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Sleep and Pregnancy-Induced Hypertension: A Possible Target for Intervention?

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Sleep disturbances in the general population are associated with elevated blood pressure. This may be due to several mechanisms, including sympathetic activation and hypothalamic-pituitary-adrenal (HPA) axis disturbance. Elevated blood pressure in pregnancy can have devastating effects on both maternal and fetal health and is associated with increased risk for preeclampsia and poor delivery outcomes. Preliminary evidence suggests that mechanisms linking sleep and blood pressure in the general population may also hold in the pregnant population. However, the effects of disturbed sleep on physiologic mechanisms that may directly influence blood pressure in pregnancy have not been well studied. The role that sleep disturbance plays in gestational blood pressure elevation and its subsequent consequences warrant further investigation. This review evaluates the current literature on sleep disturbance and elevated blood pressure in pregnancy and proposes possible treatment interventions.

Keywords: Sleep disturbance, blood pressure, pregnancy, hypertension, weight

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The association between sleep disturbances and elevated blood pressure has been extensively studied in the general population. However, relatively few studies have investigated this relationship in the pregnant population. Pregnancy predisposes women to a variety of sleep disturbances.[1,2] Similar to non-pregnant individuals, sleep disturbance in pregnancy may be a risk factor for elevated blood pressure, which can lead to maternal and fetal morbidity.[3,4] Gestational hypertension, defined as a blood pressure higher than 140/90 diagnosed after 20 weeks of gestation, is associated with fetal growth restriction and abruptio placentae and can predispose to preeclampsia, as well as cardiovascular disease later in life.[1,4-6] There are few studies evaluating the link between sleep and blood pressure during pregnancy. In this paper, we first review the relationship between sleep and blood pressure in non-pregnant adults. We then outline factors that predispose pregnant women to poor sleep. We conclude with a review of the emerging literature on the associations between sleep and blood pressure in pregnancy.

SLEEP AND BLOOD PRESSURE IN THE NON-PREGNANT POPULATION

In the U.S. the average sleep duration has decreased by 1.5-2 h/night, with > 30% of Americans sleeping < 6 h/night.[7] This phenomenon and the concurrent increase in hypertension intimated a possible link between sleep duration and blood pressure. Recently, a series of epidemiological papers have noted an association between sleep duration (both short and long) and elevated blood pressure[8-11]; for example, the Sleep Heart Health Study reported that participants who slept < 5 or ≥ 9 h/night had a greater frequency of hypertension than individuals sleeping 7 to 8 h/night.[12] Buxton et al. analyzed the 2004-2005 US National Health Interview Survey data (n = 56,507 observations, adults 18-85 years) and found those with short (< 7 h) and long (> 8 h) sleep were more likely to have elevated blood pressure than those sleeping 7 to 8 h/night.[13] These studies underscore the potential consequences of obtaining too little or too much sleep.

Similar to sleep duration, sleep quality is commonly evaluated. It can be ascertained directly with subjective methods or inferred from objective measures. Fiorentini et al., for instance, evaluated sleep quality in a cohort of hypertensive and type 2 diabetic participants. They found that poor sleep quality, defined by a Pittsburgh Sleep Quality Index score > 5, was more frequent among those with hypertension.[14] Knutson et al. examined the association between sleep quality, measured by actigraphy, and blood pressure in mid-life adults. They found that lower sleep quality, as indicated by sleep duration and sleep maintenance, was associated with higher systolic and diastolic blood pressure levels both cross-sectionally and longitudinally over 5 years.[11]

Hypertension is commonly thought to occur in mid-life or aging individuals. However, pre-hypertension and hypertension are rapidly rising in adolescents. It is possible that several health behaviors that originate in adolescence, including poor diet, smoking, and poor sleep, may increase the risk for prehypertension and an earlier development of hypertension.[15,16] This phenomenon could partly explain why adverse pregnancy outcomes, such as preeclampsia and gestational diabetes, are increasing despite advances in medical technology. Support for this hypothesis comes from Javaheri et al. who studied the sleep of 238 adolescents using actigraphy. They found that poor quality sleep, defined as sleep efficiency ≤ 85% or short

sleep duration (≤ 6.5 h), was associated with elevated blood pressure. Specifically, they found that the odds of prehypertension increased 4.5-fold in adolescents who had low sleep efficiency and 2.8-fold for those with short sleep.[17] Taken together, these studies support the hypothesis that poor sleep quality, beginning much earlier in life than previously recognized, is associated with increased risk of developing hypertension and associated morbidities. They also suggest that early intervention may prove beneficial in reducing adverse health outcomes.[15-17]

In addition to associations with quantitative aspects of blood pressure, sleep disturbance has been associated with impaired nocturnal blood pressure dipping.[18-20] During normal sleep, blood pressure dips by 10% to 20%, in part due to a decrease in sympathetic output.[21] A nocturnal blood pressure dip < 10% defines non-dipping. Several studies have shown that reduced blood pressure dipping during sleep is an indicator of cardiovascular disease.[22-24] Ohkubo and colleagues, for instance, studied 24-h ambulatory blood pressure in 1,542 Japanese adults > 40 years of age, and followed them for an average 9.2 years. They found that for each 5% deficit in normal nocturnal dipping values, there was an associated 20% greater risk of developing cardiovascular disease.[25] This study highlights emerging evidence which indicates that nocturnal blood pressure may be a better predictor for cardiovascular risk than daytime blood pressure readings.[26-28] Reduced nocturnal blood pressure dipping can have significant immediate and future cardiovascular implications, including cognitive impairment and cerebrovascular disease.[18-20,29,30] Furthermore, since sleep disturbances, such as poor sleep quality, have been associated with blunted nocturnal blood pressure dipping, the clinical importance of assessing sleep as a potential risk factor for cardiovascular disease is substantially strengthened.[19,20]

Sleep disordered breathing (SDB), also referred to as obstructive sleep apnea (OSA), has a prevalence of up to 15% in the general population, and is even greater in obese (40%) and morbidly obese (70% to 90%) patients.[31] It is strongly associated with elevated blood pressure. In OSA, repeated episodes of partial or complete upper airway collapse lead to apneas (cessation of airflow for ≥ 10 sec, usually followed by an electroencephalographically measured arousal) or hypopneas (discernible reduction in airflow for 10 sec associated with an oxyhemoglobin desaturation of 4%). The apnea-hypopnea index (AHI), defined by the number of apneas or hypopneas per hour of sleep, describes disease severity. Mild OSA is defined as AHI of 5 to 15, moderate disease as AHI of 15 to 30, and severe disease as AHI > 30.[31] Episodes of apnea or hypopnea can cause hypoxia and result in frequent arousals, and thus sleep fragmentation. Repeated episodes of hypoxia and reoxygenation have also been shown to be associated with endocrine and metabolic disturbance, as well as elevated risk for metabolic syndrome and cardiovascular disease in OSA patients.[5,6,32,33] SDB has also been shown to be an independent risk factor for hypertension.[34-36] Indeed, treatment of SDB using positive airway pressure is associated with a reduction in incident hypertension and a significant improvement in hypertensive patients.[37,38] However, these relationships have not been observed universally.[39]

PSYCHOSOCIAL CORRELATES OF SLEEP AND ELEVATED BLOOD PRESSURE

In addition to sleep disturbance, several psychosocial factors are recognized correlates of increased blood pressure. These factors may also exacerbate the occurrence and the negative consequences of sleep disturbance in pregnancy, similar to what has been observed in non-pregnant individuals.[40,41] Psychosocial stress, including occupational stress, social isolation, marital stress, and low socioeconomic status, have been associated with elevated blood pressure in the non-pregnant population.[42-50] The most commonly evaluated, however, is acute psychological stress. It has been postulated that stress and sleep disturbance interact to compound cardiovascular vulnerability.[51] A detailed examination of the role of psychosocial factors on sleep and blood pressure is beyond the scope of this review (see reviews[20,52,53]). Here, we merely highlight the importance of appreciating the complex relationships among these factors that may be particularly relevant during pregnancy.

The mechanisms that link sleep disturbances and elevated blood pressure are complex and involve several pathways. In OSA for example, nocturnal hypoxemia induces oxidative stress, inflammatory responses, and reduction in nitric oxide, which mediates vascular functions including dilatation and anticoagulation and has antioxidant properties.[54] Sleep disturbances have also been shown to increase sympathetic tone and hypothalamic-pituitary-adrenal axis function in experimental studies. Spiegel at al. found that experimentally induced short sleep (4 h) is associated with alterations in sympathovagal balance and 24-h salivary cortisol levels when compared to normal sleep (7-8 h).[55] Similar findings have been found in other experimental studies.[56,57] Sleep quality measures, including sleep latency and non-restorative sleep, have also been linked to metabolic and autonomic changes which have been associated with cardiovascular disease and hypertension.[17,58]

The relationship among cardiovascular changes, neuroendocrine changes, and sleep disturbance is not as clear. Tochikubo et al. found that in overtime workers, blood pressure, urinary norepinephrine levels, and sympathovagal disturbance (measured by heart rate variability) were higher on days after sleep restriction. However, sympathovagal disturbance was measured the evening after sleep restriction. Hence, this finding could have been due to increased stress due to sleepiness after a work day.[59] Additionally, Kato et al. described elevated blood pressure after sleep restriction, although they did not find significant changes in heart rate, forearm vascular resistance, or plasma catecholamines with sleep deprivation.[60] These studies show that although blood pressure appears to be directly influenced by sleep restriction, the exact mechanisms remain unclear.

SLEEP DISTURBANCE IN PREGNANCY

Sleep disturbances are distinctly more common in pregnant than in non-pregnant women assessed from the general population. Okun and Coussons-Read examined sleep data collected at 12, 24, and 36 weeks' gestation from 35 pregnant and once from 43 comparable non-pregnant women. As early as 12 weeks, pregnant women reported an increased number of

Table 1—Physical and hormonal changes in pregnancy, subsequent symptoms, and effect on sleep

Physical Changes	Symptom	Effect on Sleep
Increasing uterine Size	Difficulty laying supine	Sleep disruption; poor sleep quality
Hormone increases or Iron deficiency	Restless legs syndrome (RLS)	Difficulty falling asleep
Decreased tone of lower esophageal sphincter	Predisposes to gastric reflux symptoms	Sleep disruption due to discomfort
Increased rate of micturition	Increased renal blood flow; Dilation of ureters and renal pelvis; Uterine pressure on bladder	Frequent nocturnal awakenings
Hormonal Changes	**Symptom**	**Effect on Sleep**
Increased estrogen	Decrease nasopharyngeal airway patency: Potential predisposition to OSA Increase RLS symptoms	Sleep disruption via breathing occlusions. Changes in sleep architecture; decreases REM sleep
Increased progesterone	Increase minute ventilation; Increase nasopharyngeal muscle tone	Altered sleep architecture Increases NREM sleep

References: 53, 65, 68, 72, 79

naps, nocturnal awakenings, time spent awake during the night, and poorer sleep quality than non-pregnant women.[61] Sleep in the pregnant women progressively worsened, with over 50% of the women meeting sleep criteria for insomnia by the end of pregnancy. Suzuki et al. found that that among 192 pregnant women surveyed retrospectively, 88% had alterations in sleep compared with their usual experience.[62] The reported changes included insomnia, parasomnias (nightmares and night terrors), restless leg syndrome (RLS), snoring, and sleep apnea. Among the most frequent self-reported causes of sleep disturbance during pregnancy were urinary frequency, back or hip ache, and heartburn. Facco et al. investigated sleep during pregnancy in a prospective cohort of 189 women assessed at 2 points during pregnancy, with a mean baseline assessment of 13.8 (± 3.8) weeks and a mean second assessment of 30.0 (± 2.2) weeks. At the second assessment, sleep duration significantly decreased compared to baseline (7.4 ± 1.2 h vs. 7.0 ± 1.3), the number of participants who reported snoring increased (11% vs. 16.4%), incidence of restless leg syndrome increased (17.5% vs. 31.2%), and there was an increase of poor sleep quality as measured by Pittsburgh Sleep Quality Index > 5 (39.0% vs. 53.5%).[36]

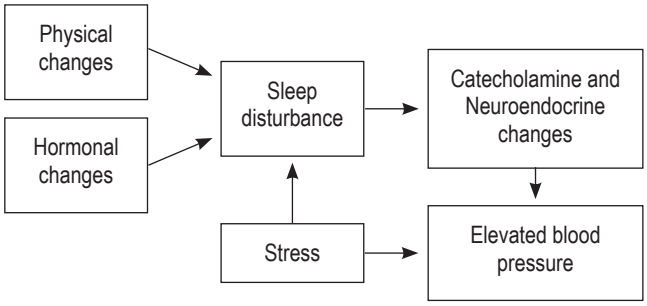
In pregnancy, hormonal changes occur to ensure the survival of the fetus. However, these hormonal changes may result in substantial sleep disturbances.[1] By the last few weeks of pregnancy, daily estrogen production is one thousand times premenopausal ovulatory levels, and progesterone levels increase from 25 ng/mL at 6 weeks to 150 ng/mL at 37 weeks.[64] Estrogen reduces rapid eye movement sleep (REM) and progesterone reduces NREM sleep.[1,65,66] Estrogen can also cause physical changes that can affect sleep, including hyperemia, mucosal edema, hypersecretion, and increased friability in the upper airways. These changes result in reduction of nasopharyngeal airway patency, which can cause a sensation of nasal stuffiness and may exacerbate sleep disordered breathing in women with elevated body mass index.[67] Progesterone is thought to act via peripheral chemoreceptors and centrally in the medulla to increase respiratory drive.[68] This, in conjunction with greater metabolic carbon dioxide production and increased minute ventilation, can cause respiratory alkalosis, which can reduce respiratory drive and predispose to central sleep apnea.[1] Furthermore, oxygen consumption is increased by approximately 20% to 33% by the third trimester due to fetal demands and changes in maternal metabolism. Increased oxygen consumption, along with a reduced functional residual capacity due to an enlarging uterus, results in a lowered oxygen reserve and can affect oxygen homeostasis more than in the non-pregnant state.[1,69] **Table 1** illustrates the major physical and hormonal changes in pregnancy that can affect sleep.

The dramatic physical changes unique to pregnancy can further affect sleep. The enlarging uterus can upwardly displace the diaphragm, further compromising functional residual capacity, which decreases by 10% to 25% at term. This, together with reduction in chest wall and total respiratory compliance, may lead many pregnant women to experience shortness of breath while lying supine. The inability to assume a comfortable sleeping position, especially during the third trimester of pregnancy, may have a significant impact on a pregnant woman's ability to initiate and maintain sleep.[36] Discomfort from back and leg cramps may also disrupt sleep. Lower esophageal sphincter tone decreases throughout pregnancy, reaching its lowest point in late pregnancy. Resulting gastroesophageal reflux can cause discomfort and sleep disruptions.[70] Additionally, renal blood flow increases in pregnancy throughout first and second trimester, along with dilation of the ureters and renal pelvises. These changes and the pressure of an enlarged uterus on the bladder cause pregnant women to wake several times per night to urinate.[36]

SLEEP AND HYPERTENSION IN PREGNANCY

Elevated maternal blood pressure during pregnancy poses great risk for both mother and fetus. Approximately 10% of pregnancies are affected by hypertension. Consequences of pregnancy-related hypertension include increased risk of abruptio placentae, disseminated intravascular coagulation, cerebral hemorrhage, hepatic failure, and acute renal failure.[5] Furthermore, elevated blood pressure in pregnancy can be part of preeclampsia and eclampsia, which carry maternal mortality rates of 10% to 15%, and future risk for cardiovascular disease.[4,71]

Figure 1—Proposed model of how physical and hormonal changes in pregnancy coupled with stress result in disturbed sleep which can result in elevated blood pressure



Normative physical changes, such as changes in body habitus, and hormonal changes, including dramatic increases in estrogen and progesterone, are recognized contributors to sleep disturbance in pregnancy. Subsequent to the sleep disturbance are various catecholamine and neuroendocrine changes which can negatively impact blood pressure. Concurrent stress, whether daily hassles or serious life events, is both an independent and dependent modifier of blood pressure. These associations are critical throughout pregnancy as elevated blood pressure is linked with increased risk of preeclampsia and preterm birth.

Systolic and diastolic blood pressure normally fall in early pregnancy by 5-10 mm Hg, reaching a mean nadir of 105/60 mm Hg, and then gradually rise to pre-pregnancy values by term.[72] However, emerging evidence indicates that sleep disturbance may disrupt the normal course of gestational blood pressure changes. Williams et al. found that self-reported short (≤ 6 h) and long (≥ 10 h) sleep durations in early pregnancy (mean 14 weeks) were associated with elevated blood pressure, particularly mean third trimester blood pressures, in 1,272 women. Mean third trimester systolic blood pressure (SBP), diastolic blood pressure (DBP), and mean arterial blood pressure (MAP) for women who reported short early pregnancy sleep durations (≤ 6 h) compared to normal sleep duration (9 h) were 3.72, 3.04, and 3.18 mm Hg higher, respectively, after adjustment for maternal age, race/ethnicity, parity, educational status, and pre-pregnancy body mass index. The differences in third trimester SBP, DBP, and MAP for women who reported long sleep durations (≥ 10 h), compared with those reporting sleeping 9 h nightly, were 4.21, 3.43, and 3.65 mm Hg higher, respectively.[3] A similar conclusion was reached by Reid and colleagues who reported that women with gestational hypertension has less total sleep time, lower sleep efficiency, and a lower percentage of REM sleep than healthy pregnant women.[73] Although the mechanisms behind these differences are not clear, they may be similar to those that link short and long sleep durations with increased BP in the non-pregnant population.

Furthermore, as in the non-pregnant population, psychological stress may play a role in both sleep disturbance and blood pressure elevation.[74] Pregnancy can be a mentally taxing time for women, especially in those with concurrent stressful life events or psychosocial stress. Stress may further elevate blood pressure in pregnant women, similar to what has been observed in non-pregnant cohorts.[40,41,74] We propose that the hormonal and physical changes, as well as stress of pregnancy, induce sleep disturbance and may result in blood pressure perturbations. **Figure 1** illustrates this model.

SLEEP RELATED BREATHING DISORDERS IN PREGNANCY

SDB is characterized by abnormal respiratory patterns (e.g., apneas, hypopneas) or abnormal gas exchange (e.g., hypoxia). Sleep related breathing disorders like snoring and obstructive sleep apnea occur in pregnancy; however, there is little data detailing their incidence or prevalence. Most investigators agree that sleep related breathing disorders are more prevalent in pregnant women than non-pregnant women. As previously noted, estimates in non-pregnant women range from 2% to 5%, whereas estimates in pregnancy range from ~10% in early pregnancy to upwards of 30% in late pregnancy.[67,75-78] There is currently a paucity of objective data on the incidence of sleep related breathing disorders in pregnancy. Our current knowledge relies primarily on self-reported symptoms, including excessive daytime sleepiness, snoring, or breathing cessations, which suggest but do not confirm the presence of OSA. The development of SDB in pregnancy is considered a consequence of necessary physiologic adaptations that occur in pregnancy,[79] such as dramatic hormonal and subsequent physical changes. Estrogen, for instance, can cause upper airway narrowing and could predispose pregnant women to snore and develop SDB. Progesterone, on the other hand, increases minute ventilation, and the resulting respiratory alkalosis enhances sensitivity of the respiratory center to carbon dioxide in pregnancy, which may predispose to central sleep apnea.[1]

SDB, independent of maternal BMI, is associated with an increased risk of hypertension in pregnancy, as well as maternal morbidity.[73,78,80-86] In one study, preeclampsia, a hypertensive syndrome in pregnancy, was significantly more common among snorers than non-snorers (10% versus 4%, $p < 0.05$), as was gestational hypertension (14% versus 6%, $p < 0.01$).[87] This was recently corroborated in a report by O'Brien and colleagues, who found that pregnancy-onset snoring was independently associated with gestational hypertension (OR 2.36 [1.48-3.77], $p < 0.001$) and preeclampsia (OR 1.59 [1.06-2.37], $p = 0.024$) in 1,719 third-trimester pregnant women.[78] Another study reported that snoring and "excessive daytime sleepiness," which could indicate poor sleep, were reported more commonly in later pregnancy in women with preeclampsia than those without preeclampsia or non-pregnant controls.[88] In a large cross-sectional study of immediately postpartum women, Perez-Chada et al. reported an increase in gestational hypertension and preeclampsia among those with symptoms of SDB even after adjusting for potential confounders such as BMI.[89] In a similar study, Bourjeily et al. adjusted for comorbid conditions and reported an increase in preeclampsia and gestational hypertension in women with SDB.[90] **Table 2** summarizes a series of studies that have examined the frequency and consequences of SDB in pregnancy.

SIGNIFICANCE

Hypertensive disorders in pregnancy are prevalent and pose risk to both mother and child. Additionally, they can carry risk

Table 2—Selected listing of sleep disordered breathing (SDB) in pregnancy studies

First Author	Study Design	Outcome Measures	Major Findings
Ayrim A[83]	Cross sectional study of 200 pregnant and 200 age-matched controls. Epworth Sleepiness Scale (ESS) were assessed to determine excessive daytime sleepiness.	Gestational hypertension and snoring	Excessive daytime sleepiness or snoring was associated with GH or other fetal outcomes.
Bourjeily G[97]	Retrospective study with 1,000 postpartum women. SDB assessed by the Multivariable Apnoea Prediction Index Questionnaire.	Pregnancy and fetal outcomes in women with SDB	SDB symptoms were found to have higher likelihood of pregnancy-induced hypertension and preeclampsia (adjusted OR 2.3, 95% CI 1.4-4.0)
Chen[98]	Retrospective study of 791 pregnant women with OSA assessed by PSG, compared to 3,955 controls.	Birth outcome by OSA status	Women with OSA were more likely than controls to have preeclampsia (OR 1.60 [95% CI, 2.16-11.26]), low birth weight (OR 1.76 (95% CI, 1.28-2.40), preterm birth 2.31 (95% CI, 1.77-3.01), small for gestational age infants 1.34 (95% CI, 1.09-1.66), CS1.74 (95% CI, 1.48-2.04)
Connolly G[99]	Case control prospective study of 15 women with preeclampsia and 15 without preeclampsia in each trimester. Both groups compared to15 non-pregnant women	Inspiratory flow measured by nasal canula, pulse oximetry and abdominal belt based on pregnancy and preeclampsia status	Women with preeclampsia had more time with inspiratory flow limitation in the third trimester subjects than the other 2 groups (31% ± 8.4% of sleep period time vs. 15.5% ± 2.3% vs. < 5%) (p = 0.001)
Facco F[77]	Retrospective cohort study of 143 postpartum women. PSG assessed mild SDB in 34 and moderate to severe SDB in 26.	The association between SDB and adverse pregnancy outcome (pregnancy-related hypertension, gestational diabetes, or preterm birth ≤ 34 weeks)	Increasing severity of SDB was associated with increasing risk of adverse pregnancy outcome: AHI < 5, 18.1%; AHI 5 to 14.9, 23.5%; AHI ≥ 15, 38.5% (p = 0.038)
Champagne K[100]	Case-control study comparing 17 pregnant women with GH and 33 without GH on frequency of PSG-assessed OSA.	OSA defined by apnea/ hypopnea index (AHI) ≥ 15 events per hour, without requirement for desaturation.	Women with GH had greater AHI (38.6 ± 36.7) compared to normotensive women (18.2 ± 12.2).
Izci B[67]	Prospective study of 100 third trimester women and 100 non-pregnant women. Upper airway dimensions and SDB symptoms were measured using acoustic reflection	Upper airway dimensions and SDB symptoms (snoring) in pregnant women versus non-pregnant women	Snoring was more common in pregnant women (41%) versus non-pregnant women (17%) and then returned back to non pregnant levels (18%) post partum. Upper airway dimensions were also smaller in pregnant women as compared to non-pregnant and post-partum women.
Louis JM[84]	Retrospective study of 57 women with OSA and 114 healthy controls.	Maternal morbidity and preterm birth	OSA patient had more preeclampsia (19.3% vs 7.0%, p = 0.02) and preterm birth (29.8% vs 12.3%, p = 0.007). OSA was associated with increased risk for maternal morbidity as well (OR 4.6 (1.5-13.7).
Maasilta P[75]	Case control study of PSG-assessed SDB in obese pregnant women. Participants were 11 obese women (BMI, 34 kg/m^2) and 11 control women (BMI, 23 kg/m^2)	Occurrence of SDB in obese women during pregnancy	More SDB symptoms occurred in obese women compared to non-obese women. Apnea-hypopnea indexes (1.7 events/h vs 0.2 events/h; p < 0.05), 4% oxygen desaturations (5.3 events/h vs 0.3 events/h; p < 0.005), and snoring times (32% vs 1%, p < 0.001) were significantly different between the 2 groups.
O'Brien L[78]	Prospective study of 1,719 pregnant women in late pregnancy. Screening for presence and duration of habitual snoring	Clinical diagnosis of gestational hypertension, preeclampsia, and gestational diabetes	New-onset snoring during pregnancy is quite frequent (25%) and is associated with an increased risk of gestational hypertension (OR 2.36 [1.48-3.77, p < 0.001]) and preeclampsia (OR 1.59 [1.06-2.37]), p = 0.024). There was no effect on gestational diabetes.
Reid J[73]	Cross-sectional comparison of self-reported sleep and PSG-assessed SDB in pregnant women with (34) and without (26) GH.	Presence of SDB in late pregnancy. Secondarily reported on PSG-assessed sleep	Women with GH have a higher frequency of SDB (53%) than healthy pregnant women (12%). This finding is confounded by obesity, which was significantly more frequent among women with GH. Women with GH also had less total sleep time (252 ± 81 min vs 311 ± 54 min, p = 0.003 and lower sleep efficiency (62% ± 19.5% vs 71.9% ± 10.3%, p = 0.003) compared to healthy women.

for maternal morbidity later in life. Preeclampsia, especially if complicated by HELLP syndrome (hemolysis, elevated liver enzymes, and low platelet count), predisposes to future cardiovascular disease.[31] A meta-analysis by Bellamy et al. found that women who developed gestational hypertension or preeclampsia had an increased risk of developing hypertension later in life. The relative risk of ischemic heart disease, stroke, and venous thromboembolism were also increased later in life in women with prior diagnoses of preeclampsia. Furthermore, Bellamy et al. found that women who developed preeclampsia had greater all-cause mortality risk compared to women who had normal blood pressure during pregnancy. This risk was even greater for women who developed preeclampsia before 37 weeks.[71] Kestenbaum et al. also found that gestational hypertension, mild and severe preeclampsia were associated with 2.8-fold higher risk of cardiovascular events, and that severe preeclampsia was associated with 2.3-fold higher risk of thromboembolic events.[4] Furthermore, hypertensive disorders in pregnancy are associated with poor fetal outcomes including preterm birth, small for gestational age infants, and abruptio placentae.[5] The delayed morbidity risk of hypertensive disorders, as well as the immediate risk to mother and fetus risk, only amplifies the need for better understanding and prevention.

CONCLUSION

Elevated blood pressure in pregnancy can have devastating effects on both maternal and fetal health during the perinatal period and beyond. The causes of hypertensive syndromes in pregnancy like preeclampsia and gestational hypertension appear to be multifactorial. However, numerous studies demonstrate a strong link between sleep duration, quality or sleep related breathing disorders and blood pressure in non-pregnant adults; emerging studies suggest a similar relationship in the pregnant population. This link represents a possible source of preventative measures for gestational hypertension and preeclampsia. However, more complete understanding of the association between sleep and blood pressure in pregnancy is needed. Well-controlled, longitudinal studies with large cohorts and both objective and subjective sleep measurements are needed to better assess sleep in pregnancy and how it relates to blood pressure. These studies should include blood pressure measurements throughout pregnancy, as well as pregnancy outcomes, to assess the effect of sleep on both maternal and fetal health. Currently, screening for sleep disruption in pregnant women is not common practice. More knowledge and widespread understanding of the effects that sleep has on pregnancy may improve upon the obstetrician's ability to screen for those with sleep disruption and who may be at risk for hypertensive disorders. Utilization of short questionnaires, such as the Insomnia Symptom Questionnaire (ISQ),[91] could be incorporated into prenatal care to assist in the identification of those women at-risk for sleep problems. Emerging data suggests that a modest number of pregnant women have difficulty initiating sleep (DIS).[92] Given the associations between DIS and adverse health outcomes,[93-95] this may be an appropriate target for intervention. Early identification of at-risk women may allow for simple interventions, including counseling on the impacts of sleep on maternal and fetal health and prescribing behavioral sleep regimens to not only improve sleep but potentially blood pressure as well. While there is currently a paucity of studies that have examined the impact of interventions on sleep in pregnant women, there is some evidence from a study of postpartum mothers that a behavioral-education intervention could be applied in pregnancy.[96] In this randomized controlled trial, women received intervention, which consisted of an in-person meeting with a nurse for sleep strategies, a booklet, and phone contacts, or usual care. Although there was no difference in the primary outcome of maternal nocturnal sleep, it is possible that the length of data collection or the measures used in the study were unable to capture the benefits of the intervention. It is probable, for instance, that improving sleep in the early postpartum is not feasible. Assessing the women further post-delivery may indicate otherwise.

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