21.7)	< 0.01[a]
ESS	11.0 ± 4.0 (10.0)	11.4 ± 4.1 (11.0)	0.08[a]
Snoring (yes/no)	5 (71%)	4 (57%)	0.57[c]
Sleep Quality VAS (%)	52.1 ± 18.7 (53.2)	51.8 ± 25.0 (66.2)	0.81[b]
TST (min)	328.8 ± 49.3 (309.0)	387.2 ± 42.0 (391.8)	0.08[a]
Sleep Efficiency (%)	80.7 ± 9.2 (81.1)	88.2 ± 3.4 (88.4)	0.11[b]
% Supine (%)	22.3 ± 22.6 (10.7)	45.5 ± 31.6 (31.0)	0.03[b]
% REM (%)	15.2 ± 2.4 (15.5)	13.8 ± 3.8 (13.8)	0.50[a]
AHI (events/h)	0.9 ± 2.4 (0.0)	1.1 ± 2.0 (0.3)	0.31[b]
RDI (events/h)	2.8 ± 5.3 (1.2)	2.1 ± 3.2 (1.2)	0.81[b]
Arousal index (events/h)	10.3 ± 6.9 (7.4)	5.6 ± 4.7 (4.8)	0.11[b]
Mean SpO_2 (%)	96.7 ± 1.3 (96.8)	97.0 ± 0.8 (97.3)	0.41[a]
Min SpO_2 (%)	93.0 ± 2.8 (94.0)	93.0 ± 3.7 (95.0)	1.00[b]

[a]Paired *t*-test comparison of means. [b]Wilcoxon signed-rank comparison of distribution. [c]Chi-square test comparison of counts; BMI, body mass index; ESS, Epworth Sleepiness Scale; AHI, apnea hypopnea index; RDI, respiratory disturbance index; TST, total sleep time.

DISCUSSION

We previously found that women with GH have a higher RDI and increased rate of SDB compared to healthy pregnant women,[4] but increased obesity in the GH group was a significant confounding factor. We had postulated for this study that we would find a high degree of SDB persisting in postpartum for the women with GH, which we did not find. Instead, we found that despite the women with GH remaining substantially more obese than the healthy group, their mean RDI decreased to be indistinguishable from the healthy postpartum women. Along the same lines, self-reported snoring was not increased in the postpartum state for women with GH. While the small number of subjects is a limitation, our findings make us consider that pregnancy-related factors, rather than just obesity, may have a causal role in the elevated antepartum RDI observed in GH subjects. It may be that the physiologic changes of pregnancy unmasked a tendency towards SDB, which then remitted once pregnancy was over. We speculate that perhaps this propensity towards SDB could be again unmasked later in life with age-related physiologic changes, menopause and/or weight gain.

Obesity is an established risk factor for SDB and this remained a characteristic of the GH group into the postpartum period. However, there are many other anthropometric and physiologic (e.g., fluid retention) features that can have a contributory role in SDB and many of these may be modifiable by pregnancy. While we failed to find predictive value for any of these features during our antepartum study, this may have been a consequence of our small sample size and we believe that these features should be evaluated in future, larger studies in this area.

The short-term follow up nature of this study has both advantages and disadvantages. The advantage is that it allows for a reasonable assessment of the effect of pregnancy physiology on sleep and breathing. However, we cannot say whether or not the improvement in SDB we observed in the first two years postpartum will be maintained as our subjects age. Therefore, our study does not assess whether the occurrence of SDB is contemporaneous with known elevated cardiovascular event rate in older women who have a history of GH.[7] Obvious limitations to our study include the small size as well as the fact that we studied only one-third of our initial antepartum cohort. However, this low follow-up rate is not surprising when considering the family commitments of young mothers and the distances from our research center that many of our subjects lived. We were encouraged to find that our postpartum subjects were reasonably representative of the antepartum cohort from our initial study (Tables S1 and S2).

An analytic limitation was not controlling for BMI differences among our participants. By focusing our antepartum vs

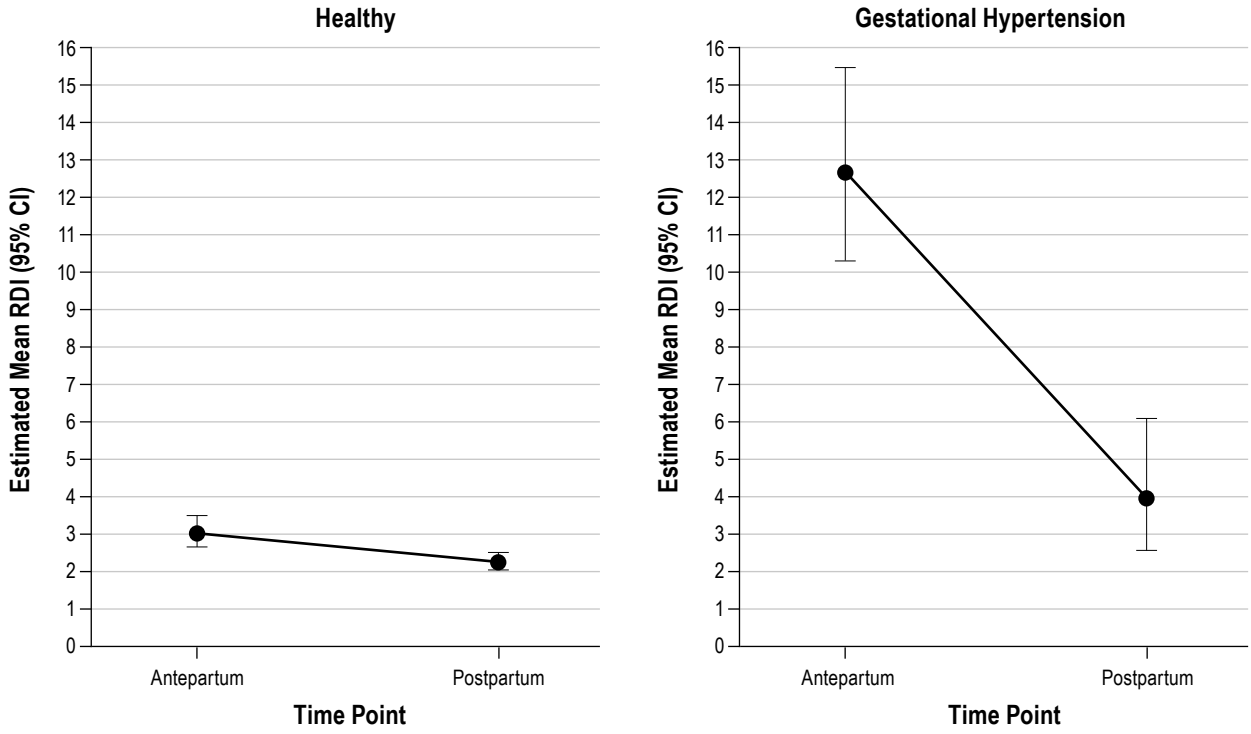

Figure 1—Modelled mean RDI of study participants. Data points represent the adjusted mean RDI of study participants. CI, confidence interval. Error bars = SEM.

postpartum comparisons of healthy women and women with GH separately we compared women with similar BMI measurements, which increased our confidence in our conclusions. Future studies would do well to address issues with BMI differences between GH and healthy women during the design of the study, perhaps matching participants based on BMI.

Although there have been a few small reports of SDB improving postpartum in women with known SDB, this is the first study we are aware of which compares women with GH and healthy women in terms of sleep and sleep related breathing changes at the antepartum and postpartum time points. Clearly larger and longer term studies are needed in this area.

CONCLUSION

Despite persistent obesity, we found that SDB in women with gestational hypertension improved in the postpartum state to levels indistinguishable from our healthy subjects. This suggests that the physiologic effects of pregnancy may have had a pathologic role in the development of antepartum SDB in women with GH. Women with GH may need to be followed for the reappearance of SDB with aging, as further physiologic changes and/or weight gain may again unmask a propensity to SDB. Ultimately, larger and longer-term follow-up studies are needed to clarify this issue.

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DISCLOSURE STATEMENT

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SUPPLEMENTAL MATERIAL

Table S1—Antepartum comparison of healthy women who did and did not participate in the follow-up study

Variable	No follow-up (n = 17) Mean ± SD (Median)	Follow-up (n = 7) Mean ± SD (Median)	P-value
Age (year)	29.8 ± 3.4 (29.0)	29.9 ± 6.9 (32.0)	0.99[a]
BMI (kg/m^2)	28.5 ± 5.0 (27.5)	28.8 ± 4.2 (27.2)	0.90[a]
Gestational Age (weeks)	35.1 ± 1.9 (35.9)	33.4 ± 4.2 (34.0)	0.31[a]
ESS	7.1 ± 2.8 (7.0)	11.0 ± 4.0 (10.0)	0.01[a]
Snoring	4 (24%)	5 (71%)	0.03[c]
TST (min)	308.1 ± 53.0 (315.0)	328.8 ± 49.3 (309.0)	0.38[a]
AHI (events/h)	0.3 ± 0.6 (0.0)	0.9 ± 2.4 (0.0)	0.71[b]
RDI (events/h)	2.4 ± 2.0 (2.0)	2.8 ± 5.3 (1.2)	0.27[b]

24 complete PSGs for assessment. [a]*t*-test comparison of means. [b]Mann-Whitney U comparison of distribution. [c]Chi-square test comparison of counts. BMI, body mass index; ESS, Epworth Sleepiness Scale; AHI, apnea hypopnea index; RDI, respiratory disturbance index; TST, total sleep time.

Table S2—Antepartum comparison of GH women who did and did not participate in the follow-up study

Variable	No follow-up (n = 22) Mean ± SD (Median)	Follow-up (n = 11) Mean ± SD (Median)	P-value
Age (year)	28.9 ± 6.0 (30.0)	27.9 ± 4.7 (27.0)	0.63[a]
Gestational Age (weeks)	34.6 ± 3.3 (35.5)	34.7 ± 3.2 (35.7)	0.96[a]
BMI (kg/m^2)	36.5 ± 7.5 (35.8)	37.6 ± 5.9 (36.2)	0.67[a]
ESS	8.6 ± 3.3 (9.0)	6.3 ± 3.2 (5.0)	0.06[a]
Snoring (yes/no)	18 (82%)	9 (82%)	1.00[c]
TST (min)	271.4 ± 67.0 (291.5)	235.2 ± 66.7 (242.0)	0.15[a]
AHI (events/h)	2.2 ± 3.8 (0.4)	4.6 ± 9.3 (0.3)	0.95[b]
RDI (events/h)	10.7 ± 10.9 (5.2)	12.0 ± 12.3 (6.4)	0.92[b]

33 Complete PSGs for assessment. [a]*t*-test comparison of means. [b]Mann-Whitney U comparison of distribution. [c]Chi-square test comparison of counts. BMI, body mass index; ESS, Epworth Sleepiness Scale; AHI, apnea hypopnea index; RDI, respiratory disturbance index; TST, total sleep time.

Table S3—Postpartum comparison of healthy vs. GH women

Variable	Healthy (n = 7) Mean ± SD (Median)	GH (n = 11) Mean ± SD (Median)	P-value
Age (year)	31.9 ± 6.9 (33.0)	29.7 ± 4.5 (28.0)	0.44[a]
Follow-up time (months)	17.6 ± 2.8 (18.0)	17.1 ± 4.4 (16.0)	0.80[a]
BMI (kg/m^2)	22.8 ± 4.0 (21.7)	32.5 ± 6.8 (32.3)	< 0.01[a]
ΔBMI (kg/m^2)	-6.0 ± 1.4 (-5.5)	-5.1 ± 3.4 (-5.5)	0.04[a]
ESS	11.4 ± 4.1 (11.0)	6.3 ± 3.1 (5.0)	0.01[a]
Arousal index (events/hour)	5.6 ± 4.7 (4.8)	6.0 ± 4.0 (6.1)	0.59[b]
Sleep Quality VAS (%)	51.8 ± 25.0 (66.2)	49.0 ± 23.3 (41.9)	0.81[b]
Postpartum snoring (yes/no)	4 (57%)	5 (45%)	0.63[c]
TST (minutes)	387.2 ± 42.0 (391.8)	363.3 ± 114.1 (408.0)	0.02[a]
Sleep Efficiency (%)	88.2 ± 3.4 (88.4)	83.1 ± 16.6 (88.3)	0.86[b]
% Supine (%)	45.5 ± 31.6 (31.0)	36.5 ± 30.0 (35.9)	0.53[b]
% REM (%)	13.8 ± 3.8 (13.8)	13.9 ± 6.4 (13.1)	0.96[a]
AHI (events/h)	1.1 ± 2.0 (0.3)	2.6 ± 2.7 (1.3)	0.26[b]
RDI (events/h)	2.1 ± 3.2 (1.2)	2.9 ± 2.9 (1.9)	0.75[b]
Mean SpO_2 (%)	97.0 ± 0.8 (97.3)	96.6 ± 1.5 (96.7)	0.47[a]
Min SpO_2 (%)	93.0 ± 3.7 (95.0)	91.1 ± 5.2 (93.0)	0.43[b]

[a]*t*-test comparison of means. [b]Mann-Whitney U comparison of distribution. [c]Chi-square test comparison of counts. BMI, body mass index; ΔBMI, change in body mass index; ESS, Epworth Sleepiness Scale; AHI, apnea hypopnea index; RDI, respiratory disturbance index; TST, total sleep time.

Table S4—Antepartum comparison of healthy vs. GH who participated in postpartum follow-up

Variable	Healthy (n = 7) Mean ± SD (Median)	GH (n = 11) Mean ± SD (Median)	P-value
Age (year)	29.9 ± 6.9 (32.0)	27.9 ± 4.7 (27.0)	0.48[a]
Gestational Age (weeks)	33.4 ± 4.2 (34.0)	34.7 ± 3.2 (35.7)	0.45[a]
BMI (kg/m^2)	28.8 ± 4.2 (27.2)	37.6 ± 5.9 (36.2)	< 0.01[a]
ESS	11.0 ± 4.0 (10.0)	6.3 ± 3.2 (5.0)	0.01[a]
Antepartum Snoring (yes/no)	5 (71%)	9 (82%)	0.61[c]
Sleep quality VAS (%)	52.1 ± 18.7 (53.2)	36.2 ± 14.9 (30.5)	0.09[b]
TST (minutes)	328.8 ± 49.3 (309.0)	235.2 ± 66.7 (242.0)	0.01[a]
Sleep efficiency (%)	80.7 ± 9.2 (81.1)	60.4 ± 16.6 (61.6)	0.01[b]
% Supine (%)	22.3 ± 22.6 (10.7)	22.2 ± 26.0 (9.9)	0.47[b]
% REM (%)	15.2 ± 2.4 (15.5)	7.4 ± 5.1 (7.3)	< 0.01[a]
AHI (events/h)	0.9 ± 2.4 (0.0)	4.6 ± 9.3 (0.3)	0.21[b]
RDI (events/h)	2.8 ± 5.3 (1.2)	12.0 ± 12.3 (6.4)	0.03[b]
Arousal index (events/h)	10.3 ± 6.9 (7.4)	19.5 ± 14.8 (15.6)	0.06[b]
Mean SpO_2 (%)	96.7 ± 1.3 (96.8)	95.7 ± 1.3 (96.0)	0.12[a]
Min SpO_2 (%)	93.0 ± 2.8 (94.0)	90.7 ± 4.0 (93.0)	0.15[b]
Gestational Age (weeks)	33.4 ± 4.2 (34.0)	34.7 ± 3.2 (35.7)	0.45[a]

[a]*t*-test comparison of means. [b]Mann-Whitney U comparison of distribution. [c]Chi-square test comparison of counts. BMI, body mass index; ESS, Epworth Sleepiness Scale; AHI, apnea hypopnea index; RDI, respiratory disturbance index; TST, total sleep time.