

SPECIAL ARTICLES

Use of polysomnography and home sleep apnea tests for the longitudinal management of obstructive sleep apnea in adults: an American Academy of Sleep Medicine clinical guidance statement

Sean M. Caples, DO, MS¹; W. McDowell Anderson, MD²; Karel Calero, MD²; Michael Howell, MD³; Sarah D. Hashmi, MPH, MSc, MBBS⁴

¹Mayo Clinic, Rochester, Minnesota; ²University of South Florida, Tampa, Florida; ³University of Minnesota, Minneapolis, Minnesota; ⁴American Academy of Sleep Medicine, Darien, Illinois

Introduction: Obstructive sleep apnea is an important and common disorder with associated health risks. Assuring successful longitudinal management is vital to patient health and sleep-related quality of life. This paper provides guidance from the American Academy of Sleep Medicine (AASM) regarding the use of polysomnography (PSG) and home sleep apnea tests (HSATs) after a diagnosis of obstructive sleep apnea has been established and, in most cases, treatment implemented.

Methods: The AASM commissioned a task force of five sleep medicine experts. A literature search was conducted to identify studies that included adult patients with OSA who underwent follow-up PSG or an HSAT. The task force developed clinical guidance statements based on a review of these studies and expert opinion. The AASM Board of Directors approved the final clinical guidance statements.

Clinical Guidance Statements: The AASM supports the following clinical guidance statements on indications for follow-up PSG and HSAT in adult patients with OSA.

1. Follow-up PSG or HSAT is *not* recommended for routine reassessment of asymptomatic patients with obstructive sleep apnea on PAP therapy, however, follow-up PSG or HSAT can be used to reassess patients with recurrent or persistent symptoms, despite good PAP adherence.
2. Follow-up PSG or HSAT is recommended to assess response to treatment with non-PAP interventions.
3. Follow-up PSG or HSAT may be used if clinically significant weight gain or loss has occurred since diagnosis of OSA or initiation of its treatment.
4. Follow-up PSG may be used for reassessment of sleep-related hypoxemia and/or sleep-related hypoventilation following initiation of treatment for OSA.
5. Follow-up PSG or HSAT may be used in patients being treated for OSA who develop or have a change in cardiovascular disease.
6. Follow-up PSG may be used in patients with unexplained PAP device-generated data.

The ultimate judgment regarding propriety of any specific care must be made by the clinician, in light of the individual circumstances presented by the patient, available diagnostic tools, accessible treatment options and resources.

Citation: Caples SM, Anderson WM, Calero K, Howell M, Hashmi SD. Use of polysomnography and home sleep apnea tests for the longitudinal management of obstructive sleep apnea in adults: an American Academy of Sleep Medicine clinical guidance statement. *J Clin Sleep Med.* 2021;17(6):1287–1293.

INTRODUCTION

Obstructive sleep apnea (OSA) is an important public health problem due to its prevalence, intersection with comorbidities, and associated health and safety risks, including cardiovascular disease and motor vehicle crashes. Several previous American Academy of Sleep Medicine (AASM) practice guidelines and position papers have addressed the diagnostic and clinical indications for polysomnography (PSG) with and without positive airway pressure (PAP) titration and home sleep apnea tests (HSATs), as well as specific management issues in sleep-disordered breathing in both adults and children/adolescents.^{1–10} However, there is a gap in AASM guidance for clinicians on the use of follow-up PSG or HSAT, as part of the longitudinal management of patients with OSA.

The AASM convened a task force of five sleep medicine experts to develop a clinical guidance statement on the use of PSG and HSATs in the longitudinal management of OSA. Additional details outlining the methodology used can be found in the supplemental material. This paper was based upon a literature review

and expert opinion, with clinical guidance statements for follow-up sleep testing predicated on the assumption that repeat testing may prompt or guide a change in disease management. Judgment regarding the appropriateness of using PSG and HSATs remains the treating clinician's responsibility, due to their knowledge of their patients' individual circumstances and available resources.

BACKGROUND

The most recent clinical practice guideline on diagnostic testing for OSA in adults addressed the initial indications for sleep apnea testing but not for follow-up testing.⁵ This clinical guidance statement focuses on indications for follow-up sleep apnea testing in patients with OSA with or without sleep-related hypoventilation or hypoxemia. It does not pertain to instances where there is suspicion for an additional clinical sleep disorder other than OSA.

A follow-up sleep apnea study is defined as PSG or an HSAT performed after an initial diagnostic test. It is important to

identify patients who may benefit from follow-up sleep apnea testing, which may impact clinically meaningful outcomes such as sleepiness, sleep-related quality of life, and therapy adherence. A comprehensive history and physical examination may provide clues for the need to consider repeat sleep apnea testing. Since OSA has a high prevalence and is a chronic disease that needs to be monitored, it is important to know when follow-up sleep apnea testing is appropriate and should be utilized. The decision to use PSG vs HSAT should be determined by accessibility, financial feasibility and the specific clinical situation (eg, decompensated heart failure).⁵

Because of limitations in the specificity of HSAT to rule out OSA, a negative HSAT should be followed by in-lab PSG if clinical concerns for OSA remain (eg, if sleep-related symptoms persist and the HSAT does not identify OSA).

CLINICAL GUIDANCE STATEMENTS

The AASM supports the following clinical guidance statements on indications for follow-up PSG and HSAT in adult patients with OSA. These guidance statements apply to the use of PSG and HSATs after a diagnosis of OSA has been established and decisions about treatment implementation have been made. The guidance statements do not apply to the initiation of treatment (eg, initial titration of PAP devices); guidance for the use of diagnostic testing devices during the initiation of treatment for OSA can be found in published guidelines on the use of these treatments.^{8,9,11,12} A summary of the clinical guidance statements is available in [Table 1](#).

For the purposes of this clinical guidance statement, statements using “recommended” and “not recommended” indicate that a test is clearly useful or ineffective/harmful for most patients, respectively, based on a qualitative assessment of the available evidence and clinical judgement of the task force. Statements using “may be used” indicate that the evidence or expert consensus is less clear, either in favor or against the use of a testing option.

Clinical guidance statement 1: Follow-up PSG or HSAT is *not* recommended for routine reassessment of asymptomatic patients with obstructive sleep apnea on PAP therapy, however, follow-up PSG or HSAT can be used to reassess patients with recurrent or persistent symptoms, despite good PAP adherence

There is insufficient evidence to suggest that routine PSG and/or HSAT should be performed in patients who are responding

adequately to PAP treatment as determined by symptom control and adherence to therapy.^{8,12,13} If symptoms return despite good adherence to PAP treatment, PSG or HSAT is appropriate. If there is an unexplained change in adherence, or there is clinical suspicion of a separate sleep disorder, then a repeat PSG may be considered, and might include PAP titration.³ If there is suspicion of another sleep disorder such as narcolepsy, then PSG should precede other testing, such as a multiple sleep latency test.

Clinical guidance statement 2: Follow-up PSG or HSAT is recommended to assess response to treatment with non-PAP interventions

PAP therapy remains the first-line therapy for most adults with OSA.³ For patients who are intolerant to PAP or who prefer alternative therapies, several non-PAP treatments for OSA are available, including oral appliance therapy, nasal expiratory positive airway pressure, upper airway surgery, positional therapy, oral pressure therapy, weight loss and hypoglossal nerve stimulation. Following implementation of non-PAP interventions for OSA, clinical reassessment should include follow-up testing to ensure successful treatment of OSA with the chosen treatment at the appropriate time interval. Follow-up testing is not necessary for positional therapy if it was proven effective on the diagnostic study. As to the type of test, PSG vs HSAT, multiple factors should be considered, including comorbidities (eg, hypoventilation, advanced cardiopulmonary disease), patient comfort, ability to wear/operate the HSAT equipment, cost of testing, and resource utilization.

Oral appliances require fitting by a dental sleep specialist, and serial advancement of the mandible to promote airway opening.^{14–16} Follow-up PSG or HSAT should be performed following patient acclimatization, reviewed by a trained sleep medicine specialist and necessary adjustments should be made to the device to ensure adequate control of OSA.¹⁵ In the AASM’s 2015 oral appliance clinical practice guideline, the recommendation to “...conduct follow-up sleep testing to improve or confirm treatment efficacy, rather than conduct follow-up without sleep testing, for patients fitted with oral appliances.” was assigned a “guideline” strength.⁹ Recommendations on timing of testing or the type of test (PSG vs HSAT) were not made and evidence was insufficient to include in a meta-analysis. Evidence is also lacking to guide the frequency with

Table 1—Summary of clinical guidance statements.

| |
|--|
| 1. Follow-up PSG or HSAT is <i>not</i> recommended for routine reassessment of asymptomatic patients with obstructive sleep apnea on PAP therapy, however, follow-up PSG or HSAT can be used to reassess patients with recurrent or persistent symptoms, despite good PAP adherence. |
| 2. Follow-up PSG or HSAT is recommended to assess response to treatment with non-PAP interventions. |
| 3. Follow-up PSG or HSAT may be used if clinically significant weight gain or loss has occurred since diagnosis of OSA or initiation of treatment. |
| 4. Follow-up PSG may be used for reassessment of sleep-related hypoxemia and/or sleep-related hypoventilation following initiation of treatment for OSA. |
| 5. Follow-up PSG or HSAT may be used in patients being treated for OSA who develop or have a change in cardiovascular disease. |
| 6. Follow-up PSG may be used in patients with unexplained PAP device-generated data. |

HSAT = home sleep apnea test, OSA = obstructive sleep apnea, PAP = positive airway pressure, PSG = polysomnography.

which follow-up testing should be performed. For those using an oral appliance, a reasonable juncture to retest would be the point at which mandibular protrusion and symptom mitigation are felt to be clinically optimized by a qualified dentist and/or sleep medicine provider. On occasion, additional (serial) testing may be needed to ensure adequate treatment. Even though overnight pulse oximetry (a type 4 device) is utilized to measure oxyhemoglobin saturation, it is not recommended for the diagnosis of OSA, and therefore is not considered an acceptable alternative to PSG or HSAT for follow-up testing of patients with OSA. Follow-up testing should be restricted to methods with sufficient evidence to support use of that method for diagnostic purposes, that is PSG and multichannel HSAT.

A variety of upper airway surgeries are performed as an alternative to PAP in the management of OSA, including uvulopalatopharyngoplasty, maxillomandibular advancement, hyoid suspension, genioglossus advancement and adenotonsillectomy; these procedures and their approaches and outcomes have been reviewed and summarized elsewhere.¹⁷ Following the surgical procedure and an appropriate period of healing (weeks to months), PSG or HSAT should be used to evaluate treatment efficacy. Patients should be assessed clinically on a periodic basis, since OSA may recur postoperatively.^{18,19} Following hypoglossal nerve stimulation surgery and activation, the hypoglossal nerve stimulation system requires PSG titration of stimulation parameters to optimize treatment outcomes for OSA.²⁰ For patients with a tracheostomy for OSA who are under consideration for decannulation, PSG or HSAT with the trach capped may be useful.²¹

Clinical guidance statement 3: Follow-up PSG or HSAT may be used if clinically significant weight gain or loss has occurred since diagnosis of OSA or initiation of treatment

Remark: Auto-adjusting PAP, which is considered to be therapeutically equivalent to fixed CPAP in uncomplicated OSA,⁸ may obviate the need for repeat testing in certain circumstances of weight change.

There is an incontrovertible relationship between body weight and OSA which is mediated by influences on pharyngeal airway size and flow dynamics.²² These findings highlight the potential need for repeat sleep apnea testing when there are significant changes in weight. Data from the Sleep Heart Health Study suggest a 10 to 20% increase in body weight results in a significant rise in AHI.²³ When the patient treated for OSA gains weight, resulting in inadequate therapy of OSA, there might be re-emergence of symptoms such as daytime sleepiness or interrupted sleep or signs such as snoring. Such occurrences might represent reasons for repeat testing. Options include PSG with PAP titration or HSAT to evaluate for ongoing OSA followed by empiric CPAP adjustment. Some practitioners may elect to forego testing in this setting and instead empirically increase CPAP pressure and reassess for resolution of findings.

The task force evaluated the literature on surgical and nonsurgical weight loss interventions and their impact on OSA, as measured by follow-up sleep apnea testing. Thirty-two articles were reviewed; twenty-three described nonsurgical

interventions and nine articles evaluated various forms of bariatric surgery. Nonsurgical interventions included medication,²⁴ cognitive behavioral therapy (CBT),²⁵ dietary changes,^{22,26–37} high intensity aerobic and resistance exercise,^{32,38–42} as well as lifestyle modification.^{43,44} Interventions for the nine surgical studies predominantly included gastric lap band surgery and gastric bypass to include modified Roux-en-Y surgery.^{19,38,43–49} None of the articles were designed to specifically address whether follow-up testing impacted outcomes in patients with OSA and there were no studies that compared PSG with HSAT. In addition, no articles specifically addressed when follow-up testing should be performed. However, based upon review of clinical trials that performed repeat testing at predefined intervals, we recommend that follow-up PSG or HSAT can be considered after a 10 to 20% reduction or increase in weight, and in the case of surgery, after at least 3 months of recovery. It is implied that the decision to repeat PSG or HSAT would lead to some change in management, such as discontinuation of OSA treatment, recognizing that empiric adjustments in therapy (such as small, 1–2 cm water, PAP pressure reductions) can occur in the absence of testing. Ongoing clinical evaluation, which can include follow-up sleep apnea testing, is ultimately important, since none of the analyzed studies revealed complete correction of the apnea-hypopnea index (AHI) or respiratory event index (REI) (AHI/REI < 5 events/h) after bariatric surgery.^{19,38,43–49}

Clinical guidance statement 4: Follow-up PSG may be used for reassessment of sleep-related hypoxemia and/or sleep-related hypoventilation following initiation of treatment for OSA

Physiologic mechanisms resulting in hypoxemia while awake or asleep include residence at high-altitude (or other causes of low fraction of inspired oxygen, FiO₂), ventilation-perfusion mismatch, pulmonary diffusion abnormality, shunting and hypoventilation.² The most commonly associated conditions found in the sleep center include ventilation-perfusion mismatch (resulting in hypoxemia with/or without hypercapnia) and hypoventilation (resulting in hypercapnia with/or without hypoxemia). Some patients with OSA, particularly those with underlying cardiopulmonary disease, may have hypoxemia or hypoventilation that persists or develops following initial implementation of treatment such as PAP therapy. Such developments may be insidious and unaccompanied by symptoms that might otherwise bring a patient to clinical attention for intervention. For this reason, and because there may be long-term consequences such as pulmonary hypertension or heart failure, it is important to identify these derangements with follow-up testing and appropriate treatment changes.

Our literature review did not identify any articles that addressed when follow-up testing should be performed to assess sleep-related hypoxemia or hypoventilation while on PAP therapy. We believe that PSG can be useful as a follow-up test, such as when precise treatment titration is needed (eg, the addition of supplemental oxygen or a respiratory assist device such as bilevel PAP). Repeat titration PSG could be performed when advanced treatment modalities are being considered (eg,

volume adjusted pressure support).⁵⁰ Adjunctive diagnostic tools that may be useful to detect hypoventilation during PSG include arterial blood gas analysis, end-tidal carbon dioxide or transcutaneous carbon dioxide monitoring. Despite these potential benefits, depending upon the clinical circumstance, the decision of whether to pursue repeat PSG should consider the clinical circumstance, resource utilization, and likelihood that the resultant information will lead to a change in management.

An AASM guideline published in 2007 found insufficient evidence to support the use of type 4 portable monitors (pulse oximetry) to make a diagnosis of OSA.⁵¹ However, in those already diagnosed with OSA and on PAP therapy, overnight pulse oximetry is sometimes used to detect sleep-related hypoxemia in the setting of cardiopulmonary or neuromuscular disease. Overnight pulse oximetry, performed for a clinical suspicion of nocturnal hypoxemia or hypoventilation in those being treated for OSA, has not been adequately studied. While some practitioners may utilize this tool in this particular setting, interpretation of results from overnight oximetry is not standardized. Therefore, PSG is still the diagnostic test of choice to determine if another sleep-related breathing disorder exists. Although the interpretation of overnight pulse oximetry is not standardized, some patterned abnormalities (a sawtooth pattern of oxyhemoglobin desaturation⁵¹ or persistent hypoxemia in the setting of chronic obstructive pulmonary disease⁵¹) can identify patients who might benefit from subsequent PSG and/or treatment adjustment. In patients on long-term supplemental oxygen, overnight pulse oximetry itself might guide empiric changes in prescribed flow rates.

With technology designed to detect respiratory events associated with acute changes in variables such as nasal airflow, chest wall excursion, or arterial tone, HSAT is not ideally suited to detect derangements in gas exchange outlined above that result in hypoxemia and/or hypoventilation. However, because many devices are also equipped with a pulse oximeter and can determine postural changes, HSAT might be useful to identify abnormalities to prompt subsequent PSG and change in management. For example, persistent sleep-related hypoxemia associated with a hypopnea-predominant pattern on HSAT may indicate the presence of sleep-related hypoventilation.⁵² Discrete intervals of hypoxemia may suggest the presence of hypoventilation during periods of REM-related skeletal muscle hypotonia.⁵³

Clinical guidance statement 5: Follow-up PSG or HSAT may be used in patients being treated for OSA who develop or have a change in cardiovascular disease

Various cardiovascular (CV) diseases are strongly linked to OSA, with increased risk of conditions such as systemic hypertension, heart failure (HF), cardiac arrhythmias, stroke and sudden death.⁵⁴ Many patients are referred for evaluation and treatment of OSA to potentially improve CV outcomes. For initial diagnosis of suspected sleep apnea, current guidelines strongly recommend attended PSG over HSAT for patients with HF and/or history of stroke, since such patients are at risk for nonobstructive sleep-disordered breathing (central sleep apnea, sleep-related hypoxemia). For CV conditions other than HF and

stroke, where the risk of nonobstructive sleep apnea is less, either PSG or HSAT is recommended.⁵

There is ample evidence to implicate OSA as one of the direct causes of systemic hypertension. However, when considering other CV disorders such as HF and cardiac arrhythmias, despite strong associations, the causal role of OSA remains to be definitively proven, in part because interventional trials either have not been conducted or have not shown improvements in CV outcomes.⁵⁵ With these limitations in mind, clinical guidance statements for follow-up sleep apnea testing are predicated on the assumption that repeat testing will prompt or guide a change in disease management, either of OSA or of the underlying CV disease(s). Our literature search found no direct evidence to guide if and when follow-up testing should be performed in the population with CV disease, but it could be considered in certain clinical situations:

- Systemic hypertension: Development of the disorder, or worsening control of blood pressure while on OSA therapy, might prompt follow-up testing and reassessment of therapy of OSA. Based upon interpretation of data from randomized clinical trials^{56,57} measuring blood pressure before and after PAP treatment, we recommend that repeat sleep testing not occur before at least three months of adherent PAP usage. Furthermore, in terms of blood pressure control, it should be noted that short-term head-to-head trials show antihypertensive drugs may be more effective at reducing blood pressure than PAP.⁵⁸ How to weigh the cost and benefits of changes in OSA therapy against implementing or adjusting antihypertensive drug treatment to manage hypertension is not known.
- Heart failure: Evidence of decompensated heart failure may be an indication for repeat testing, though some caveats apply. First, decompensated HF can congest the soft tissues of the upper airway and exacerbate OSA. Second, the initial management of congestion due to an HF exacerbation should be optimization of medical therapy (such as diuresis), which could be enforced before sleep apnea testing, and which itself will probably improve upper airway mechanics. Sleep-related breathing abnormalities in this setting can be central sleep apnea/Cheyne-Stokes breathing, which also should be initially managed medically, and for which PAP device management is not standardized.⁵⁹
- Cardiac arrhythmias: Newly discovered arrhythmias, such as atrial fibrillation or bradycardias such as atrioventricular block, would be reasonable indications for retesting and reassessment of OSA therapy, particularly if the arrhythmias occur during sleep. If cardiac monitoring is desired, in-lab testing with a dedicated electrocardiogram channel is preferred. It should be recognized that no prospective clinical trials have proven a role for PAP therapy in improving rhythm outcomes.⁶⁰
- Stroke: A stroke or transient ischemic attack that occurs in a patient treated with PAP therapy for OSA should prompt follow-up evaluation and possible sleep apnea testing to ensure adequate control of sleep apnea and/or to rule out its presence, recognizing that patients are at risk for both obstructive and central sleep apnea.

Clinical guidance statement 6: Follow-up PSG may be used in patients with unexplained PAP device-generated data

Monitoring automated event detection (AED) has become common practice in the management of patients with OSA treated with PAP devices. Event data can be downloaded by smartcard or displayed on the internet for viewing by patients, durable medical equipment companies, and practitioners.^{61–65} Some studies have shown a correlation between PAP-scored AHI and manual-scored AHI.^{66–69} An AED obtained from a PAP device may be considered in the decision to perform follow-up PSG or HSAT, though it is an important caveat that such data are derived from proprietary software, and their validity has been questioned.^{3,12}

The task force review did not identify any articles measuring outcomes from the use of AED to determine when to perform follow-up PSG or HSAT. In addition to allowing for follow-up of treatment adherence and effectiveness, PAP device software downloads may reveal reasons for suboptimal therapy (such as interface leak).⁷⁰ Once minimum adherence has been confirmed and interface issues have been optimized, follow-up PSG may be indicated based on the AED when PAP device software residual AHI remains elevated, particularly in the face of sleep symptoms. It is likely that many clinicians will empirically adjust machine pressure settings based upon the AED, which can obviate the need for testing in some cases. The threshold for repeat testing is a matter of debate and is contextual. For example, a mildly elevated residual AHI (5 to 15) in a patient with re-emergence of sleep apnea symptoms warrants either empiric intervention or repeat testing. However, an elevated residual AHI in the absence of signs or symptoms of under-treated OSA is of unknown significance and may not represent an obvious indication for follow-up testing.

Notwithstanding controversies surrounding the significance of treatment-emergent central sleep apnea, evidence for treatment-emergent central sleep apnea on PAP download (a high central apnea index) could represent an indication for repeat testing. Since treatment-emergent central sleep apnea dissipates over time in a substantial proportion of patients,⁷¹ we recommend a minimum of three months of PAP therapy prior to repeat testing. Whether such testing can or should result in a change in treatment management to alter outcomes has not been proven.

FUTURE DIRECTIONS

There was no direct evidence found from the literature reviewed to inform best practice on follow-up sleep apnea testing. Indirect evidence was derived from a handful of studies, such as those that incorporated follow-up sleep apnea testing as part of a protocolized clinical trial which also measured outcomes of interest relevant to our clinical guidance statements.

There are several areas, therefore, where future research may be useful:

- Studies designed to enhance HSAT specificity to reliably rule out OSA among patients receiving treatment.
- Comparison of clinician global assessment, HSAT and PSG as reliable tools to evaluate changes in symptoms and/or outcomes in patients receiving therapy.

- Establish reliability and utility of HSAT in patients with cardiopulmonary or neuromuscular disease.
- Determine the optimal timing of follow-up sleep apnea testing after surgical modifications of the upper airway for OSA.
- Identify the ideal timing for follow-up testing in patients with OSA who have experienced a change in weight, both by medical/dietary means, and following bariatric surgery.
- Explore the utility of pulse oximetry as a tool for follow-up of patients with OSA, including those with cardiopulmonary disease.
- Comparative studies of outcomes between empiric adjustments of OSA therapy based upon clinical assessment or PAP machine AED with interventions based upon follow-up testing.
- Collaboration with partners in industry to broadly validate and standardize algorithms and adopt common language and terminology for events on PAP downloads.
- Increase understanding of whether treatment-emergent central sleep apnea encountered on machine AED or on follow-up testing is significant and warrants intervention.

REFERENCES

1. Aurora RN, Chowdhuri S, Ramar K, et al. The treatment of central sleep apnea syndromes in adults: practice parameters with an evidence-based literature review and meta-analyses. *Sleep*. 2012;35(1):17–40.
2. Berry RB, Chediak A, Brown LK, et al. Best clinical practices for the sleep center adjustment of noninvasive positive pressure ventilation (NPPV) in stable chronic alveolar hypoventilation syndromes. *J Clin Sleep Med*. 2010;6(5):491–509.
3. Epstein LJ, Kristo D, Strollo PJ Jr, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *J Clin Sleep Med*. 2009;5(3):263–276.
4. Gurubhagavatula I, Sullivan S, Meoli A, et al. Management of obstructive sleep apnea in commercial motor vehicle operators: recommendations of the AASM sleep and transportation safety awareness task force. *J Clin Sleep Med*. 2017;13(5):745–758.
5. Kapur VK, Auckley DH, Chowdhuri S, et al. Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: an American Academy of Sleep Medicine clinical practice guideline. *J Clin Sleep Med*. 2017;13(3):479–504.
6. Kirk V, Baughn J, D'Andrea L, et al. American Academy of Sleep Medicine position paper for the use of a home sleep apnea test for the diagnosis of OSA in children. *J Clin Sleep Med*. 2017;13(10):1199–1203.
7. Kushida CA, Chediak A, Berry RB, et al. Clinical guidelines for the manual titration of positive airway pressure in patients with obstructive sleep apnea. *J Clin Sleep Med*. 2008;4(2):157–171.
8. Patil SP, Ayappa IA, Caples SM, Kimoff RJ, Patel SR, Harrod CG. Treatment of adult obstructive sleep apnea with positive airway pressure: an American Academy of Sleep Medicine clinical practice guideline. *J Clin Sleep Med*. 2019;15(2):335–343.
9. Ramar K, Dort LC, Katz SG, et al. Clinical practice guideline for the treatment of obstructive sleep apnea and snoring with oral appliance therapy: an update for 2015. *J Clin Sleep Med*. 2015;11(7):773–827.
10. Rosen IM, Kirsch DB, Chervin RD, et al. Clinical use of a home sleep apnea test: an American Academy of Sleep Medicine position statement. *J Clin Sleep Med*. 2017;13(10):1205–1207.
11. Aurora RN, Zak RS, Karipott A, et al. Practice parameters for the respiratory indications for polysomnography in children. *Sleep*. 2011;34(3):379–388.
12. Morgenthaler TI, Aurora RN, Brown T, et al. Practice parameters for the use of autotitrating continuous positive airway pressure devices for titrating pressures and treating adult patients with obstructive sleep apnea syndrome: an update for 2007. An American Academy of Sleep Medicine report. *Sleep*. 2008;31(1):141–147.

13. Sawyer AM, Gooneratne NS, Marcus CL, Ofer D, Richards KC, Weaver TE. A systematic review of CPAP adherence across age groups: clinical and empiric insights for developing CPAP adherence interventions. *Sleep Med Rev*. 2011; 15(6):343–356.
14. Marklund M. Long-term efficacy of an oral appliance in early treated patients with obstructive sleep apnea. *Sleep Breath*. 2016;20(2):689–694.
15. Palotie T, Riekkki S, Mäkitie A, Bachour A, Arte S, Bäck L. The effect of mandible advancement splints in mild, moderate, and severe obstructive sleep apnea—the need for sleep registrations during follow up. *Eur J Orthod*. 2017;39(5):497–501.
16. Takaesu Y, Tsuiki S, Kobayashi M, Komada Y, Nakayama H, Inoue Y. Mandibular advancement device as a comparable treatment to nasal continuous positive airway pressure for positional obstructive sleep apnea. *J Clin Sleep Med*. 2016;12(8):1113–1119.
17. Caples SM, Rowley JA, Prinsell JR, Pallanch JF, Elamin MB, Katz SG, Harwick JD. Surgical modifications of the upper airway for obstructive sleep apnea in adults: a systematic review and meta-analysis. *Sleep*. 2010;33(10):1396–1407.
18. Amin R, Anthony L, Somers V, et al. Growth velocity predicts recurrence of sleep-disordered breathing 1 year after adenotonsillectomy. *Am J Respir Crit Care Med*. 2008;177(6):654–659.
19. Feigel-Guiller B, Drui D, Dimet J, et al. Laparoscopic gastric banding in obese patients with sleep apnea: a 3-year controlled study and follow-up after 10 years. *Obes Surg*. 2015;25(10):1886–1892.
20. Strollo PJ Jr, Soose RJ, Maurer JT, et al. Upper-airway stimulation for obstructive sleep apnea. *N Engl J Med*. 2014;370(2):139–149.
21. Tunkel DE, McColley SA, Baroody FM, Marcus CL, Carroll JL, Loughlin GM. Polysomnography in the evaluation of readiness for decannulation in children. *Arch Otolaryngol Head Neck Surg*. 1996;122(7):721–724.
22. Pahkala R, Seppä J, Ikonen A, Smirnov G, Tuomilehto H. The impact of pharyngeal fat tissue on the pathogenesis of obstructive sleep apnea. *Sleep Breath*. 2014;18(2):275–282.
23. Peppard PE, Young T, Palta M, Dempsey J, Skatrud J. Longitudinal study of moderate weight change and sleep-disordered breathing. *JAMA*. 2000;284(23):3015–3021.
24. Ferland A, Poirier P, Sériès F. Sibutramine versus continuous positive airway pressure in obese obstructive sleep apnoea patients. *Eur Respir J*. 2009;34(3):694–701.
25. Kajaste S, Brander PE, Telakivi T, Partinen M, Mustajoki P. A cognitive-behavioral weight reduction program in the treatment of obstructive sleep apnea syndrome with or without initial nasal CPAP: a randomized study. *Sleep Med*. 2004;5(2):125–131.
26. Foster GD, Borradaile KE, Sanders MH, et al. A randomized study on the effect of weight loss on obstructive sleep apnea among obese patients with type 2 diabetes: the Sleep AHEAD study. *Arch Intern Med*. 2009;169(17):1619–1626.
27. Gilardini L, Lombardi C, Redaelli G, et al. Glucose tolerance and weight loss in obese women with obstructive sleep apnea. *PLoS One*. 2013;8(4):e61382.
28. Johansson K, Neovius M, Lagerros YT, Harlid R, Rössner S, Granath F, Hemmingsson E. Effect of a very low energy diet on moderate and severe obstructive sleep apnoea in obese men: a randomised controlled trial. *BMJ*. 2009; 339:b4609.
29. Kempainen T, Ruoppi P, Seppä J, et al. Effect of weight reduction on rhinometric measurements in overweight patients with obstructive sleep apnea. *Am J Rhinol*. 2008;22(4):410–415.
30. Kline CE, Reboussin DM, Foster GD, et al. The effect of changes in cardiorespiratory fitness and weight on obstructive sleep apnea severity in overweight adults with type 2 diabetes. *Sleep*. 2016;39(2):317–325.
31. Kuna ST, Reboussin DM, Borradaile KE, et al. Long-term effect of weight loss on obstructive sleep apnea severity in obese patients with type 2 diabetes. *Sleep*. 2013;36(5):641–649.
32. Maki-Nunes C, Toschi-Dias E, Cepeda FX, et al. Diet and exercise improve chemoreflex sensitivity in patients with metabolic syndrome and obstructive sleep apnea. *Obesity (Silver Spring)*. 2015;23(8):1582–1590.
33. Papandreou C, Hatzis CM, Fragkiadakis GA. Effects of different weight loss percentages on moderate to severe obstructive sleep apnoea syndrome. *Chron Respir Dis*. 2015;12(3):276–278.
34. Shechter A, St-Onge MP, Kuna ST, et al. Sleep architecture following a weight loss intervention in overweight and obese patients with obstructive sleep apnea and type 2 diabetes: relationship to apnea-hypopnea index. *J Clin Sleep Med*. 2014;10(11):1205–1211.
35. Tuomilehto H, Seppä J, Uusitupa M, et al. The impact of weight reduction in the prevention of the progression of obstructive sleep apnea: an explanatory analysis of a 5-year observational follow-up trial. *Sleep Med*. 2014;15(3):329–335.
36. Tuomilehto H, Seppä J, Uusitupa M, Tuomilehto J, Gylling H. Weight reduction and increased physical activity to prevent the progression of obstructive sleep apnea: A 4-year observational postintervention follow-up of a randomized clinical trial. *JAMA Intern Med*. 2013;173(10):929–930.
37. Tuomilehto HP, Seppä JM, Partinen MM, et al. Lifestyle intervention with weight reduction: first-line treatment in mild obstructive sleep apnea. *Am J Respir Crit Care Med*. 2009;179(4):320–327.
38. Dixon JB, Schachter LM, O'Brien PE, et al. Surgical vs conventional therapy for weight loss treatment of obstructive sleep apnea: a randomized controlled trial. *JAMA*. 2012;308(11):1142–1149.
39. Kline CE, Crowley EP, Ewing GB, et al. The effect of exercise training on obstructive sleep apnea and sleep quality: a randomized controlled trial. *Sleep*. 2011;34(12):1631–1640.
40. Kline CE, Ewing GB, Burch JB, et al. Exercise training improves selected aspects of daytime functioning in adults with obstructive sleep apnea. *J Clin Sleep Med*. 2012;8(4):357–365.
41. Norman JF, Von Essen SG, Fuchs RH, McElligott M. Exercise training effect on obstructive sleep apnea syndrome. *Sleep Res Online*. 2000;3(3):121–129.
42. Schütz TC, Cunha TC, Moura-Guimaraes T, et al. Comparison of the effects of continuous positive airway pressure, oral appliance and exercise training in obstructive sleep apnea syndrome. *Clinics (São Paulo)*. 2013;68(8):1168–1174.
43. Ng SSS, Chan RSM, Woo J, et al. A randomized controlled study to examine the effect of a lifestyle modification program in OSA. *Chest*. 2015;148(5):1193–1203.
44. Sahlman J, Seppä J, Herder C, et al. Effect of weight loss on inflammation in patients with mild obstructive sleep apnea. *Nutr Metab Cardiovasc Dis*. 2012;22(7):583–590.
45. Bakker JP, Campana LM, Montesi SB, et al. A pilot study investigating the effects of continuous positive airway pressure treatment and weight-loss surgery on autonomic activity in obese obstructive sleep apnea patients. *J Electrocardiol*. 2014;47(3):364–373.
46. de Assunção Machado AC, da Silva AMV, Signori LU, da Costa Alvarez G, Mottin CC. Endothelial function of patients with morbid obesity submitted to roux-en-y gastric bypass with and without obstructive sleep apnea-hypopnea syndrome. *Obes Surg*. 2018;28(11):3595–3603.
47. Gabrielsen AM, Omland T, Brokner M, et al. The effect of surgical and non-surgical weight loss on N-terminal pro-B-type natriuretic peptide and its relation to obstructive sleep apnea and pulmonary function. *BMC Res Notes*. 2016;9(1):440.
48. Kennedy-Dalby A, Adam S, Ammori BJ, Syed AA. Weight loss and metabolic outcomes of bariatric surgery in men versus women - A matched comparative observational cohort study. *Eur J Intern Med*. 2014;25(10):922–925.
49. Zou J, Zhang P, Yu H, Di J, Han X, Yin S, Yi H. Effect of laparoscopic roux-en-y gastric bypass surgery on obstructive sleep apnea in a chinese population with obesity and T2DM. *Obes Surg*. 2015;25(8):1446–1453.
50. Orlikowski D, Prigent H, Quera Salva MA, et al. Prognostic value of nocturnal hypoventilation in neuromuscular patients. *Neuromuscul Disord*. 2017;27(4):326–330.
51. Collop NA, Anderson WM, Boehlecke B, et al. Clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients. *J Clin Sleep Med*. 2007;3(7):737–747.
52. Mathew R, Castriotta RJ. High hypopnea/apnea ratio (HAR) in extreme obesity. *J Clin Sleep Med*. 2014;10(4):391–396.
53. Fletcher EC, Gray BA, Levin DC. Nonapneic mechanisms of arterial oxygen desaturation during rapid-eye-movement sleep. *J Appl Physiol*. 1983;54(3):632–639.
54. Peppard PE, Young T, Barnett JH, Palta M, Hagen EW, Hla KM. Increased prevalence of sleep-disordered breathing in adults. *Am J Epidemiol*. 2013;177(9):1006–1014.

55. McEvoy RD, Antic NA, Heeley E, et al. CPAP for prevention of cardiovascular events in obstructive sleep apnea. *N Engl J Med*. 2016;375(10):919–931.
56. Drager LF, Lopes HF, Maki-Nunes C, et al. The impact of obstructive sleep apnea on metabolic and inflammatory markers in consecutive patients with metabolic syndrome. *PLoS One*. 2010;5(8):e12065.
57. Durán-Cantolla J, Aizpuru F, Montserrat JM, et al. Continuous positive airway pressure as treatment for systemic hypertension in people with obstructive sleep apnoea: randomised controlled trial. *BMJ*. 2010;341:c5991.
58. Pépin JL, Tamisier R, Barone-Rochette G, Launois SH, Lévy P, Baguet JP. Comparison of continuous positive airway pressure and valsartan in hypertensive patients with sleep apnea. *Am J Respir Crit Care Med*. 2010;182(7):954–960.
59. Cowie MR, Woehrle H, Wegscheider K, et al. Adaptive servo-ventilation for central sleep apnea in systolic heart failure. *N Engl J Med*. 2015;373(12):1095–1105.
60. Caples SM, Mansukhani MP, Friedman PA, Somers VK. The impact of continuous positive airway pressure treatment on the recurrence of atrial fibrillation post cardioversion: A randomized controlled trial. *Int J Cardiol*. 2019;278:133–136.
61. Gagnadoux F, Pevernagie D, Jennum P, et al. Validation of the system one RemStar auto a-flex for obstructive sleep apnea treatment and detection of residual apnea-hypopnea index: a European randomized trial. *J Clin Sleep Med*. 2017;13(2):283–290.
62. Kuna ST, Shuttleworth D, Chi L, et al. Web-based access to positive airway pressure usage with or without an initial financial incentive improves treatment use in patients with obstructive sleep apnea. *Sleep*. 2015;38(8):1229–1236.
63. Light M, Orr JE, Malhotra A, Owens RL. Continuous positive airway pressure device detects atrial fibrillation induced central sleep apnoea. *Lancet*. 2018;392(10142):160.
64. Reiter J, Zleik B, Bazalakova M, Mehta P, Thomas RJ. Residual events during use of CPAP: prevalence, predictors, and detection accuracy. *J Clin Sleep Med*. 2016;12(8):1153–1158.
65. Ueno K, Kasai T, Brewer G, et al. Evaluation of the apnea-hypopnea index determined by the S8 auto-CPAP, a continuous positive airway pressure device, in patients with obstructive sleep apnea-hypopnea syndrome. *J Clin Sleep Med*. 2010;6(2):146–151.
66. Berry RB, Kushida CA, Kryger MH, Soto-Calderon H, Staley B, Kuna ST. Respiratory event detection by a positive airway pressure device. *Sleep*. 2012;35(3):361–367.
67. Denotti AL, Wong KK, Dungan GC 2nd, Gilholme JW, Marshall NS, Grunstein RR. Residual sleep-disordered breathing during autotitrating continuous positive airway pressure therapy. *Eur Respir J*. 2012;39(6):1391–1397.
68. Desai H, Patel A, Patel P, Grant BJ, Mador MJ. Accuracy of autotitrating CPAP to estimate the residual apnea-hypopnea index in patients with obstructive sleep apnea on treatment with autotitrating CPAP. *Sleep Breath*. 2009;13(4):383–390.
69. Stepnowsky C, Zamora T, Barker R, Liu L, Sarmiento K. Accuracy of positive airway pressure device-measured apneas and hypopneas: role in treatment followup. *Sleep Disord*. 2013;2013:314589.
70. Rühle KH, Domanski U, Franke KJ, Nilius G. [Studies of leakage measurements of automatic CPAP-devices]. *Pneumologie*. 2007;61(4):213–218.
71. Liu D, Armitstead J, Benjafeld A, et al. Trajectories of emergent central sleep apnea during CPAP therapy. *Chest*. 2017;152(4):751–760.

ACKNOWLEDGMENTS

The task force thanks and acknowledges the early contributions of Erik St. Louis, MD and Jonathan Heald, MA. Dr. St. Louis served as a member of the task force during the initial stages of development of this clinical guidance statement. Mr. Heald is former staff of the Science and Research department at the AASM.

SUBMISSION & CORRESPONDENCE INFORMATION

Submitted for publication February 26, 2021

Submitted in final revised form March 8, 2021

Accepted for publication March 8, 2021

Address correspondence to: Sean M. Caples, DO, MS, Sleep Disorders Center, Mayo Clinic, 200 First Street SW, Rochester, MN 55906; Email: caples.sean@mayo.edu

DISCLOSURE STATEMENT

The development of this clinical guidance statement was funded by the American Academy of Sleep Medicine. Ms. Hashmi is employed by the American Academy of Sleep Medicine. The other authors have indicated no financial conflicts of interest.