Consensus Statement

Society of Anesthesia and Sleep Medicine and the Society for Obstetric Anesthesia and Perinatology Consensus Guideline on the Screening, Diagnosis, and Treatment of Obstructive Sleep Apnea in Pregnancy

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The Society of Anesthesia and Sleep Medicine and the Society for Obstetric Anesthesia and Perinatology tasked an expert group to review existing evidence and to generate recommendations on the screening, diagnosis, and treatment of patients with obstructive sleep apnea during pregnancy. These recommendations are based on a systematic review of the available scientific evidence and expert opinion when scientific evidence is lacking. This guideline may not be appropriate for all clinical situations and patients, and physicians must decide whether these recommendations are appropriate for their patients on an individual basis. We recognize that not all pregnant people may identify as women. However, data on non–cisgendered pregnant patients are lacking, and many published studies use gender-binary terms; therefore, depending on the study referenced, we may refer to preg-

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The final version of this consensus guideline was reviewed and endorsed by the Society of Anesthesia and Sleep Medicine, the Society for Obstetric Anesthesia and Perinatology, and the Society for Maternal-Fetal Medicine. The American College of Obstetricians and Gynecologists supports the value of this clinical document as an educational tool, May 2023.

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Each author has confirmed compliance with the journal's requirements for authorship.

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nant individuals as women. This guideline may inform the creation of clinical protocols by individual institutions that consider the unique considerations of their patient populations and the available resources.

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Recent studies have highlighted the increased risk of morbidity associated with obstructive sleep apnea (OSA), a type of sleep-disordered breathing, in pregnant people. However, little has been written specifically to guide clinical decision making in the screening, diagnosis, and treatment of OSA during pregnancy and the postpartum period. The increased morbidity associated with OSA during pregnancy, particularly for hypertensive disorders of pregnancy and gestational diabetes mellitus, has been demonstrated in several small prospective studies, meta-analyses, and large retrospective database studies and bring urgency to the purpose of this guideline.[1–10] Obstructive sleep apnea has also been associated with severe cardiovascular morbidity in national and population-based samples.[7,11]

Despite considerable interest in the potential implications of maternal OSA on fetal and neonatal outcomes, the results of the few retrospective and prospective studies and meta-analyses that have examined these associations have been inconsistent. Studies linking OSA with fetal heart rate abnormalities,[12–15] fetal growth restriction,[15,16] preterm birth,[3,4,11,17] Apgar scores,[3,4,11,17] and low birth weight[3,4,11] have been contradictory. This may be related to the definition of exposure or outcome, differences in adjusting for confounders, or the methodology used. Emerging data link OSA or high risk for OSA with adverse fetal outcomes such as congenital anomalies,[8] large for gestational age,[3] and shortened telomere length,[18] but these have not been reproduced in other studies. There appears to be an increased risk for admission to the neonatal intensive care unit (NICU)[3,8,9,11,17] among the neonates of people with OSA, but the drivers of the association are unknown. Despite the evidence associating OSA with adverse perinatal outcomes, there are no existing guidelines on the screening, diagnosis, or treatment of OSA in pregnancy.

AIMS

This guideline focuses on the screening, diagnosis, and treatment of OSA during pregnancy and postpartum. The specific aims were to 1) determine the appropriate assessments to identify patients with suspected or diagnosed OSA in pregnancy and the optimal timing of these assessments and 2) to evaluate the current evidence informing best practices for the diagnosis and treatment of peripartum OSA. In areas lacking sufficient published evidence, the Guideline Committee sought to establish expert consensus opinion, obtained through a series of virtual discussions with the Guideline Committee members until agreement was reached.

This practice guideline is not intended to define standards of care or to represent absolute requirements for patient care. Adherence to these guidelines cannot in any way guarantee successful outcomes; rather, these guidelines are meant to help individuals and institutions formulate plans to better care for peripartum patients with OSA. These recommendations reflect the current state of knowledge and its interpretation by a group of experts in the field at the time of publication. Although these guidelines will be updated periodically, new information that becomes available between updates should be considered.

GUIDELINE COMMITTEE

The Guideline Committee is composed of 11 members of the Society of Anesthesia and Sleep Medicine, an international society devoted to advancing standards of care for clinical problems shared by anesthesiology and sleep medicine; three Guideline Committee members are also members of Society for Obstetric Anesthesia and Perinatology, an international society devoted to advancing standards of care in obstetric anesthesiology and perinatology. The Guideline Committee included six anesthesiologists, four sleep medicine specialists and research scientists, one maternal–fetal medicine subspecialist, and a research librarian. They practice in academic settings in various parts of the United States, Canada, and the United Kingdom.

METHODS

Research Questions

Research questions were agreed on by all Guideline Committee members, and a systematic review of the literature was performed for articles related to the screening, diagnosis, treatment, and peripartum management of OSA in pregnancy (Box 1). Inclusion and exclusion criteria are detailed in Box 1. As a result of scant available evidence on the peripartum management of OSA in pregnant people, a decision was made to conduct a separate Delphi process to gain consensus of expert opinions on the peripartum management of OSA in pregnant people. These recommendations will be published separately.

Box 1. Inclusion Criteria and Study Questions

Population: pregnant people
Languages: Arabic, English, French, Spanish
Study designs: randomized controlled trials, observational studies, case series, meta-analyses
Excluded: Editorials, letters, case reports, conference abstracts, comments, systematic and narrative reviews, animal studies, and studies published in other languages

1. Screening for OSA in pregnant people
- 1.1. Should all pregnant people be screened for OSA or just specific pregnant people at higher risk of OSA?
- 1.2. What is the optimal timing for OSA screening during pregnancy?
- 1.3. What are the most effective screening tools to identify people with OSA in pregnancy?
  - 1.3.1. Berlin questionnaire?[64]
  - 1.3.2. STOP-BANG questionnaire?[20,21]
  - 1.3.3. The individual components of the STOP-BANG questionnaire?
  - 1.3.4. Epworth Sleepiness Scale?[68]
  - 1.3.5. ASA checklist?[60]
  - 1.3.6. Screening criteria proposed by Facco et al[34]?
  - 1.3.7. Screening criteria proposed by Louis et al[59]?
  - 1.3.8. BATE screening criteria?[69]

2. Diagnosis of OSA in pregnant people
- 2.1. Is home sleep apnea testing an effective diagnostic tool for OSA in pregnant people?
- 2.2. Is overnight oximetry an effective diagnostic tool for OSA in pregnant people?
- 2.3. Is repeat diagnostic testing warranted in the postpartum period for people who are diagnosed with OSA in pregnancy?
- 2.4. If repeat diagnostic testing is performed in the postpartum period for people who are diagnosed with OSA in pregnancy, what is the recommended timing?

3. Treatment of OSA in pregnant people
- 3.1. Does treatment of OSA with CPAP affect markers of cardiovascular and metabolic risk in pregnant people?
- 3.2. Does treatment of OSA in pregnant people influence their fetal and neonatal outcomes?
- 3.3. Does treatment of OSA alter the symptoms, apnea–hypopnea index, and nocturnal oxygen saturations of pregnant people?
- 3.4. Does treatment of OSA affect maternal obstetric outcomes?
- 3.5. Is treatment of preexisting OSA, pregnancy-onset (gestational) OSA, and postpartum OSA different from treatment in nonpregnant people?
- 3.6. Do treatment approaches for preexisting OSA, pregnancy-onset (gestational) OSA, and postpartum OSA differ?
- 3.7. What are the clinical indications for treatment of OSA in pregnant people, and when should treatment be initiated?
- 3.8. Are lifestyle changes useful in the treatment of OSA in pregnant people?

OSA, obstructive sleep apnea; STOP-BANG, snoring history, tired during the day, observed stop of breathing while sleeping, high blood pressure, body mass index higher than 35 (or 30), age older than 50 years, neck circumference greater than 40 cm, and male gender; ASA, American Society of Anesthesiologists; CPAP, continuous positive airway pressure.

Information Sources and Search Strategy

We searched MEDLINE (through PubMed), EMBASE (through Elsevier), CINAHL Complete (through EBSCO), and Scopus (through Elsevier) from database inception to August 26, 2020, using a combination of key words and database-specific subject headings for the following concepts: pregnancy and sleep apnea. No restrictions were placed by date or language. Editorials, letters, conference abstracts, and comments were excluded from the search. The full reproducible search strategies for all databases, including controlled vocabulary and specific key words are included in Appendix 1, available online at http://links.lww.com/AOG/D225.

Study Selection

All Guideline Committee members participated in reference review in a two-phase approach. Two reviewers, blinded to each other's assessments, independently screened references by title and abstract to determine inclusion in the Covidence systematic review software (Veritas Health Innovation). After title and abstract screening, the remaining articles were screened at the full-text level. Articles were included if the two assigned reviewers agreed on the decision for inclusion or exclusion. A third reviewer blinded to the previous reviewers' decisions resolved any disagreements at both phases of screening.

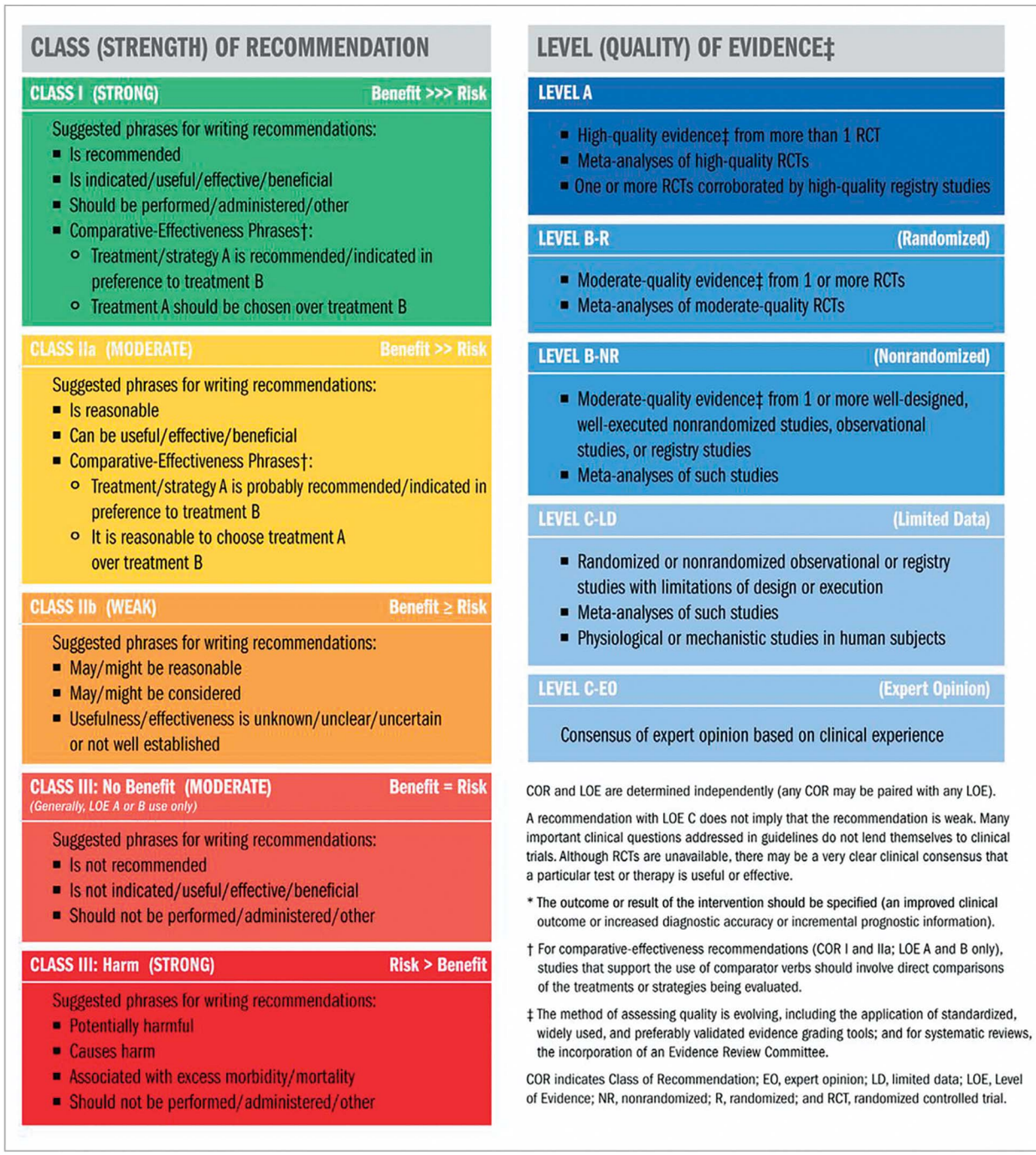

Fig. 1. ACC/AHA recommendation system: applying Class of Recommendation and Level of Evidence to clinical strategies, interventions, treatments, or diagnostic testing in patient care. Reproduced with permission from Halperin JL, Levine GN, Al-Khatib SM, Birtcher KK, Bozkurt B, Brindis RG, et al. Further evolution of the ACC/AHA clinical practice guideline recommendation classification system: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. Circulation. 2016;133:1426–28. doi: 10.1161/CIR.0000000000000312. ©2016 American Heart Association, Inc. All requests to use this information must come through the American Heart Association.

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Levels of Evidence and Grading of the Strength of Recommendations

Evidence to support each recommendation was graded using the American College of Cardiology/American Heart Association Clinical Practice Guideline recommendation classification system.[19] Each recommendation received an independently determined Level (quality) of Evidence that was used to guide the recommendation and a Class (strength) of Recommendation (Fig. 1).

Data Extraction

Data were extracted from the references by Guideline Committee members. Data points varied depending on the question. Details are provided in Appendix 2, available online at http://links.lww.com/AOG/D225.

Meta-analyses

When a meta-analysis was not available but three or more studies with the outcome were available, we conducted our own meta-analysis to inform the recommendations.[20,21] The data were analyzed with Meta-Disc 1.4.

RESULTS

The searches yielded a total of 3,669 citations after removal of 8,504 duplicates. All citations were imported into Covidence. Two phases of screening resulted in 192 included studies (Fig. 2).

Research Questions and Recommendations

The process of developing these guidelines involved creating recommendations that take into account not only the quality of evidence, but also the balance between benefit and harm to patients, patients' values and preferences, and the use of resources within the health system. Each recommendation was considered to ensure that it preserves patient safety and is practical within the clinical practice of perinatal medicine.

1. Screening for OSA in Pregnant People

Question 1.1. Should all pregnant people be screened for OSA or just specific pregnant people at higher risk for OSA?

The Guideline Committee carefully considered this research question considering the following factors: Is OSA prevalent in pregnant people; is there an effective treatment for OSA; and, if OSA is screened for and detected in pregnant people, will the treatment be effective at treating OSA and its associated symptomatology or at preventing adverse short-term and long-term pregnancy outcomes? These recommendations pertain to standard OSA screening during prenatal care and do not address the evaluation of people who present with symptoms that are consistent with or suggestive of sleep-disordered breathing. The latter group would need to be evaluated, and a decision to refer for diagnostic testing or a medical specialist would be based on phenotype, severity of symptoms, and physical examination findings. In addition, because OSA has a high chance of persisting postpartum, patients with symptoms in the third trimester should be evaluated by a specialist in the postpartum period.

Although there is significant evidence from retrospective database studies, meta-analyses, and prospective studies that OSA is associated with adverse pregnancy outcomes, namely gestational diabetes and hypertensive disorders of pregnancy, there is insufficient evidence to date that treating OSA during pregnancy

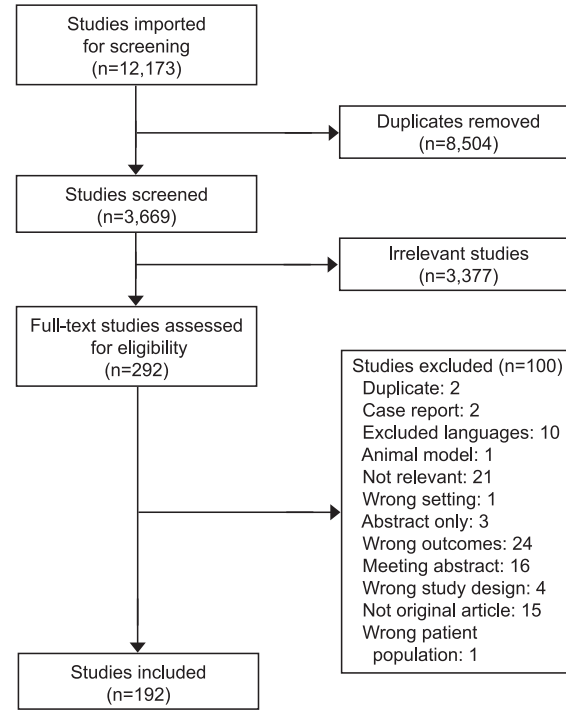

Fig. 2. Results of literature search and two-phase reference screening. An experienced medical librarian devised and conducted the literature searches, which were entered into the systematic review software. References were reviewed in a two-phase approach. Two blinded reviewers independently screened references by title and abstract to determine inclusion. A third reviewer blinded to the previous reviewers' decisions resolved disagreements. Next, the included references were screened at the full-text level by two independent, blinded reviewers to determine eligibility. Disagreements were again resolved by a third blinded reviewer. Possible duplications were reviewed, and, if deemed duplicates, one was excluded.

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mitigates these outcomes.[1,3,7,11,22,23] In the absence of such evidence, our group proceeded with the premise that knowing a pregnant person's OSA status and treating OSA would provide sufficient benefit to their overall health outside of these associated pregnancy-associated outcomes. A similar argument has been made for OSA screening before elective surgery.[24,25] To answer the question of whether screening is warranted for the general obstetric population regardless of known risk factors, we examined studies that were not limited to pregnancies with risk factors for either OSA or adverse pregnancy outcomes but enrolled broadly, including people at low risk.[1,14,26–34] Although some of the studies were large, well designed, and well executed, we did not identify any studies specifically evaluating the benefit of OSA screening in pregnancy.

When we consider other risk factors for OSA in pregnancy, there is considerable evidence that pregnant people with obesity (body mass index [BMI, calculated as weight in kilograms divided by height in meters squared] 30 or higher) have higher rates of OSA, and that OSA risk increases with increasing BMI.[9,35,36] The prevalence of OSA (apnea–hypopnea index of five or more events per hour) among pregnant people with obesity ranges from 2% to 24% in available studies.[9,35–41]

The evidence from studies of people with hypertensive disorders of pregnancy is more difficult to interpret given the heterogeneity of inclusion criteria (chronic hypertension, gestational hypertension, and preeclampsia) and timing of OSA testing during pregnancy. However, there is substantial evidence that people with hypertensive disorders of pregnancy are at higher risk of OSA.[7,9–11,23,42–49] As a result of the onset of preeclampsia's proximity to the timing of delivery, it is impractical to recommend that people diagnosed with preeclampsia get screened for OSA.

Similarly, studies that examined OSA prevalence among people with diabetes are heterogeneous in their inclusion criteria and in the timing of OSA diagnosis during pregnancy. Studies including people with gestational diabetes (type A1 [diet-controlled] compared with A2 [medication-controlled]) suggest that there is a higher prevalence of OSA among people with gestational diabetes.[50–56] There were no studies of pregnant people with pregestational type 1 or type 2 diabetes; however, data from the general, nonpregnant population suggest a strong association between OSA and diabetes.[57] Screening based on a diagnosis of gestational diabetes in the current pregnancy (between 24 and 28 weeks of gestation) was not thought to be useful given the limited available time for diagnostic testing and treatment initiation.

We do not recommend universal screening of all pregnant people for OSA (Class of Recommendation III [no benefit, moderate]; Level of Evidence C-EO). This recommendation is based on expert opinion and extrapolation from the available evidence on the low prevalence of OSA among the general population of pregnant people in the United States. The potential burden on the health system of screening all pregnant people for OSA would be significant. Screening tools with high sensitivity and specificity have not yet been validated, and sleep studies and consultations with sleep medicine specialists are limited in many settings.

We suggest screening pregnant people with obesity (BMI 30 or higher) for OSA (Class of Recommendation IIa [moderate]; Level of Evidence C-EO). There was insufficient evidence to suggest a particular BMI at which OSA risk markedly increases, but the Guideline Committee agreed that in the absence of such data, screening all pregnant people with class I obesity (BMI 30 or higher) and above when measured in the first or second trimester is reasonable. In the absence of symptoms of sleep-disordered breathing, referring patients whose BMIs increase to higher than 30 in the third trimester for sleep evaluation is unlikely to be practical given the time it takes to arrange for a sleep study, initiate therapy, and become acclimated to therapy.

We suggest OSA screening for pregnant people with hypertensive disorders of pregnancy or diabetes in the index or prior pregnancy. Although the potential benefit outweighs the risks, its usefulness is not well established (Class of Recommendation IIb [weak]; Level of Evidence C-EO).

Maternal age emerged as a risk factor for OSA in pregnancy in several studies.[1,9,58,59] However, there was no clear consensus among studies or by expert opinion at which age OSA risk in pregnancy increases. **We do not recommend OSA screening of pregnant people with advanced maternal age and no other risk factors (Class of Recommendation III [no benefit, moderate]; Level of Evidence C-EO).**

Question 1.2. What is the optimal timing for OSA screening during pregnancy?

Although there is still no evidence that treating OSA prevents or mitigates the adverse pregnancy outcomes that have been associated with OSA in pregnancy, there may be other benefits to pregnant people. Obstructive sleep apnea has been associated with daytime sleepiness, poor daytime functioning, cognitive impairment, and roadside accidents. Such

symptomatology improves with treatment of OSA.[3] In the absence of evidence of improved perinatal outcomes in response to OSA treatment, our Guideline Committee deliberated the risks and benefits of timing of screening in each trimester of pregnancy.

Screening for OSA in the third trimester (28 weeks of gestation and later) would likely yield a greater prevalence of OSA compared with screening in early pregnancy. However, the practical challenges of scheduling diagnostic testing and instituting treatment during the third trimester are considerable. Knowing a person's OSA status before obstetric and nonobstetric surgery, because there are existing guidelines that make recommendations for OSA management for perioperative patients, could be beneficial.[60–62] This is especially true for individuals at risk for cesarean delivery because delivery in these people may require intubation and exposure to sedating medications that could complicate underlying OSA. Furthermore, the recent Society for Obstetric Anesthesia and Perinatology guideline on monitoring after neuraxial morphine administration in obstetric patients suggests that OSA be considered a risk factor for respiratory depression.[63] On the basis of these factors, **we recommend that OSA screening of pregnant people at higher risk of OSA by the criteria defined previously in the first or second trimester (6 0/7–28 6/7 weeks) is reasonable (Class of Recommendation IIa [moderate]; Level of Evidence C-EO).**

Question 1.3. What are the most effective screening tools to identify people with OSA in pregnancy?

Question 1.3.1. Is the Berlin questionnaire an effective screening tool for OSA in pregnant people?

The Berlin questionnaire was developed and validated in nonpregnant populations to screen for OSA.[64] It was evaluated in a published meta-analysis of OSA screening questionnaires performed during pregnancy that used objective testing to determine OSA status.[65] Use of the Berlin questionnaire improved the pretest probability of detecting OSA from 26% to 38%.[65] In this meta-analysis, the pooled sensitivity and specificity of the Berlin questionnaire to detect OSA in pregnant people were 0.66 (95% CI 0.45–0.83) and 0.62 (95% CI 0.48–0.75), respectively. **The Berlin questionnaire is a poor predictor of OSA status in pregnant people; therefore, we do not recommend that it be used as a screening tool in this population (Class of Recommendation III [no benefit, moderate]; Level of Evidence B-NR).**

Question 1.3.2. Is the STOP-BANG (snoring history, tired during the day, observed stop of breathing while sleeping, high blood pressure, BMI higher than 35 [or 30], age older than 50 years, neck circumference greater than 40 cm, and male gender) questionnaire an effective screening tool for OSA in pregnant people?

Question 1.3.3. Are the individual components of the STOP-BANG questionnaire associated with OSA in pregnant people?

The STOP-BANG questionnaire was developed and validated in patient populations that did not include pregnant people.[20,21] One component of the eight-item score (male sex) is not applicable to obstetric patients; a second component (age greater than 50 years) is rarely applicable to obstetric patients. Although some studies have suggested that STOP-BANG might be a useful screening tool in pregnant people in the second and third trimesters,[66,67] its sensitivity is low and ranges from 53% to 63%.[32,35,67] Data on specificity are mixed; two studies reported high specificity,[32,67] suggesting that the tool might be acceptable at identifying those without OSA. However, Dominguez et al[35] reported poor specificity of 64%.

We conducted a meta-analysis of three prospective studies that measured the ability of the STOP-BANG tool to detect OSA in pregnant people and found a pooled sensitivity of 0.58 (95% CI 0.46–0.70) and specificity of 0.82 (95% CI 0.77–0.86) with an area under the curve (AUC) of 0.67 (Fig. 3; Appendix 3, available online at http://links.lww.com/AOG/D225).[32,35,67]

A published multivariate logistic regression analysis of variables of STOP-BANG evaluated in each trimester showed that prepregnancy BMI was the only significant predictor of OSA status in the first trimester (odds ratio [OR] 1.4, 95% CI 1.01–2.0, *P*=.04). In the second trimester, snoring often (OR 10.5, 95% CI 5.7–19.33, *P*=.002) was associated with OSA in pregnancy. However, STOP-BANG assesses the volume of snoring rather than the frequency of snoring. In the third trimester, BMI during pregnancy (OR 1.47, 95% CI 1.03–2.10, *P*=.049) was associated with OSA-positive status.[67]

The association of loud snoring with OSA (defined by oxygen desaturation index of five or more events per hour) was also demonstrated in a second-trimester study of pregnant people with class III obesity (OR 6.8, 95% CI 1.7–28.2).[66] Lockhart et al[32] demonstrated that BMI of 35 or higher (OR 4.79, 95% CI 1.79–12.79, *P*=.002) and hypertension (OR 3.66, 95% CI 1.40–9.60, *P*=.008) were associated with OSA status.

Individual components of the STOP-BANG score found to be associated with OSA in pregnant people

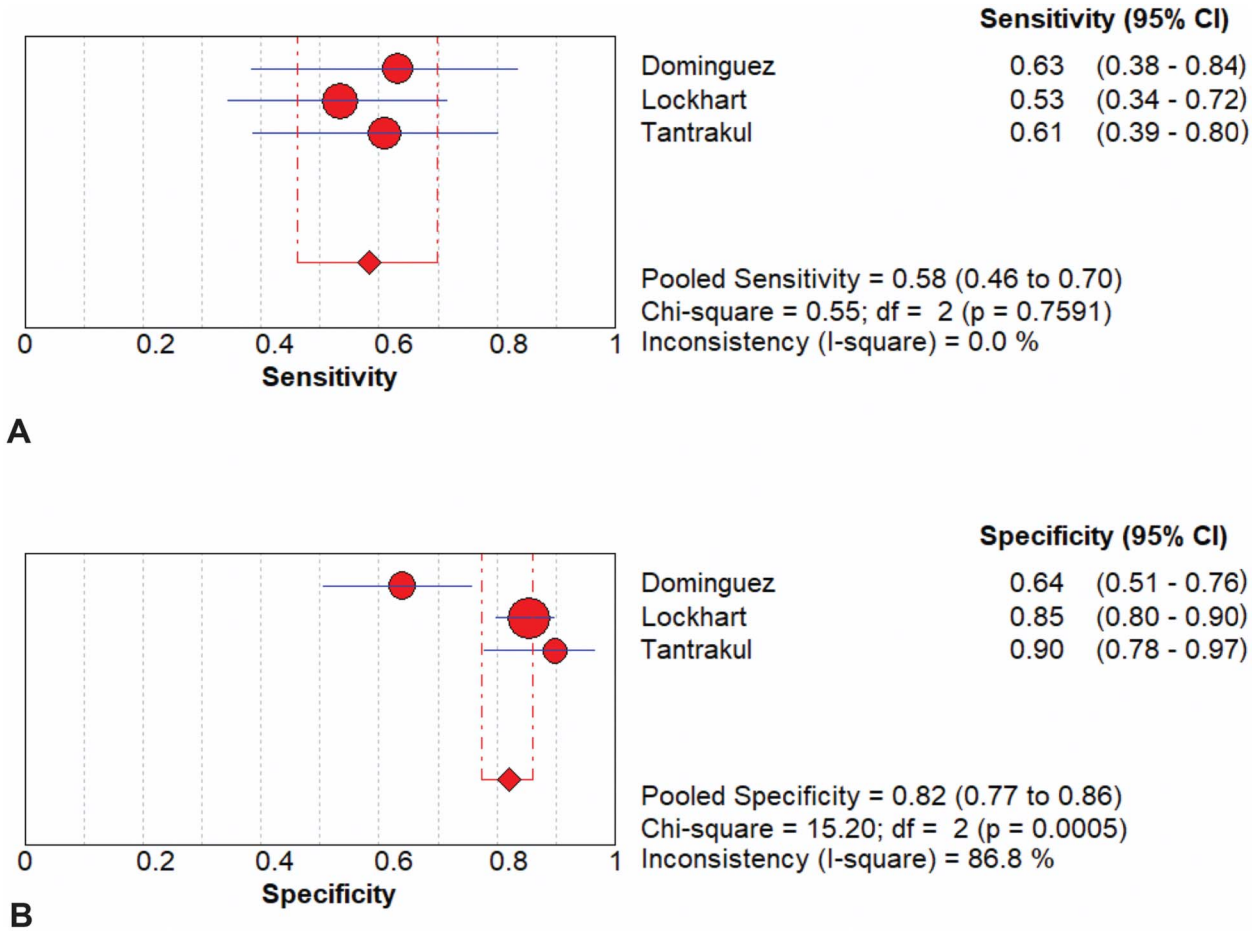

Fig. 3. We conducted a meta-analysis of three prospective studies that measured the ability of the STOP-BANG tool to detect obstructive sleep apnea in pregnant women and found a pooled sensitivity of 0.58 (95% CI 0.46–0.70) (**A**) and pooled specificity of 0.82 (95% CI 0.77–0.86) (**B**).[32,35,67] df, degrees of freedom.

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include loud snoring,[66,67] observed apneas,[32] neck circumference of 16 inches or more,[67] BMI,[32,67] and hypertension.[32,67]

According to our own meta-analysis, **the STOP-BANG questionnaire is a poor predictor of OSA status in pregnant people; therefore, we recommend that it should not be used as a screening tool for OSA in this population (Class of Recommendation III [no benefit, moderate]; Level of Evidence B-NR). We suggest that some of its individual components may be useful predictors of OSA status in combination or in novel screening tools (Class of Recommendation IIb [weak]; Level of Evidence C-LD).**

However, given the dynamic nature of pregnancy, questionnaires may have differential ability to detect OSA in pregnancy, depending on the stage of pregnancy. Furthermore, variability in the data may be related to the population studied (eg, high risk for OSA vs low risk).

Question 1.3.4. Is the Epworth Sleepiness Scale an effective screening tool for OSA in pregnant people?

The Epworth Sleepiness Scale was developed in nonpregnant populations as a self-reported numerical scale to assess daytime sleepiness.[68] It was evaluated in a meta-analysis of OSA screening questionnaires performed during pregnancy and found to be a poor predictor of OSA status.[65] The pooled sensitivity and specificity of the Epworth Sleepiness Scale to detect OSA in pregnant people were 0.44 (95% CI 0.33–0.56) and 0.62 (95% CI 0.57–0.67), respectively.[65] **The Epworth Sleepiness Scale is a poor predictor of OSA status in pregnant people; therefore, we do not recommend that it be used as a screening tool in this population**

(Class of Recommendation III [no benefit, moderate]; Level of Evidence B-NR).

Question 1.3.5. Is the American Society of Anesthesiologists (ASA) checklist an effective screening tool for OSA in pregnant people?

The ASA checklist was proposed by the ASA task force on the perioperative management of nonpregnant surgical patients with OSA to better identify patients at risk for OSA in the perioperative setting.[60] It combines clinical signs and symptoms and can be applied to adults and pediatric patients. In one prospective study of pregnant people, it had reasonable sensitivity to detect OSA in the third trimester (0.767, 95% CI 0.573–0.893) but poor specificity (0.610, 95% CI 0.542–0.675).[32] The ASA checklist did not perform well in another prospective observational study of pregnant people with a low sensitivity (0.23) and specificity (0.05) for the detection of OSA.[35] **The ASA checklist is a poor predictor of OSA status in pregnant people; therefore, we do not recommended that it be used as a screening tool in this population (Class of Recommendation III [no benefit, moderate]; Level of Evidence B-NR).**

Question 1.3.6. Are the screening criteria proposed by Facco et al[34] an effective screening tool for OSA in pregnant people?

Facco and colleagues[34] proposed pregnancy-specific OSA screening criteria that include the sum of age, prepregnancy BMI, and 15 points each for chronic hypertension and frequent snoring if they are present. Subsequently, three small prospective cohort studies applied the tool to pregnant women, and it did not perform as well as in the original derived cohort.[26,35,69]

We performed a meta-analysis on the four studies that applied the Facco et al[34] criteria to their study cohorts (including the derived cohort).[26,34,35,69] We found a pooled sensitivity of 0.74 (95% CI 0.64–0.82) and pooled specificity of 0.64 (95% CI 0.58–0.70, AUC 0.82) (Fig. 4; Appendix 4, available online at http://links.lww.com/AOG/D225). **We suggest that the published pregnancy-specific OSA screening criteria proposed by Facco et al may be considered as a screening tool for OSA in pregnant people but performs suboptimally in populations at high risk for OSA (Class of Recommendation IIb [weak]; Level of Evidence B-NR).** Additional studies in larger cohorts of patients at higher risk for OSA are needed to establish its usefulness in these populations.

Question 1.3.7. Are the screening criteria proposed by Louis et al[59] an effective screening tool for OSA in pregnant people?

Louis et al[59] developed a new prediction model for OSA in pregnancy in the large, observational nuMoM2b (Nulliparous Pregnancy Outcomes Study: Monitoring Mothers-to-Be) Sleep-Disordered Breathing Substudy that includes current maternal age, BMI, and frequent snoring (three or more times per week during the previous 4 weeks). The model (calculator available at https://www.ajog.org/cms/10.1016/j.ajog.2018.01.031/attachment/0c744a7b-39b4-4236-a2a6-fbc8125c892c/mmc1.xlsx) generates the predicted probability of an individual pregnant person having an apnea–hypopnea index of five or more events per hour in early (6–13 weeks of gestation) pregnancy with an AUC of 0.87 and midpregnancy (22–29 weeks of gestation) with an AUC of 0.84, as well as the predicted probability of new-onset OSA in midpregnancy with an AUC of 0.81. Although promising, this tool needs to be validated in another cohort. One limitation of this study was the low prevalence of OSA in this relatively healthy cohort. The screening criteria should be tested outside of the derived cohort to establish their usefulness in a cohort of patients with a higher prevalence of OSA risk factors. **We suggest that the published pregnancy-specific OSA screening criteria proposed by Louis et al[59] need to be further validated outside the derived cohort (Class of Recommendation IIb [weak]; Level of Evidence C-LD).**

Question 1.3.8. Is the BATE screening criteria an effective screening tool for OSA in pregnant people?

Izci-Balserak et al[69] developed a new model (BATE) to predict OSA in pregnant people in the first trimester that includes BMI+age+(15×tongue enlargement [0 or 1]). Tongue enlargement was assessed with the study participant breathing through their nose with the tongue in a relaxed position; the tongue was graded as enlarged if it protruded beyond the teeth or alveolar ridge in this resting position. Scores of 65 or greater were associated with OSA in pregnant people with a sensitivity of 0.77 and specificity of 0.82 (AUC 0.86). In the third trimester, the model is adjusted to include BMI+age+(20×tongue enlargement [0 or 1]). A score of 75 or greater was associated with OSA in pregnant people with a sensitivity of 0.76 and specificity of 0.82 (AUC 0.87). BATE may be a useful screening tool but needs to be validated in a separate cohort and relies on the ability of the rater to assess tongue enlargement, which requires training. **We suggest that the published pregnancy-specific screening model (BATE)[69] may be a useful predictor of OSA status in pregnant people, but it requires clinical training and needs to be further validated outside the derived cohort (Class of**

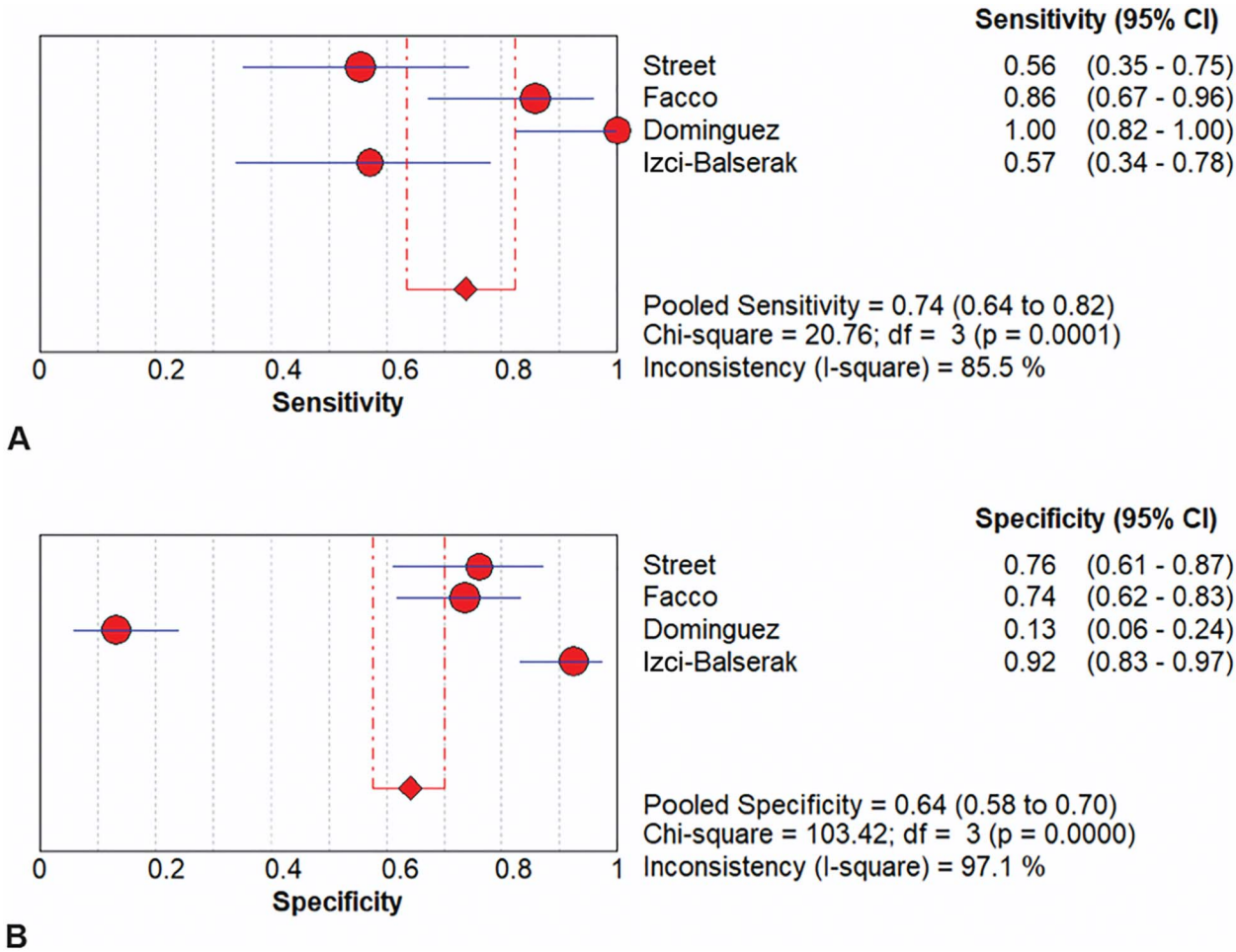

Fig. 4. We performed a meta-analysis on the four studies that applied the Facco et al criteria to their study cohorts (including the derived cohort).[26,35,69] We found a pooled sensitivity of 0.74 (95% CI 0.64–0.82) (**A**) and specificity of 0.64 (95% CI 0.58–0.70) (**B**). df, degrees of freedom.

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Recommendation IIb [weak]; Level of Evidence C-LD).

2. Diagnosis of OSA in Pregnant People

Question 2.1. Is home sleep apnea testing an effective diagnostic tool for OSA in pregnant people?

Several studies directly compared portable sleep apnea testing with polysomnography and showed good agreement between an apnea–hypopnea index or respiratory disturbance index (apnea–hypopnea index+respiratory event–related arousals) derived by portable devices, and apnea–hypopnea index or respiratory disturbance index measured by polysomnography.[9,41,70,71] Portable devices are particularly helpful in pregnant people for convenience and practicality. Several studies describing associations between sleep-disordered breathing and adverse pregnancy outcomes have been based on portable monitoring.[1,9,72]

The validity and limitations of portable sleep monitoring in the general population and in pregnancy are discussed in more detail in Appendix 5, available online at http://links.lww.com/AOG/D225. Studies show that portable monitoring compared with polysomnography is generally less sensitive for detecting disease in pregnant people. This is likely related to multiple factors, including unknown sleep time, self-application or technical issues, and less sensitivity for subtle events such as flow limitations or respiratory event–related arousals, which tend to be observed more commonly in young people. Sharkey et al[70] showed the strength of correlation between the Apnea Risk Evaluation System–apnea–hypopnea index and polysomnography–apnea–hypopnea index varied

markedly, depending on the desaturation criteria applied to the Apnea Risk Evaluation System Unicorder's apnea–hypopnea index calculation. Using a 3% algorithm on the Apnea Risk Evaluation System had the best balance of sensitivity and specificity for detecting OSA if OSA was defined by polysomnography–respiratory disturbance index or five or more events per hour that included 4% hypopneas and respiratory event–related arousals. Facco et al[41] did not specify how apnea–hypopnea index was calculated in their study, but they found excellent categorical agreement for a negative OSA diagnosis with the ApneaLink and polysomnography in pregnant people in the third trimester. However, the ApneaLink missed two of four positive OSA diagnoses on polysomnography (apnea–hypopnea index of five or more events per hour), suggesting that the ApneaLink device may underdiagnose OSA in pregnant people. Furthermore, a higher WatchPAT–apnea–hypopnea index threshold of 6.1 events per hour was required for the best sensitivity (0.88) and specificity (0.91) for detecting polysomnography–apnea–hypopnea index of five events or more per hour (3% desaturation or arousal hypopnea criteria) in pregnant people.[71]

Portable monitoring has been validated largely among people with obesity during late pregnancy. Portable sleep apnea testing devices have not been rigorously validated in people without obesity in early pregnancy, in whom there may be less risk for OSA or more subtle forms of OSA. In the absence of evidence, it may be reasonable to extrapolate from young nonpregnant people and to recommend portable testing for OSA in patients in early pregnancy without obesity only if there is a clinical suspicion for severe disease.

Despite these potential pitfalls of home sleep testing, portable monitoring remains an attractive diagnostic modality compared with in-laboratory polysomnography because of its relative simplicity and convenience, which may ultimately result in greater ascertainment of OSA during pregnancy. **We suggest that out-of-center (home) sleep apnea testing may be a reasonable diagnostic tool for OSA in pregnant people (Class of Recommendation IIb [weak]; Level of Evidence B-NR).**

Question 2.2. Is overnight oximetry an effective diagnostic tool for OSA in pregnant people?

There are no studies validating the use of overnight oximetry for diagnosing OSA in pregnant people. In the nonpregnant population, overnight oximetry has been shown to be potentially useful in detecting severe OSA but has limited sensitivity for detecting mild or moderate disease.[73,74] Several studies show that very few pregnant people have oxygen desaturation events during sleep.[66,75–77] Moreover, the prevalence of the oxygen desaturation index of five or more events per hour in pregnant people varied greatly, depending on desaturation criteria. **Because of the lack of evidence in pregnant people, great variance in prevalence with changing desaturation criteria, and poor correlation with symptoms and clinical outcomes, we do not recommend that overnight pulse oximetry be used as a diagnostic tool for OSA in pregnant people (Class of Recommendation III [no benefit]; Level of Evidence C-EO).** Whether oximetry may be useful as a screening tool for OSA during pregnancy is unknown.

Question 2.3. Is repeat diagnostic testing warranted in the postpartum period for people who are diagnosed with OSA in pregnancy?

Three small prospective studies assessed the course of OSA during and after pregnancy and found that OSA may improve in the postpartum period for some people.[78,79] In the Amnakkittikul et al[79] study of people with obesity, gestational diabetes, and OSA diagnosed during pregnancy (mean±SD gestational age 28.9±2.9 weeks), the postpartum median apnea–hypopnea index (measured by WatchPAT) improved from 9.5 (interquartile range 4.8–14.6) during pregnancy to 5.5 (interquartile range 2.4–9.7) postpartum, with resolution of OSA in 47% of people. Postpartum weight loss was not associated with resolution of OSA in all people.

Reid et al[78] showed that people with gestational hypertension compared with those with uncomplicated pregnancies were more likely to have elevated respiratory disturbance index during pregnancy (polysomnography–respiratory disturbance index 12.0±12.3 vs 2.8±5.3) but experienced improvement in the postpartum period, reaching respiratory disturbance index levels comparable with those in uncomplicated pregnancies (respiratory disturbance index 2.9±2.9 vs 2.1±3.2). Most of the people in this study with uncomplicated pregnancies did not have abnormal apnea–hypopnea index or respiratory disturbance index (polysomnography–apnea–hypopnea index 4% 0.9±2.4, respiratory disturbance index 2.8±5.3) during pregnancy and therefore did not experience significant changes in the postpartum period. It was noted that, although both groups lost weight postpartum, people with gestational hypertension persistently had more obesity postpartum than people with uncomplicated pregnancies. Mean±SD BMI decreased antepartum to

postpartum from 37.6±5.9 to 32.5±6.8 ($P<.01$) in people with gestational hypertension and from 28.8±4.2 to 22.8±4.0 ($P<.01$) in people who were normotensive.[78]

Street et al[26] found that among people diagnosed with OSA during their third trimester (diagnosed by WatchPAT as five or more events per hour), at least 59% (n=16/24) had persistent OSA at 6–15 weeks postpartum, which persisted in the majority of people who completed repeat testing at 6–8 months postpartum. People with persistent postpartum OSA tended to have greater OSA severity during their third trimester compared with people whose OSA resolved postpartum.

Several prospective cohort studies have demonstrated progression of OSA during pregnancy, which suggests that OSA risk and severity may be influenced directly by factors related to pregnancy and therefore warrant re-evaluation after pregnancy. It is unknown why OSA resolves in some people who develop OSA during pregnancy whereas others have persistent postpartum OSA and, moreover, why some people go on to develop OSA postpartum when it was not present during pregnancy.[26] **We suggest that repeat diagnostic testing in the postpartum period for people diagnosed with OSA during pregnancy may be considered (Class of Recommendation IIb [weak]; Level of Evidence C-LD).**

Question 2.4. If repeat diagnostic testing is performed in the postpartum period for people who are diagnosed with OSA in pregnancy, what is the recommended timing?

There is a paucity of data to guide the timing of repeat sleep testing; available studies differ in terms of timing of repeat testing.[26,78–80] A summary of available studies is presented in Appendix 6, available online at http://links.lww.com/AOG/D225. Reports of snoring and Epworth Sleepiness Scale scores remained similar before and after pregnancy; thus, symptoms poorly inform the timing of repeat testing. Without more data, the timing of repeat postpartum testing can be recommended only on an individual or case-by-case basis. For instance, it may be reasonable to schedule repeat testing once postpartum weight loss plateaus and nocturnal responsibilities related to infant care have decreased. **The recommended timing of postpartum OSA testing for people diagnosed with OSA during pregnancy is unknown (Class of Recommendation IIb [weak]; Level of Evidence C-EO).**

1. Treatment of OSA in Pregnant People

Question 3.1. Does treatment of OSA with continuous positive airway pressure (CPAP) affect markers of cardiovascular and metabolic risk in pregnant people?

Evidence in nonpregnant populations indicates that CPAP treatment improves cardiometabolic outcomes such as producing clinically significant reductions in blood pressure.[81–84] In pregnancy, however, data from studies examining the effect of CPAP therapy on cardiovascular and metabolic pregnancy outcomes are limited by study design, small sample size, short duration of therapy, and study execution. There are studies of CPAP in people with preeclampsia (without documented OSA).[85–89] Although the current scientific evidence on the effect of CPAP on cardiometabolic outcomes is limited,[85–92] the potential treatment benefit on cardiovascular and metabolic risk is deemed to exceed any anticipated risk of treatment. The safety of CPAP therapy has not been regularly evaluated in studies of pregnant people, but there is no evidence that CPAP therapy is harmful in pregnancy.[15]

Given the current state of the science on the long-term use of CPAP in pregnant people with OSA, pregnant people being evaluated for treatment of OSA should be counseled on the current lack of evidence that treatment of OSA with CPAP significantly modifies cardiovascular or metabolic pregnancy outcomes. **The usefulness of OSA treatment on markers of cardiovascular and metabolic risk in pregnant people is unknown, but the potential benefit of CPAP therapy of patients with OSA is thought to outweigh the risk, and CPAP is recommended (Class of Recommendation IIb [weak]; Level of Evidence B-NR [one study]/C-LD [six studies]).**

Question 3.2. Does treatment of OSA in pregnant people influence their fetal and neonatal outcomes?

Sleep-disordered breathing and OSA have been associated with adverse neonatal outcomes in population-based and animal studies. Population-based studies demonstrated that maternal OSA and sleep-disordered breathing were associated with an increased risk for a neonate to be admitted to the NICU[3,8] and to be diagnosed with congenital anomalies compared with neonates of mothers without sleep-disordered breathing.[8] Several studies have demonstrated a significant association between sleep-disordered breathing and preterm birth.[3,8,23,93] The association of sleep-disordered breathing with fetal growth abnormalities is more complicated because

the effect varied, depending on definitions of the disorder and the outcome. Furthermore, although there is evidence supporting an association between OSA and growth restriction, there has also been evidence supporting growth acceleration.[93] Data from animal studies suggest that gestational intermittent hypoxia may alter respiratory motor control in the offspring.[94] Moreover, gestational intermittent hypoxia in a rat model was associated with behavioral phenotypes associated with excessive synapse numbers, implicating hyperactivity of specific pathways in behavioral aberrations.[95] These findings have potential implications for neuropsychiatric disorders.[95]

Hence, we aimed to examine the level of evidence for whether OSA treatment affects 1) duration of gestation, 2) fetal growth, 3) preterm birth, or 4) NICU admission. We found no evidence in the literature examining these questions to date. We also aimed to examine the level of evidence for whether treatment of OSA affects measures of fetal well-being (fetal heart rate, movement, nonstress tests). In a small study by Blyton et al,[85] the investigators measured fetal movement with and without CPAP in people with preeclampsia with severe features. Sleep characteristics were measured during the first night of sleep in participants with no known sleep disorders and showed increased mild-to-moderate airway obstruction and airway flow limitations in the participants with preeclampsia. This study showed a significant increase in fetal movement and fetal hiccups after the application of CPAP over 1 night. These data show promise as to the role of CPAP in preeclampsia; however, this study used an experimental design in participants without known OSA over 1 night and had a small sample size. Future studies are needed to examine how CPAP influences fetal well-being. **The usefulness of treatment of OSA on fetal and neonatal outcomes is unknown; thus, treatment for this indication alone is not recommended (Class of Recommendation IIb [weak]; Level of Evidence C-LD).**

Question 3.3. Does treatment of OSA alter the symptoms, apnea–hypopnea index, and nocturnal oxygen saturations of pregnant people?

Treatment of OSA in pregnancy is likely to be safe,[96] and although studies evaluating the effect on symptoms such as apnea–hypopnea index and nocturnal oxygen saturation are scarce, available data suggest CPAP is effective and beneficial. A case series included 12 people in early pregnancy who were started on nasal CPAP at a mean gestational age of 10 weeks. Seven participants had a preexisting diagnosis of OSA and were established on CPAP, and five participants were diagnosed in their first trimester. Fatigue and sleepiness worsened despite CPAP and were attributed to pregnancy itself. Other signs and symptoms of sleep-disordered breathing improved, and at 33.4 (32–35) weeks of gestation, no apneas, hypopneas, tachypneas, or oxygen desaturations were detected. Blood pressure remained stable in these people, and all participants delivered healthy term neonates with no adverse events.[96] Subsequently, another small case series (n=5) of pregnant patients at 16–24 weeks of gestation with OSA of varying severity reported improvement in symptoms in all (n=4) who used CPAP. In addition, two of these patients had improvement of blood pressure recordings.[91] A randomized controlled trial of nasal CPAP in 24 individuals with preeclampsia with severe features demonstrated a reduction in respiratory disturbance index after 2 nights of CPAP. Respiratory disturbance index varied widely with CPAP during the first night of sleep, and there was no statistically significant decrease.[88] Although gastroesophageal reflux disease is prevalent in pregnancy[97] and CPAP may increase the risk for aerophagia and possibly aspiration, this risk appears to be more theoretical, with no reports of gastroesophageal reflux disease in pregnant individuals treated with CPAP.[98] Furthermore, sleep medicine clinicians on our Consensus Guideline committee have not seen this as a common concern in clinical practice. **We recommend CPAP therapy to reduce symptoms of OSA and apnea–hypopnea index in pregnant people (Class of Recommendation IIa [moderate]; Level of Evidence C-LD/EO).**

Question 3.4. Does treatment of OSA affect maternal obstetric outcomes?

Maternal OSA has been associated with an increased risk for hysterectomy, postoperative wound complications, and cesarean delivery.[7,23] However, the evidence for the treatment of OSA improving maternal obstetric outcomes is very limited and based on small studies that were not designed or sufficiently powered to detect differences in obstetric outcomes.[85–87]

Data suggest a dose–response relationship between OSA severity by apnea–hypopnea index and the risk of cardiovascular and metabolic, pregnancy-specific outcomes.[1] However, even mild forms of sleep-disordered breathing are associated with the development of complications such as hypertensive disorders of pregnancy.[1] Despite consistent associations between OSA and adverse perinatal outcomes, there is no high-level evidence to demonstrate that treatment of OSA would reduce the risk of adverse

pregnancy outcomes. Hence, discussions with the pregnant person with OSA and their family should focus on a discussion of OSA and its symptoms, consequences, and associated outcomes, as well as the limitations of our knowledge of the effect of treatment on outcomes. It is also important to keep in mind that OSA, although it improves in the postpartum period, persists in nearly 50% of people diagnosed in pregnancy. Hence, the discussion with the pregnant person should make the point that this is not a condition that is necessarily self-limited to pregnancy; counseling should take into consideration the influence of OSA on long-term cardiovascular and metabolic outcomes.

Because the safety profile of OSA treatment in pregnancy is thought to be favorable, the risk of treating OSA in pregnancy is minimal. The usefulness of OSA treatment on maternal obstetric outcomes is unknown because available evidence is based on small samples, and outcomes were not primary. **If therapy is offered to pregnant people with OSA, we suggest counseling patients that therapy is not aimed at the modification of pregnancy-specific outcomes but rather directed at the treatment of symptoms, normalization of objective measures of OSA, and improvement in quality of life in general (Class of Recommendation IIb [weak]; Level of Evidence C-LD).**

Question 3.5. Is treatment of preexisting OSA, pregnancy-onset (gestational) OSA, and postpartum OSA different from treatment in nonpregnant people?

Pregnancy-related physiologic changes may worsen preexisting OSA or lead to gestational OSA. Given the adverse health consequences of OSA in pregnant and nonpregnant populations, people with preexisting OSA need to continue their treatment during pregnancy. CPAP pressure may need to be adjusted[96] because of weight gain and other pregnancy-related changes that may exacerbate OSA severity. However, there are no pregnancy-specific guidelines for OSA treatment during pregnancy. Gestational OSA has been reported to improve after delivery,[99–101] possibly as a result of reductions in levels of sex hormones, weight loss, and loss of fluid accumulated during the course of pregnancy, but may persist in nearly half of patients diagnosed in pregnancy. Current evidence to inform practice is lacking. **We suggest treating pregnant and postpartum people with OSA with the standard of care for nonpregnant people with OSA (Class of Recommendation IIb [weak]; Level of Evidence C-EO).**

Question 3.6. Do treatment approaches for preexisting OSA, pregnancy-onset (gestational) OSA, and postpartum OSA differ?

There is no evidence or guidance in the literature on whether preexisting OSA, pregnancy-onset (gestational) OSA, or postpartum OSA should be treated differently. Hence, our recommendations are based on the Guideline Committee members' clinical experience with sleep-disordered breathing in pregnancy and their research experience, including unpublished data.

Issues that need to be taken into consideration include pressure requirements, factors that may affect adherence to therapy, and outcomes. There is some evidence that pressure requirements increase slightly with gestational age; hence, the use of autotitrating CPAP is advisable, with evaluation of CPAP downloads for adequacy of pressures two to three times during pregnancy and postpartum. Although case series and small studies describe adequate treatment at low pressures, in our experience, some patients may require pressures as high as 16–18 cm H_2O to eliminate OSA. Hence, we recommend initiating therapy with a wide range of pressures and then narrowing the range on the basis of patients' individual data.

Barriers to the use of CPAP in pregnancy may be similar to those in the nonpregnant population, and others are specific to pregnancy. Several studies report and comment on adherence to CPAP in pregnancy, although at the time of this writing no study has evaluated adherence as a primary outcome. Some members of the Guideline Committee are using a qualitative approach to examine CPAP adherence in pregnant and nonpregnant people of reproductive age to identify barriers and facilitators to the use of CPAP in pregnancy. Common barriers to adherence in nonpregnant people can be related to patient characteristics, machine type, fit, and titration method or psychosocial issues.[102] Symptoms of pregnancy itself may compound these issues such as restless sleep attributable to discomfort or fetal movement. Interventions that may improve adherence include patient choice of mask; behavioral treatments such as educational programs, cognitive behavioral therapy, and motivational interviewing[103]; and in particular partner support because it has been shown that married women have worse adherence compared with unmarried women, probably attributable to the "intrusiveness of the treatment into couple intimacy."[104]

Nasal congestion is a common occurrence in pregnancy and may influence adherence to CPAP treatment. The application of nasal dilator strips does not affect self-reported measures or objective measures

of OSA.[105] However, treatment with nasal saline or nasal corticosteroids may help with nasal congestion. Sleep is most disrupted in the third trimester because of various physiologic factors. These disruptions may also influence comfort and adherence to CPAP.

In the postpartum period, nocturnal obligations such as newborn care can significantly disrupt sleep. In addition, logistic barriers may affect CPAP adherence. These barriers include difficulty finding a space by the bedside for the device because of the newborn sleeping nearby or hesitation to disconnect from the CPAP device repeatedly with newborn awakenings. Postpartum individuals can be counseled to plan for positioning of the device near the bed if the newborn will be sleeping in the same room as the mother. Mothers can be encouraged to use their CPAP device while sleeping and disconnect when awakened. Conversations about anticipated challenges may be helpful to have before delivery, which may also enable planning for the delivery hospitalization. **Expert consensus suggests the use of autotitrating CPAP with a wide range similar to nonpregnant individuals at initiation with periodic review of nocturnal CPAP data during pregnancy and postpartum (Class of Recommendation IIb [weak]; Level of Evidence C-EO).**

Question 3.7. What are the clinical indications for treatment of OSA in pregnant people, and when should treatment be initiated?

To date, there are no data to guide which pregnant patients need to be treated. Indications for treatment are unclear in pregnancy, and extrapolation from the nonpregnant population may be helpful, with the caveat that there are no data to demonstrate that CPAP clearly modifies pregnancy, fetal, or neonatal cardiovascular or metabolic outcomes. Counseling of pregnant people with OSA should be performed accordingly. Another important consideration in the pregnant population that is not as central in the nonpregnant population is the timing (and hence the urgency) of treatment initiation during pregnancy. Pregnancy is a dynamic physiologic state with some profound hemodynamic, hormonal, and respiratory changes that change with pregnancy progression, with pathology predisposing to adverse outcomes that occur at specific time intervals during pregnancy. In addition, cardiovascular and metabolic outcomes associated with sleep-disordered breathing occur after a significantly more contracted period of time in pregnancy compared with the nonpregnant population. Although hypertension and diabetes develop over nearly a decade in nonpregnant people with OSA, the equivalent pregnancy-related disorders (gestational hypertension, preeclampsia, and gestational diabetes) develop over a few months. Hence, if future data demonstrate improvement of perinatal outcomes with treatment of OSA, initiation of treatment will need to be timely to maximize the benefit over the three trimesters of pregnancy. **Expert consensus opinion suggests similar treatment of pregnant and nonpregnant individuals, with the understanding that there are insufficient data to demonstrate that CPAP clearly modifies perinatal cardiovascular, metabolic, fetal, or neonatal outcomes (Class of Recommendation IIb [weak]; Level of Evidence C-EO).**

Question 3.8. Are lifestyle changes useful in the treatment of OSA in pregnant people?

There were no published studies on whether lifestyle changes modify OSA in pregnancy. However, general recommendations usually provided in routine clinical prenatal care can be further emphasized in people with OSA during pregnancy. These include avoidance of alcohol and smoking. Weight loss is usually not advised during pregnancy, but adherence to recommended weight gain by body habitus is advisable. **There are no data that examine the effect of lifestyle changes on OSA in pregnant people, and their usefulness is unknown. Weight loss is not recommended in pregnancy. However, because alcohol cessation and smoking cessation are established recommended lifestyle changes for pregnant people, we suggest that they be recommended for the treatment of OSA in pregnant people (Class of Recommendation IIb [weak]; Level of Evidence C-EO).**

SUMMARY

A summary of our recommendations can be found in Table 1.

GAPS AND FUTURE RESEARCH DIRECTIONS

Many questions remain unanswered on the screening, diagnosis, and treatment of OSA in pregnant people and the potential for treatment to mitigate adverse maternal and neonatal outcomes that have been associated with OSA. There is still insufficient evidence to recommend any one OSA screening tool. In the absence of validated screening tools, the Guideline Committee recommends assessing pregnant people at high risk for OSA (those with BMIs higher than 30, chronic hypertension, or pregestational diabetes or gestational diabetes in a prior

Table 1. Summary Recommendations for the Screening, Diagnosis, and Treatment of Obstructive Sleep Apnea in Pregnant People

Recommendation	Class of Recommendation	Level of Evidence
1. Screening		
1.1.1. We do not recommend universal screening of all pregnant people for OSA.	III (no benefit, moderate)	C-EO
1.1.2. We suggest screening pregnant people with obesity (BMI 30 or higher) for OSA.	IIa (moderate)	C-EO
1.1.3. We suggest OSA screening for pregnant people with hypertensive disorders of pregnancy or diabetes in the index or prior pregnancy. Although the potential benefit outweighs the risks, its usefulness is not well established.	IIb (weak)	C-EO
1.1.4. We do not recommend OSA screening of pregnant people with advanced maternal age and no other risk factors.	III (no benefit, moderate)	C-EO
1.2. We recommend OSA screening of pregnant people at higher risk of OSA in the first or second trimester (6 0/7–28 6/7 wk) is reasonable.	IIa (moderate)	C-EO
1.3.1. The Berlin questionnaire is a poor predictor of OSA status in pregnant people; therefore, we do not recommend it be used as a screening tool in this population.	III (no benefit, moderate)	B-NR
1.3.2. The STOP-BANG questionnaire is a poor predictor of OSA status in pregnant people; therefore, we recommend it should not be used as a screening tool for OSA in this population.	III (no benefit, moderate)	B-NR
1.3.3. We suggest that some individual components of the STOP-BANG questionnaire may be useful predictors of OSA status in pregnancy in combination or in novel screening tools.	IIb (weak)	C-LD
1.3.4. The Epworth Sleepiness Scale is a poor predictor of OSA status in pregnant people; therefore, we do not recommend that it be used as a screening tool in this population.	III (no benefit, moderate)	B-NR
1.3.5. The ASA checklist is a poor predictor of OSA status in pregnant people; therefore, we do not recommend it be used as a screening tool in this population.	III (no benefit, moderate)	B-NR
1.3.6. We suggest the published pregnancy-specific OSA screening criteria proposed by Facco et al[34] may be considered as a screening tool for OSA in pregnant people but performs suboptimally in populations at high risk for OSA.	IIb (weak)	B-NR
1.3.7. We suggest that the published pregnancy-specific OSA screening criteria proposed by Louis et al[59] need to be further validated outside the derived cohort.	IIb (weak)	C-LD
1.3.8. We suggest that the published pregnancy-specific screening model (BATE)[69] may be a useful predictor of OSA status in pregnant people, but it requires clinical training and needs to be further validated outside the derived cohort.	IIb (weak)	C-LD
2. Diagnosis		
2.1. We suggest that out-of-center (home) sleep apnea testing may be a reasonable diagnostic tool for OSA in pregnant people.	IIb (weak)	B-NR
2.2.Because of the lack of evidence in pregnant people, great variance in prevalence with changing desaturation criteria, and poor correlation with symptoms and clinical outcomes, we do not recommend overnight pulse oximetry be used as a diagnostic tool for OSA in pregnant people.	III (no benefit)	C-EO
2.3.We suggest that repeat diagnostic testing in the postpartum period for people diagnosed with OSA during pregnancy may be considered.	IIb (weak)	C-LD
2.4. The recommended timing of postpartum sleep apnea testing for people diagnosed with OSA during pregnancy is unknown.	IIb (weak)	C-EO
3. Treatment		
3.1. The usefulness of OSA treatment on markers of cardiovascular and metabolic risk in pregnant people is unknown, but the potential benefit of CPAP therapy in patients with OSA is thought to outweigh the risk, and CPAP is recommended.	IIb (weak)	B-NR (1 study)/C-LD (6 studies)
3.2. The usefulness of treatment of OSA on fetal and neonatal outcomes is unknown; thus, treatment for this indication alone is not recommended.	IIb (weak)	C-LD
3.3. We recommend CPAP therapy to reduce symptoms of OSA and AHI in pregnant people.	IIa (moderate)	C-LD/EO

(*continued*)

Table 1. Summary Recommendations for the Screening, Diagnosis, and Treatment of Obstructive Sleep Apnea in Pregnant People (*continued*)

Recommendation	Class of Recommendation	Level of Evidence
3.4. If therapy is offered to pregnant people with OSA, we suggest counseling patients that therapy is not aimed at the modification of pregnancy-specific outcomes but rather directed at the treatment of symptoms, normalization of objective measures of OSA, and improvement in quality of life in general.	IIb (weak)	C-LD
3.5. We suggest treating pregnant and postpartum people with OSA with the standard of care for nonpregnant people with OSA.	IIb (weak)	C-EO
3.6. Expert consensus suggests the use of autotitrating CPAP with a wide range similar to nonpregnant individuals at initiation with periodic review of nocturnal CPAP data during pregnancy and postpartum.	IIb (weak)	C-EO
3.7. Expert consensus opinion suggests treatment of pregnant individuals similar to that of nonpregnant individuals, with the understanding that there are insufficient data that demonstrate that CPAP clearly modifies perinatal cardiovascular, metabolic, fetal, or neonatal outcomes.	IIb (weak)	C-EO
3.8. There are no data that examine the effect of lifestyle changes on OSA in pregnant people, and their usefulness is unknown. Weight loss is not recommended in pregnancy. However, because alcohol cessation and smoking cessation are established recommended lifestyle changes for pregnant people, we suggest that they be recommended for the treatment of OSA in pregnant individuals.	IIb (weak)	C-EO

OSA, obstructive sleep apnea; BMI, body mass index; STOP-BANG, snoring history, tired during the day, observed stop of breathing while sleeping, high blood pressure, body mass index higher than 35 (or 30), age older than 50 years, neck circumference greater than 40 cm, and male gender; ASA, American Society of Anesthesiologists; CPAP, continuous positive airway pressure; AHI, apnea–hypopnea index.

or index pregnancy) in the first or second trimester for symptoms of sleep-disordered breathing (loud snoring, nocturnal gasping or witnessed apneas, or daytime sleepiness or excessive fatigue). Additional research is needed to further elucidate these areas of uncertainty and to guide future evidence-based clinical practice. A more in-depth discussion of gaps in knowledge and future research directions is included in Appendix 7, available online at http://links.lww.com/AOG/D225.

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