

Review

Management of Obstructive Sleep Apnea in People with Type 2 Diabetes

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Abstract

Obstructive sleep apnea (OSA) is a common condition defined by recurrent upper airway collapse, intermittent hypoxia, and sleep fragmentation that leads to sympathetic activation and adverse cardiometabolic effects. OSA and type 2 diabetes mellitus (T2DM) share a complex, bidirectional relationship driven in part by obesity, but also by overlapping pathophysiologic mechanisms. Diabetes may worsen upper airway collapsibility, while untreated OSA may exacerbate hyperglycemia, contributing to a self-perpetuating cycle of metabolic abnormalities. Continuous positive airway pressure (CPAP) remains the first-line therapy for OSA, and improves symptoms and some cardiometabolic parameters, although effects on glycemic control have been mixed and may depend on patient factors. Alternative therapies play important roles in some patients. Incretin-based therapies, particularly tirzepatide, have emerged as promising treatments for obesity-related OSA following the SURMOUNT-OSA trial, demonstrating significant reductions in apnea–hypopnea index and cardiometabolic risk factors, though their effect in patients with T2DM remains unclear. Considerations such as sarcopenia with weight loss and evolving pharmacologic and procedural strategies further shape management decisions. This review synthesizes current evidence on the pathophysiologic overlap, therapeutic approaches, and future directions in optimizing care for patients with coexisting OSA and diabetes.

Keywords: diabetes; obstructive sleep apnea; treatment; GLP1RA

1. Introduction

Obstructive sleep apnea (OSA) is a common disorder estimated to affect up to 1 billion people worldwide [1]. OSA is defined by intermittent pauses in breathing which occur during sleep and lead to recurrent arousals from sleep as well as desaturations and catecholamine surges [1–3] (Figure 1). As a result, OSA has major neurocognitive and cardiometabolic consequences. OSA is increasing in prevalence based on the obesity pandemic and the aging of the population [4]. With increasing appreciation for the importance of OSA, more patients are undergoing sleep testing, yielding a higher prevalence of diagnosed OSA. Although treatment can be transformative for some, there remains a large burden of untreated and/or unrecognized OSA patients. Thus, increasing awareness about this condition is important. Although there are many endophenotypes of OSA, including non-obese OSA, craniofacial anatomical factors, impaired neuromuscular control



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and unstable ventilatory control (elevated loop gain) [5,6], this article will focus most on the links between obesity, diabetes and OSA.

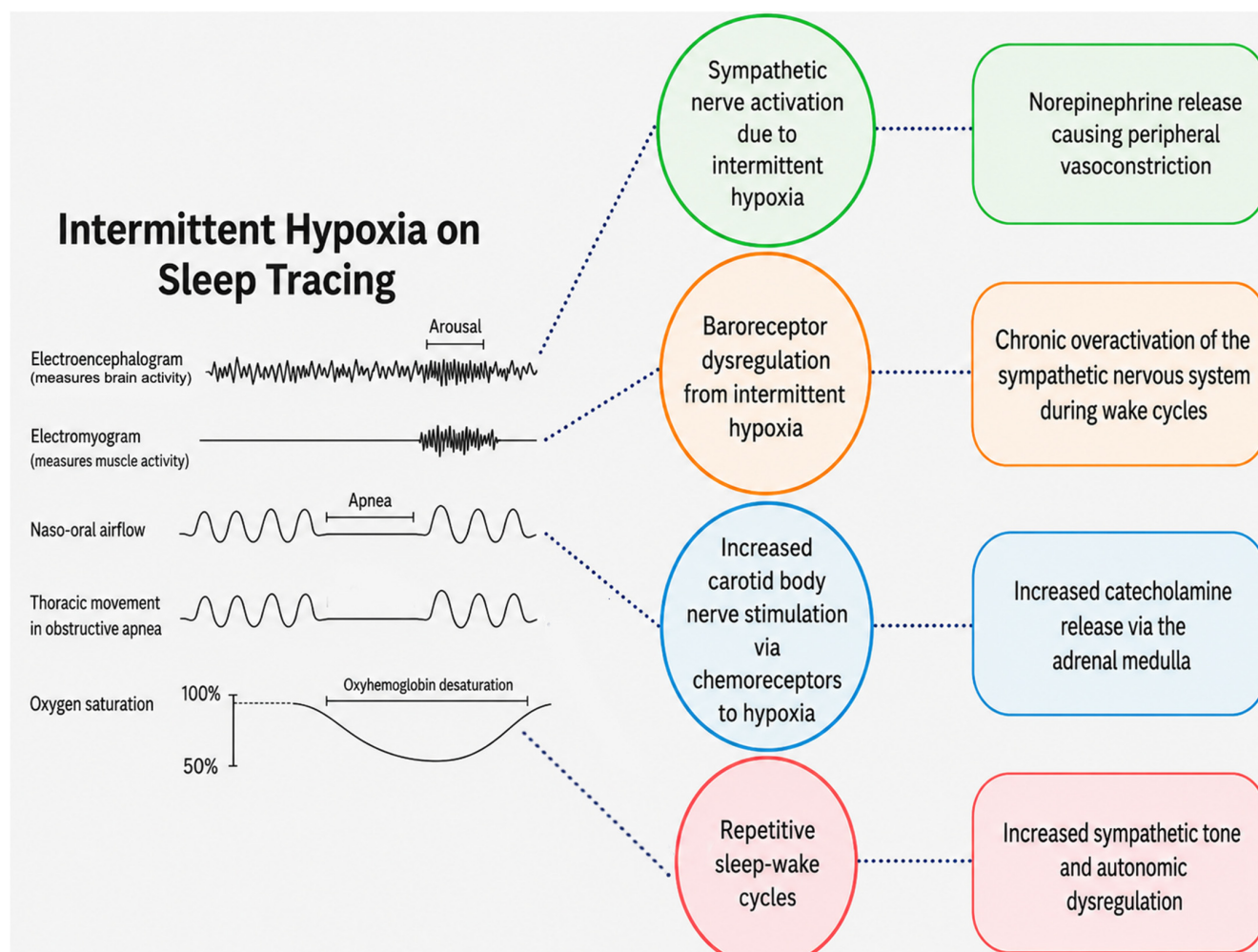


Figure 1. Pathophysiology of obstructive sleep apnea.

There are important links between OSA and type 2 DM (T2DM), (Figure 2). First, obesity is a major risk factor both for OSA and for T2DM [7,8]. In the SLEEP-AHEAD study which was a sub-study of LOOK-AHEAD, roughly 87% of patients with obesity and T2DM had clinically important OSA [9,10]. Despite this fact, fewer than 5% of patients were on OSA therapy even one year after the diagnosis of OSA was given to both the patients and their providers. Thus, an unmet need clearly exists particularly when other therapeutic targets in T2DM have been optimized. Second, DM may contribute at least theoretically to upper airway collapsibility and OSA via neuromyopathy. Both T1DM and T2DM have been associated with OSA in prior studies, although this article will focus on T2DM based on its prevalence and the availability of the current literature. Upper airway reflexes are felt to be impaired in some patients with T2DM and this abnormality may contribute to OSA, at least in a subset of patients [11–13]. Both sensory and motor abnormalities have been demonstrated in the upper airway of OSA patients [14,15]. Third, OSA may contribute to worsening glucose control in people with T2DM [16]. Counter-regulatory hormones are released with repetitive apneas; these hormones may contribute to worsening insulin resistance, which over time, contributes to glucose dysregulation as well as metabolic complications in T2DM. Fourth, some evidence suggests that T2DM contributes to microcirculatory abnormalities as well as dysfunction in vascular smooth

muscle, whereas OSA leads to dysfunction primarily in the endothelium [17–20]. Thus, at least in theory, both OSA and T2DM need to be addressed to optimize vascular outcomes in afflicted patients. Regardless of the mechanisms, OSA, DM, and obesity clearly have complex and interactive relationships which have likely been under-appreciated in the literature to date. Additional pathophysiological mechanisms may also be important in these patient populations, including fragmentation of sleep, alterations in gut microbiome, sleep deprivation and circadian misalignment; these factors are outside of the scope of this review but well-discussed in the literature [16,21–24].

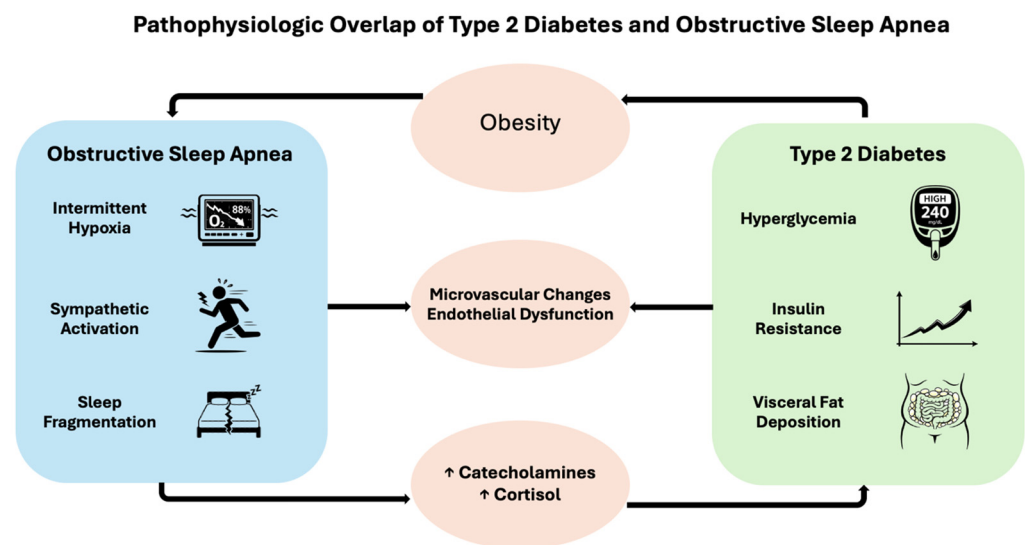


Figure 2. Obesity is a major risk factor for OSA. Patients with OSA experience intermittent hypoxia which results in sleep fragmentation and frequent arousals which lead to sympathetic activation. This pattern of events in turn leads to catecholamine surges which contribute to the hyperglycemia, insulin resistance, and visceral fat deposition observed in T2DM. Both conditions contribute to microvascular changes and T2DM is associated with increased obesity, ultimately resulting in a pathophysiologic cycle of worsening disease if not appropriately treated.

Regarding OSA diagnosis, a high index of clinical suspicion is required for this highly prevalent disease. Conventional stereotypes of older men with obesity have led to under-appreciation of the prevalence of OSA in lean individuals, in women, and in non-Caucasian cohorts [3,25,26]. Screening methods have been developed including various questionnaires (STOP-BANG, Maislin, Berlin), although further validation is required in diverse populations with T2DM before they can be widely recommended [27–29]. Home sleep testing has facilitated OSA diagnosis greatly as compared to in-laboratory polysomnography. Home sleep testing uses highly variable approaches and equipment, but generally can provide acceptable sensitivity and specificity compared to more cumbersome in-laboratory evaluation [30,31].

2. Conventional OSA Treatments

The treatment of OSA until recently has been largely focused on CPAP (continuous positive airway pressure) which provides a pneumatic splint for the upper airway for patients during sleep [32]. CPAP can have transformative benefits for some patients but can be poorly tolerated in other OSA patients. On aggregate, the data suggest that the majority of OSA patients are adherent with therapy if given adequate education and modern technology [33]. A recent meta-analysis of CPAP studies showed potential benefits in cardiovascular and all-cause mortality [34]. Combining both randomized and non-randomized studies, the data showed improvement in these outcomes, whereas the

randomized studies in isolation showed only trends towards benefits. Randomized studies are clearly the gold standard, but in the OSA context, include fewer than 5000 patients in total, many who are minimally symptomatic and generally followed for relatively short durations. Thus, the event rates are quite low, and the studies are underpowered to show improvements in outcomes. In contrast, non-randomized OSA studies include roughly 1.1 million patients who tend to be symptomatic and followed for long durations, leading to high event rates. However, non-randomized studies are susceptible to the healthy user effect (i.e., those using CPAP are systematically different from those not using CPAP) as well as issues with residual confounding, such as that unrecognized covariates may be important in driving the observed outcomes [35]. Thus, the data are consistent in showing major benefits from CPAP, although further randomized studies are encouraged.

However, there is general acknowledgement that alternative therapies are needed and may be helpful for some OSA patients. Oral appliances (e.g., mandibular advancement devices) are made by certified dentists and can serve to protrude the mandible during sleep, thereby reducing the risk of upper airway collapse. Oral appliances have shown comparable results to CPAP in meta-analyses, although clinically they are generally reserved for second-line therapy [36–38]. Upper airway surgery can also be undertaken, although the results can be somewhat variable. Surgical approaches have the advantage of not requiring patient adherence but have the risk of complications associated with the procedures. Surgical outcomes have been somewhat mixed in prior studies although work is ongoing in this context. Upper airway stimulation including hypoglossal nerve stimulation is being increasingly utilized although data are still evolving regarding its impact on major clinical outcomes [39–43]. Conservative measures for OSA can include diet and exercise, the avoidance of alcohol and other depressants, the maintenance of nasal patency and in some patients the avoidance of supine posture. Despite these available therapies, many patients remain largely untreated leading to further discussions about optimizing therapy.

3. OSA and GLP-1RAs

The glucagon-like peptide-1 receptor agonists (GLP1RAs) have been widely used for the treatment of obesity and T2DM. GLP1RAs have been available for over 20 years, but newer-generation medications have only become available more recently. Tirzepatide is a GLP1RA with GIP (gastric inhibitor peptide or glucose-dependent insulintropic polypeptide) which was initially approved for T2DM and subsequently for obesity. Based on the SURMOUNT-OSA study, tirzepatide received FDA approval in December 2024 for treatment of moderate to severe OSA in people with obesity.

The SURMOUNT-OSA study consisted of two studies: study 1 with OSA patients not using CPAP and study 2 with OSA patients who were using CPAP [44,45]. In both studies, participants were randomized to receive either tirzepatide at the maximum tolerated dose or placebo. It is of note that there was no direct comparison of CPAP to tirzepatide in this study; thus, CPAP remains the first-line therapy for OSA until comparative effectiveness studies have been completed. The studies examined the primary outcome of the apnea–hypopnea index (marker of OSA severity). Prespecified secondary outcomes were controlled for multiple comparisons and included systolic blood pressure, body weight, hsCRP, patient-reported outcomes, and sleep–apnea-specific hypoxic burden (based on the area under the desaturation curve). The data showed marked improvement in the primary as well as all prespecified secondary outcomes at one year. Subsequent mediation analyses have been published which examine the influences on various outcomes of interest. For example, the improvement in systolic blood pressure observed in SURMOUNT OSAS was largely mediated by body weight. In contrast, improvements in HOMA-IR and hsCRP were partially mediated by OSA and partially mediated by changes in body weight [44]. Direct

effects of the medication are also thought to be important, e.g., at the level of the carotid body, which may impact autonomic function [46].

It is noteworthy that people with diabetes mellitus were excluded from SURMOUNT-OSA, leaving open the question of what should be done for this large group of OSA patients. Several arguments could be made. Due to T2DM not being included in SURMOUNT-OSA, one might argue to await further data before giving evidence-based recommendations regarding these patients. On the other hand, tirzepatide has been shown to have major benefits in T2DM from the standpoint of glycemic control, and thus these patients may well be amenable to intervention, with or without OSA [47]. Some data suggest the degree of weight loss with GLP1RA in DM is less than in patients without DM [48]. Thus, one might argue that the benefits for patients with OSA with DM may be less than for OSA patients without DM based on the anticipated weight loss. However, given the high vascular risk of DM patients, one might argue that OSA patients with DM may be the most likely to benefit from tirzepatide from a cardiovascular outcomes perspective. Thus, the role of tirzepatide in OSA patients with DM is unclear, but we would argue for a careful discussion regarding risks and benefits of various interventions for these OSA patients until further data emerge.

4. T2DM and CPAP

The role of CPAP therapy in OSA patients with DM is also unclear. Notably, Chirinos et al. [49] had a prominent paper in *NEJM* examining the impact of treating either obesity or OSA, or both, in afflicted patients. On aggregate, the data suggested that treating both OSA and obesity was generally better than treating either entity alone from the standpoint of various metabolic outcomes such as systolic blood pressure, insulin resistance, and triglyceride levels. Thus, treating both OSA and its underlying causes such as obesity is generally recommended.

The impact of CPAP therapy on glucose control has had somewhat mixed results. In general, CPAP adherence in these studies has been somewhat variable, perhaps driving some of the variability observed in various interventional studies. In some studies, enrollment of T2DM patients with beta cell failure has not shown improvement in glucose control with CPAP, perhaps as these patients were too late in their disease course for such interventions to show benefit. Weinstock et al. [50] performed a randomized study of CPAP vs. sham CPAP in OSA patients with impaired glucose tolerance but showed no benefit to OSA treatment in this context. The authors did see some improvement in the subgroup of severe OSA patients, but these findings will clearly require prospective corroboration. One study by Aronsohn et al. [51] used an approach of bringing patients into a carefully monitored hospital setting to apply CPAP, where adherence could be optimized. In this context, Aronson et al. did observe improved glycemic control with CPAP therapy, ostensibly via suppression of surges in counter-regulatory hormones. Although the approach of hospitalizing patients to apply CPAP is not scalable, it does provide proof of concept that optimizing OSA therapy may have important metabolic benefit.

5. Sarcopenia

One issue which is frequently discussed with major weight loss is sarcopenia. Estimates suggest that roughly 20% of the weight loss from GLP1RA is attributable to muscle rather than fat. The clinical implications of this finding are unclear; however, several issues are worth emphasizing. Some reduction in bone density and skeletal muscle is likely appropriate with major weight loss. An individual with severe obesity places a large axial load on their bones and muscles during standing and ambulation; thus, major weight loss may reduce the demand on skeletal structures and postural muscles.

Several interventions have been evaluated to mitigate sarcopenia. Increasing dietary protein intake may help reduce skeletal muscle loss during GLP1RA therapy. Resistance exercises have also been recommended as a means to maintain skeletal muscle, which is critical to preserving basal metabolic rate. Pharmacologically, selective androgen modulators have been suggested as a strategy to preserve skeletal muscle, although further outcomes data are required to evaluate this approach. In addition, new agents are in development to preserve or even build skeletal muscle. Bimagrumab, an activin inhibitor which helps prevent the breakdown of actin and myosin, is currently being assessed in studies, and preliminary data suggest benefit in sarcopenia when used in combination with GLP1RA [52]. SiRNAs such as WVE007 are also being developed which may present an additional approach to preserving skeletal muscle in the context of weight loss.

6. Future Directions

Although considerable advances have been made in this area, several newer therapeutics are currently being studied. Retatrutide is a GLP1RA with GIP and glucagon agonist properties that is currently undergoing large-scale randomized studies. The initial data suggest that retatrutide has increased body weight loss compared to other incretin-based therapies [53]. Ongoing studies such as TRIUMPH should provide further clarification on retatrutide's outcomes, including on OSA, in the near future [54]. In addition, orforglipron is an oral GLP1RA which is currently undergoing randomized studies including ATTAIN-OSA [55]. The results of these studies should be available soon and provide further information regarding the role of oral GLP1RA in treatment of OSA patients. In general, patients prefer using oral agents to subcutaneous medications, but additional data will be required regarding optimizing oral therapy for OSA patients with excess body weight. Moreover, oral agents are relatively easy to produce and do not require refrigeration which can be a barrier to injectable medications, particularly in under-resourced areas.

Eloralintide, an amylin analog, was recently studied in *The Lancet* and showed clinically meaningful and dose-dependent weight loss over a 48-week period [56]. Data regarding eloralintide suggest it does promote up to 20% weight loss with fewer gastrointestinal side effects compared to incretin-based therapies. Its impact in OSA is currently unclear. Further, cagrasema, a combination GLP1-RA and amylin analog, has been studied and found to provide up to 22.7% weight loss in adults with diabetes [57].

The role of bariatric surgery in the future is currently unclear. To date, the best available data for sustained weight loss are seen with bariatric procedures [58]; however, with the increasing efficacy of incretin-based therapies, there is considerable speculation that bariatric surgery may be recommended less frequently in the future. Indeed, recent data have shown a reduction in the frequency of bariatric surgery procedures in the USA. Minimally invasive techniques are also being developed which may have a role depending on further outcome data. All in all, other agents are rapidly in development and this area of medicine is quickly changing.

Additionally, it should be noted that although GLP1Ras are rather new to the clinical setting, there are newer data that confirm the durability of these medications' effects [59]. It has been found that with discontinuation of GLP1Ras, there are instances of rebound weight gain [60].

7. Conclusions

In conclusion, several points deserve emphasis. First, diet, exercise and sleep are considered the three pillars of health, with the dogma being that if one ignores one, the other two will suffer. Second, clinical suspicion for sleep disorders should be high in people with DM, particularly given the high prevalence of clinically important abnormalities.

Third, the diagnosis and treatment of OSA have evolved considerably in recent years such that most OSA patients with DM can receive effective interventions. Fourth, active research will likely be crucial in defining the future of optimal management of these patients.

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Abbreviations

The following abbreviations are used in this manuscript:

OSA	Obstructive sleep apnea
T2DM	Type 2 diabetes mellitus
CPAP	Continuous positive airway pressure
GLP1RA	Glucagon-like peptide-1 receptor agonist
HOMA-IR	Homeostatic model assessment of insulin resistance
hsCRP	High-sensitivity C reactive protein

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