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CARDI•OH
Ohio Cardiovascular and Diabetes Health Collaborative

Recognizing Sleep Disorders to Manage Cardiovascular Disease Risk

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The International Classification of Sleep Disorders identifies seven categories of sleep disorders.[1] These seven categories include more than 60 specific disorder diagnoses.

This document will focus on chronic insomnia and obstructive sleep apnea (OSA), two common sleep disorders associated with elevated risk for cardiovascular disease (CVD). Although treatments for chronic insomnia and OSA are effective for symptom management and cardiovascular disease risk reduction, both disorders are underdiagnosed and undertreated.

Chronic Insomnia and CV Risk

The American Academy of Sleep Medicine's International Classification of Sleep Disorders defines chronic insomnia as a persistent difficulty with sleep initiation, duration, and/or quality at least 3 nights a week, lasting at least 3 months, leading to consequences/symptoms while awake despite an adequate opportunity for sleep.[1] Studies on the pathophysiology of insomnia and CV risk are confounded by inconsistencies in diagnostic criteria, particularly the inclusion of sleep duration in the definitions of insomnia.[2] Still, studies suggest that chronic insomnia with sleep duration of less than 6 hours (both of which are associated with increased CV risk) is associated with a two- to four-fold increase in rates of hypertension, 50% increase in rates of coronary heart disease, four- to five-fold increase in rates of heart failure, and 33–58% increase in cardiovascular mortality.[2-7] The American Heart Association (AHA) has recognized the importance of adequate sleep time by adding it as a *Life's Essential 8* for CV health.[8]

Pathophysiology

Adequate sleep time is essential for the body to recover from daytime activities. Without sufficient time in sleep or with recurrent sleep disruptions, the normal nocturnal blood pressure dip of 10–20% does not occur, which leads to nocturnal and overall hypertension, a known risk factor for atherosclerotic cardiovascular disease (ASCVD), congestive heart failure, and stroke.[8-10] Other mechanisms of disruption include dysregulation of the hypothalamic-pituitary axis; abnormal modulation of the autonomic nervous system; increased sympathetic nervous system activity, systemic inflammation, adrenocorticotropic hormone and cortisol secretion; and worsened glucose metabolism.[2] Sleep duration of less than 6 hours increases the risk of heart attack by 20%.[5,6] Diabetes mellitus, a known risk factor for ASCVD and stroke, is more prevelant in those with shortened or disrupted sleep cycles.[11]

Prevalence

Insomnia is the most common sleep disorder; about 30% of adults report short-term sleep disturbances and 10% report chronic insomnia.[12] It is more common in older adults, affecting up to 63% of people over the age of 60.[13] Other factors that may increase an individual's risk for chronic insomnia include being Black, female, divorced or separated, low socioeconomic status, shift work, family history of insomnia, genetic factors, and inactivity. Prevalence data in Hispanic populations show greater heterogeneity based on ancestral origin, but both Black and Hispanic populations have increasing prevalence of short sleep duration as social disadvantages increase.[14]

Table 1. Sleep Times in America

Sleep time	% American population
>9 hours	6%
7-9 hours	48%
6-7 hours	26%
<6 hours	20%

Adapted from National Sleep Foundation *2012 Bedroom Poll Summary of Findings*

For adults, the American Academy of Sleep Medicine recommends 7 or more hours of sleep in any 24-hour period, whereas the National Sleep Foundation recommends 7-9 hours.[15] Fifty-two percent of the American population reports a sleep duration that chronically falls outside this recommended sleep time (Table 1).[16]

Diagnosis

The diagnosis of chronic insomnia is made from patient-reported history and is aided by information from wearable devices; smart beds; sleep diaries; and/or sleepiness assessment tools, such as the Epworth Sleepiness Scale, RU SATED questionnaire, and the Pittsburgh Sleep Quality Index. Sleep testing via actigraphy or polysomnography is not part of routine screening for chronic insomnia.

Although no specific quantitative sleep parameters define insomnia, common complaints from insomnia patients include an average sleep latency of greater than 30 minutes, wake after sleep onset for more than 30 minutes, sleep efficiency of less than 85%, and/or total sleep time of less than 6.5 hours.[17]

The difficulty in initiating sleep should not be associated with a lack of opportunity to sleep or by another sleep disorder (e.g., obstructive sleep apnea or restless legs syndrome). If chronic insomnia is caused or affected by other conditions, such as cardiovascular disease, chronic pain disorders, cancer, and neurologic disorders, it is referred to as *comorbid insomnia*. If chronic insomnia is unaffected by a comorbid condition, it is termed *primary insomnia*.

Treatment

Treatment for impaired sleep duration and insomnia includes lifestyle management, cognitive behavioral therapy, and medication management, in order of priority (Figure 1). Treatment has been shown to reduce symptoms and CV effects,[1,17-21] including improvement of the normal nighttime blood pressure dip[18] and glucose control.[19]

Lifestyle. Lifestyle changes may have a marked effect on sleep hygiene and, therefore, symptoms and cardiovascular risk.

Whereas individual components of sleep hygiene have demonstrated benefits in studies, there is inconsistent evidence of sleep hygiene as a primary intervention for insomnia.[22]

Cognitive Behavioral Therapy (CBT). This therapy is 50-75% effective, at least as effective as medications, and considered the first-line treatment for chronic insomnia.[20-21] CBT-insomnia (CBT-i) emphasizes behavioral components, including sleep education, sleep restriction therapy, sleep compression, stimulus control, sleep hygiene, cognitive therapy, and counter-arousal measures. CBT-i is typically performed in four to eight one-on-one sessions with a trained mental health provider, but group-based and internet options are increasingly available. This treatment is covered by most insurance.

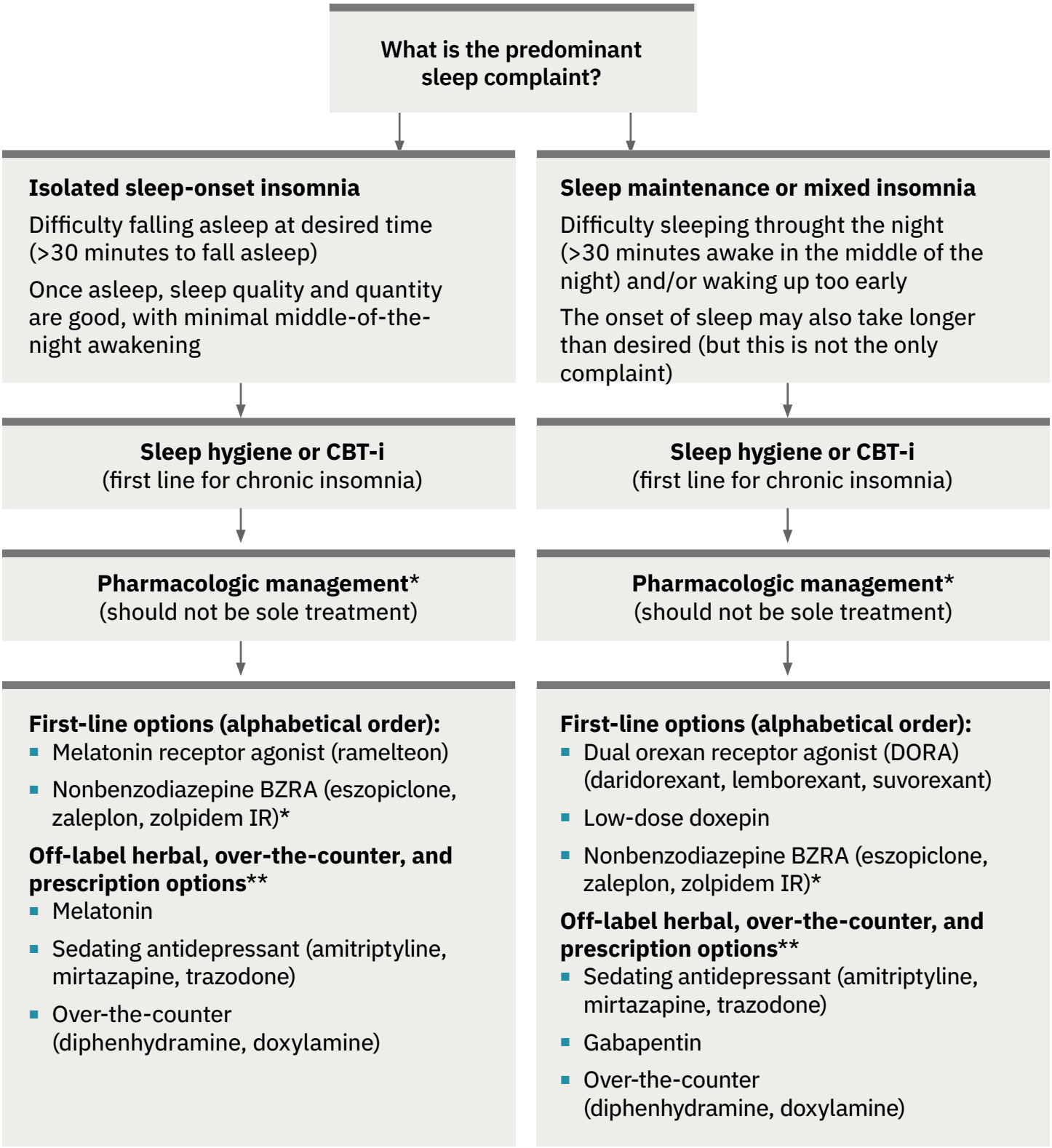
Medication Management. Medications for insomnia include dietary supplements, over-the-counter, and prescription options. Treatment depends on the type of sleep disruption and patient comorbidities.[23] Referral to a sleep specialist is appropriate if initial treatments are not successful.

Lifestyle

Cognitive Behavioral Therapy

Medication Management

Figure 1. Insomnia-Related Sleep Complaints and Treatment Options



*Desirable to avoid first-line benzodiazepine receptor agonists (BZRA)
**Use clinical judgement before recommending, considering such factors as limited efficacy and potential for adverse effects
Adapted from *Pharmacotherapy for insomnia in adults*[24]

Obstructive Sleep Apnea and CV Risk

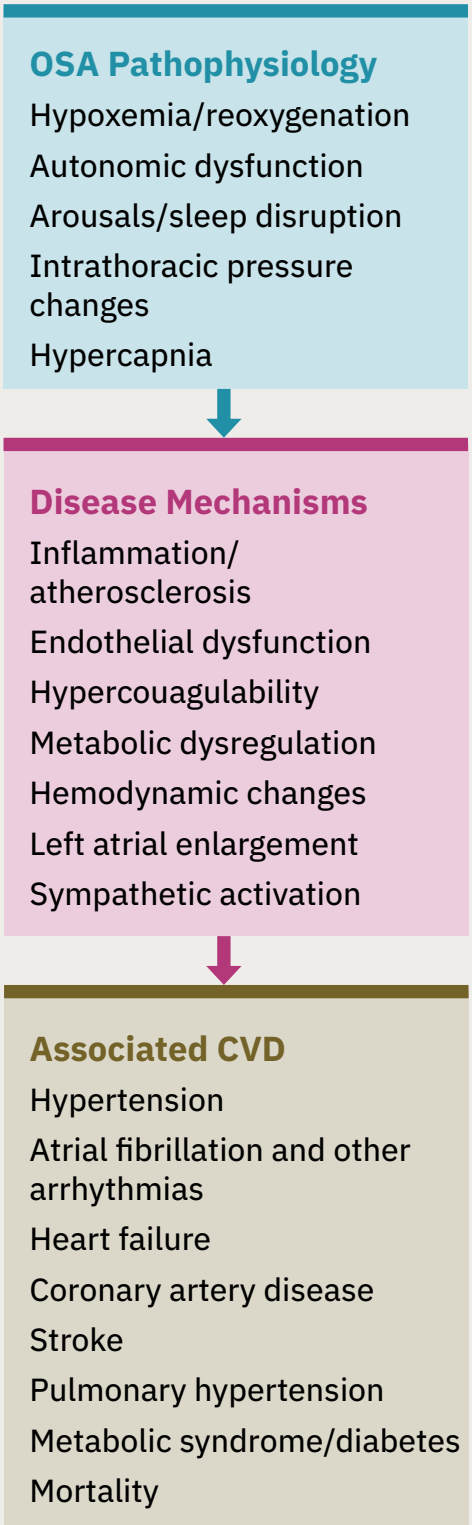
Obstructive Sleep Apnea (OSA), the most common type of sleep-disordered breathing, is characterized by recurrent complete (apnea) and partial (hypopnea) obstructive events of the upper airway, resulting in intermittent hypoxemia, autonomic fluctuation, and sleep fragmentation.[25,26]

Individuals with severe sleep-disordered breathing have two- to four-fold higher odds of complex arrhythmias,[27] and the consequent excessive daytime sleepiness is associated with an increased prevalence of CV disease and events.[28] Evaluation for OSA is important for patients with poorly controlled hypertension, pulmonary hypertension, recurrent atrial fibrillation after prior cardioversion or ablation, and New York Heart Association Class II to IV heart failure who have suspected OSA.[25] Patients with nocturnal angina, myocardial infarction, arrhythmias, or implanted cardiac defibrillator activity may be at especially high risk for comorbid OSA.[25]

Pathophysiology

Several theories explain how OSA increases CV risk. The pharyngeal occlusion of OSA causes an abrupt drop in intrathoracic pressure, which leads to increased transmural pressure in all cardiac chambers and great vessels.[29] Recurrent airway obstruction also results in intermittent hypoxia, autonomic fluctuation, and sleep fragmentation (Figure 2).[25] Airway obstruction, with concomitant elevations in blood pressure, increased oxidative stress, inflammation, and hypercoagulation, subjects the heart and great vessels to chemical stress.[28] The culmination of repeated sleep disturbances over time results in long-term deficits in the heart-brain circulation and metabolism.[29] Other theories include heightened upper airway resistance, impaired respiratory load compensation, and increased arteriolar carbon dioxide.[25]

Figure 2. Cardiovascular Complications of OSA



Adapted from *Obstructive sleep apnea and cardiovascular disease: A Scientific Statement from the American Heart Association*

Prevalence

Among patients age 45-75, 34% of men and 17% of women have been diagnosed with OSA.[30] However, the disease may be widely underdiagnosed in certain patients. According to population surveys, 86–95% of individuals with clinically significant OSA reported no prior OSA diagnosis.[31] In patients with concomitant CV health problems, including hypertension, heart failure, coronary artery disease, pulmonary hypertension, atrial fibrillation, and stroke, the prevalence of OSA is as high as 40–80%.[32]

Risk factors for OSA include being male, being older, being Black,[33] family history of OSA, craniofacial dysmorphisms, and obesity.[25] Anatomical abnormalities, including retrognathia, enlarged tonsils, and increased soft tissue in the neck, are the most common factors contributing to airway obstruction (Figure 3).[25] Black patients have more severe symptoms and more severe OSA on presentation compared with people who are white.[34]

Figure 3.
OSA Symptoms and Diagnosis

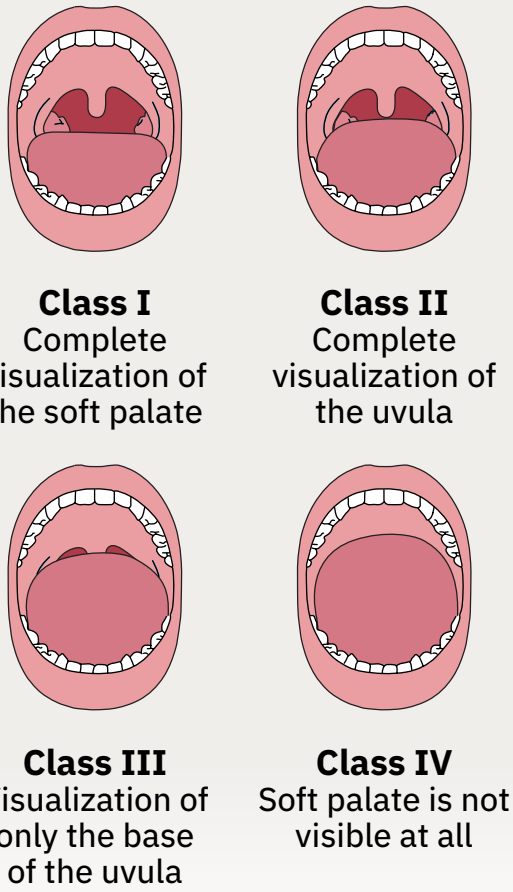
Signs and Symptoms

- Excessive daytime sleepiness
- Morning headaches
- Memory impairment
- Irritability and/or changes in affect
- Difficulty concentrating
- Nocturia
- Decreased libido and erectile dysfunction

Exam Findings

- Obesity
- Increased neck circumference
- Mallampati score ≥3
- Craniofacial abnormalities

The Mallampati Score



Class I
Complete visualization of the soft palate

Class II
Complete visualization of the uvula

Class III
Visualization of only the base of the uvula

Class IV
Soft palate is not visible at all

Adapted from *Obstructive sleep apnea and cardiovascular disease: A Scientific Statement from the American Heart Association*

Diagnosis

Evaluation should be considered in patients with the risk factors, noted signs and symptoms, and exam findings summarized in Figure 3. The American Academy of Sleep Medicine recommends an annual OSA screening for all adult patients with the risk factors listed in Figure 4.[35]

Figure 4. Risk Factors for OSA

- **H** – Heart failure
- **E** – Elevated blood pressure
- **A** – Atrial fibrillation (A-fib)
- **R** – Resistant hypertension
- **T** – Type 2 diabetes
- **S** – Stroke

Patients who should also be screened for OSA include those who are obese and those who have nocturnal dysrhythmias, pulmonary hypertension, or coronary artery disease.[35] Preferred outpatient screening questionnaires include the Berlin Questionnaire, the STOP, and the STOP-BANG (Figure 5).[36] These questionnaires have reported sensitivity between 77–89% and a specificity of 32–34%.[37]

Figure 5. STOP-BANG OSA Questionnaire

STOP	Yes or No?
Do you **S**NORE loudly (louder than talking or loud enough to be heard through closed doors)?	
Do you often feel **T**IRED, fatigued, or sleepy during daytime?	
Has anyone **O**BSERVED you stop breathing during your sleep?	
Do you have or are you being treated for high blood **P**RESSURE?	
BANG	**Yes or No?**
BMI more than 35kg/m^2?	
AGE over 50 years old?	
NECK circumference > 16 inches (40cm)?	
GENDER: Male?	

High risk of OSA: Total score of 5 - 8 Yes
Intermediate risk of OSA: Total score of 3 - 4 Yes
Low risk of OSA: Total score of 0 - 2 Yes

TOTAL SCORE: _____
(Total Number of Yes)

Adapted from *STOP-BANG, STOP, and Epworth sleepiness scale in detecting obstructive sleep apnea: a bivariate meta-analysis*

Suspected OSA can be confirmed with polysomnography, which is usually done at an overnight center. Home sleep apnea testing can be arranged but is less sensitive and specific. Diagnosis of OSA in the presence of symptoms, including nocturnal breathing disturbances, daytime sleepiness, and fatigue despite a sufficient opportunity to sleep (without other known medical cause), is established by an Apnea-Hypopnea Index (AHI) greater than 5 as well as a respiratory event index greater than or equal to 5 (Table 2).[25] In the absence of OSA symptoms, AHI scores of less than 15 are considered mild OSA, 15 to 30 are considered moderate OSA, and greater than 30 are considered severe OSA.[29]

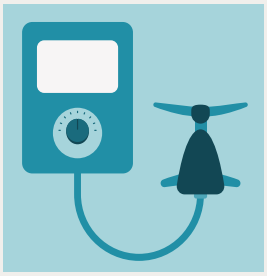
Table 2. Apnea-Hypopnea Index

	Range
Normal	<5
Mild OSA	5-15
Moderate OSA	15-30
Severe OSA	>30

Adapted from *Obstructive sleep apnea and cardiovascular disease: A Scientific Statement from the American Heart Association*

Treatment

Treatment of OSA varies based on the patient and the level of severity. Treatments may include a dentist-fitted oral appliance, a Continuous Positive Airway Pressure (CPAP) device, or upper airway surgery (Table 3). If the patient is overweight, weight loss should initially be addressed with lifestyle changes and a weight loss goal of at least 5 to 10 percent of total body weight.[25,38,39] Bariatric surgery may be considered in cases of severe obesity with concomitant OSA.[25] Randomized controlled trials show that weight loss can result in the resolution of OSA and has the collateral benefits of lowering blood pressure and improving insulin sensitivity.[40] Modest weight loss may be effective in reducing new sleep disordered breathing. One study found that a weight gain of 10% of total body weight equals a 32% increase in AHI, demonstrating that weight is the most modifiable risk factor for OSA.[41]



CPAP Device

Weight Loss

CPAP treatment may improve in-office measurements of blood pressure and overnight readings. Whereas CPAP treatment has not been shown to improve survival rates in patients with previous coronary or cerebrovascular disease, it may lower the risk of stroke and improve overall well-being.[28] CPAP has not yet been proven to reduce adverse cardiovascular outcomes.[25,28] CPAP for the prevention of CV disease appears to have limited supportive data largely due to patient intolerance of CPAP.[25,28] Continued studies of CPAP and CV disease with improved therapeutic precision are needed.[42]

Pharmacologic therapies for OSA are being investigated, and further studies are needed to demonstrate efficacy.[43] SGLT2 inhibitors, in particular, show promise, potentially related to both weight loss and other variables.

Table 3. Options for Treatment of Obstructive Sleep Apnea

Treatment Type	Effectiveness	Potential Issues	Comments
Lifestyle Intervention With Medical Weight Loss	10% weight loss reduces AHI by 26%	Risks of medical weight loss pharmacotherapy	Behavioral lifestyle interventions should be considered foundational to OSA treatment; multiple metabolic benefits
Positional Therapy	Reduces AHI to the same degree as CPAP in select patients[44]	Discomfort leads to poor adherence	Long-term adherence about 10% due to discomfort
Oral Appliances	Adherence greater than CPAP with comparable improvements in sleepiness	Discomfort, excess salivation	Tongue retaining or mandibular advancement
CPAP	Clinical trials have shown improvement in blood pressure and sleepiness	Discomfort, claustrophobia, nasal congestion, dry mouth	Targets airway collapsibility using inspiratory/expiratory pressure as a splint
APAP	Trials have not consistently shown improved adherence or inferiority to CPAP	Same as CPAP	Pressure settings automatically adjusted based on changes in ventilation
BiPAP	Similar to CPAP	Same as CPAP	Allows different pressures to be used in inspiration vs. expiration
ASV	Increased mortality with ASV use in those with systolic heart failure[45]	Same as CPAP	Provides continuous pressure and volume adjustments to maintain constant ventilatory volume
Upper Airway Surgery	Rarely curative, may improve clinical outcomes	Perioperative risks including pain	Usually used in conjunction with other treatments, such as nasal septal surgery
Neurostimulation	Reduces AHI to the same degree is CPAP in select patients[24]	Invasive	Women and older persons appear to be more responsive
Bariatric Surgery	OSA-specific trials have not been performed to assess the impact on OSA severity	Perioperative risks; postoperative complications including acid reflux; procedure failure	Multiple metabolic benefits; follow up polysomnography essential

CPAP = Continuous positive airway pressure; APAP = Automatic positive airway pressure; BiPAP = Bilevel positive airway pressure; ASV = Adaptive-servo ventilation
Adapted from *Obstructive sleep apnea and cardiovascular disease: A Scientific Statement from the American Heart Association*

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