

OBSTRUCTIVE SLEEP APNOEA SYNDROME: ETIOLOGY, DIAGNOSIS, AND CURRENT TREATMENT METHODS

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ABSTRACT

Background: Obstructive sleep apnoea (OSA) is a prevalent disorder associated with significant cardiovascular, metabolic, and neurocognitive morbidity. Despite advances in understanding its mechanisms and management, awareness and early detection-particularly in primary and dental care-remain limited.

Aims: This review aims to summarise recent advances in the aetiology, diagnosis, and treatment of obstructive sleep apnoea (OSA), with a focus on evidence published between 2020 and 2025, and to highlight emerging strategies for personalised patient care.

Methods: This narrative review synthesises literature published between 2020 and 2025 identified through targeted searches of PubMed/MEDLINE, Embase, Cochrane Library, guideline repositories (AASM, ERS), and ClinicalTrials.gov using the terms "obstructive sleep apnoea", "OSA", "diagnosis", "home sleep test", "hypoxic burden", "CPAP", "mandibular advancement", "hypoglossal", "GLP-1", "tirzepatide", and "solriamfetol". Approximately 120 studies were screened, and 40 were included based on relevance, methodological quality, and contribution to recent clinical evidence. Priority was given to systematic reviews, high-quality randomized trials, and current clinical guidelines; older seminal studies were cited when foundational.

Results: Recent work refines OSA as a set of overlapping phenotypes: anatomic collapsibility; loop gain; arousal threshold and muscle responsiveness that influence diagnostic yield and treatment response. Advances include expanded use of home sleep apnea testing (HSAT) and oximetry for case-finding, the hypoxic-burden metric for risk stratification, growing evidence for personalised non-CPAP(non-Continuous PositiveAirway Pressure) therapies (mandibular advancement devices, hypoglossal nerve stimulation, multi-level surgery), improved adherence via telemonitoring, and the emergence of obesity pharmacotherapies (GLP-1 agonists and dual agonists such as tirzepatide) that reduce AHI in patients with obesity. CPAP remains first-line for symptom control, but its effect on hard cardiovascular endpoints is inconsistent and appears moderated by adherence. [4-8]

Conclusion: Management of OSAS is shifting from a one-size-fits-all CPAP paradigm toward phenotype-guided, multimodal strategies. Future trials must prioritise clinically meaningful endpoints, adherence strategies and health-system implementation.

Keywords: Obstructive Sleep Apnea; Sleep Apnea Syndromes; Continuous Positive Airway Pressure; Hypoglossal Nerve Stimulation; Mandibular Advancement Device; Hypoxic Burden; Telemedicine; GLP-1 Receptor Agonists

INTRODUCTION

BACKGROUND AND RATIONALE

Obstructive sleep apnoea syndrome (OSAS) is a highly prevalent, yet frequently underdiagnosed disorder that affects an estimated 936 million adults worldwide, including 425 million with moderate to severe disease [1]. Its prevalence continues to rise in parallel with global increases in obesity and ageing populations. OSAS contributes substantially to public health burden through its associations with daytime sleepiness, cognitive impairment, accident risk, metabolic dysfunction, and increased cardiovascular morbidity and mortality [2-5]. Long-term cohort studies demonstrate that untreated OSAS is linked to a two-to threefold higher risk of stroke and all-cause mortality [4,5].

Pathophysiologically, OSAS is a heterogeneous disorder characterised by the interplay of anatomical upper-airway collapsibility and physiological traits-loop gain, arousal threshold, and pharyngeal muscle responsiveness-that together shape disease expression and therapeutic response [2-4]. Recognising this complexity has led to a shift from uniform continuous positive airway pressure (CPAP) treatment toward individualised, multimodal care informed by endotype and phenotype.

Despite several prior reviews addressing OSAS pathogenesis and management, most predate the recent expansion of precision medicine, digital diagnostics, and metabolic pharmacotherapy. In particular, major updates since 2020 include the introduction of hypoxic-burden metrics for cardiovascular risk stratification [13,14], validated wearable technologies for sleep assessment [11,37], hypoglossal nerve stimulation and positional devices as CPAP alternatives [21-23,31], and the emergence of GLP-1 and dual agonist therapies (e.g., tirzepatide) demonstrating AHI reduction in obese patients [29]. These advances necessitate an up-to-date synthesis integrating evolving diagnostic tools, therapeutic innovations, and phenotype-based treatment selection.

AIMS

This review aims to summarise and critically appraise recent (2020–2025) developments in the aetiology, endotyping, diagnostics, and treatment of obstructive sleep apnoea (OSA), with a focus on identifying evidence gaps and priorities for clinical practice and research. Its novelty lies in integrating advances in pathophysiological phenotyping, diagnostic technology, and personalised therapeutic strategies, highlighting shifts beyond the traditional one-size-fits-all CPAP paradigm toward multimodal, patient-tailored approaches.

RESEARCH QUESTIONS

Specifically, this review addresses the following research questions:

1. What recent insights have refined the phenotypic and endotypic understanding of OSA and its clinical heterogeneity?
2. How have diagnostic methods evolved toward more accessible, technology-supported screening and monitoring?
3. Which emerging or adjunctive therapies demonstrate clinically meaningful efficacy, safety, and adherence?
4. How can phenotype-guided strategies be implemented in clinical practice to optimise patient outcomes and reduce the burden of untreated OSA?

METHODS

SEARCH STRATEGY

A focused, reproducible literature search was conducted across PubMed/MEDLINE, Embase, and the Cochrane Library, including publications up to September 2025. Additional sources comprised international guideline repositories (American Academy of Sleep Medicine [AASM], European Respiratory Society [ERS]), ClinicalTrials.gov, and relevant high-impact journals. The search strategy used combinations of the following terms: “obstructive sleep apnea”, “OSA”, “hypoxic burden”, “home sleep test”, “HSAT”, “STOP-Bang”, “CPAP”,

“mandibular advancement device”, “hypoglossal nerve stimulation”, “tirzepatide”, “GLP-1”, “solriamfetol”, “pitolisant”, and “telemedicine”.

Priority was given to systematic reviews, clinical guidelines, and randomized controlled trials published between 2020 and 2025. Earlier seminal studies were included selectively to provide context or to reference foundational mechanisms and interventions.

Approximately 120 articles were screened, and 40 were finally included in this review based on their methodological robustness, clinical relevance, and original contribution to the understanding of OSA pathophysiology, diagnosis, or treatment.

This review is narrative in nature and does not conform to PRISMA systematic review standards. Instead, it aims to provide an integrative synthesis and critical interpretation of current evidence, highlighting emerging research and clinical implications.

INCLUSION/EXCLUSION

Included: adult human studies, systematic reviews, RCTs, large cohort studies and clinical guidelines published 2020-2025 (exceptions for key earlier trials). Excluded: pediatric-only studies (unless clearly relevant), case reports without broader evidence, non-English papers where no translation was available.

RECENT EVIDENCE AND ADVANCES IN ETIOLOGY, DIAGNOSIS AND TREATMENT OF OSA

ETIOLOGY AND PATHOPHYSIOLOGY (ENDOTYPES)

OSAS results from interactions between anatomy (fat deposition, craniofacial structure, upper-airway soft tissues) and physiological control systems. Contemporary conceptual models emphasise four modifiable pathophysiological traits: pharyngeal collapsibility, neuromuscular compensation, loop gain (ventilatory control instability), and arousal threshold. These traits explain heterogeneity in presentation and predict response to treatments (e.g., patients with dominant anatomical collapse tend to respond better to mechanical splints, surgery or HNS; those with high loop gain may respond to respiratory-stabilising strategies). [2-4] Several translational and imaging studies have refined trait quantification, supporting personalised treatment pathways.

Diagnostic pathways and new metrics

SCREENING TOOLS

Screening remains an important first step because a large proportion of patients with obstructive sleep apnoea (OSA) remain undiagnosed. Questionnaires such as the STOP-Bang and NoSAS scores are widely used in clinical and perioperative settings. STOP-Bang (Snoring, Tiredness, Observed apnoeas, high blood Pressure, BMI, Age, Neck circumference, Gender) has been consistently validated across different populations. Its high sensitivity for detecting moderate-to-severe OSA makes it valuable for case finding, though its limited specificity can generate false positives and requires confirmatory testing [9,10]. The NoSAS score, a more recent tool, incorporates neck circumference, obesity, snoring, age and sex, and demonstrates comparable diagnostic accuracy with fewer items. Both tools are simple and inexpensive, making them well suited for triage in primary care, dental practices and pre-operative assessments. However, they should not be used as stand-alone diagnostic tests, particularly in patients with atypical presentations such as non-obese OSA phenotypes.

SLEEP TESTING MODALITIES

Full overnight polysomnography (PSG) remains the diagnostic gold standard, offering comprehensive evaluation of sleep stages, arousals, respiratory events and comorbid sleep disorders. However, cost, limited accessibility, and the burden on sleep laboratories restrict its universal application. To improve access, home sleep apnoea testing (HSAT) has gained acceptance in recent years for uncomplicated adults with suspected moderate-to-severe OSA. Both the American Academy of Sleep Medicine and European professional societies have updated guidelines endorsing HSAT in appropriate cases [7,8]. Evidence shows HSAT performs reliably in high-pretest probability patients, but it is less sensitive in mild OSA and may underestimate severity due to the absence of electroencephalographic sleep staging.

Overnight oximetry is a lower-cost option that measures oxygen desaturation indices (ODI). It has high sensitivity for moderate-to-severe disease and is useful as a triage tool, particularly in resource-limited settings [12]. Portable monitors and multi-channel cardiorespiratory polygraphy expand diagnostic capacity further, although they share limitations with HSAT in patients with comorbidities.

Consumer wearables equipped with photoplethysmography and accelerometry represent an emerging frontier.

Validation studies suggest reasonable agreement with PSG for screening purposes [11], though variability across device platforms and populations remains a limitation. Algorithmic and AI-based analyses of oximetry and wearable data are promising for automated large-scale screening, but further independent validation is required before widespread adoption [33].

HYPOXIC BURDEN AND NOVEL PROGNOSTIC METRICS

The Apnoea-Hypopnoea Index (AHI) has long been the standard measure of OSA severity, but its prognostic value is increasingly questioned. Hypoxic burden, which integrates the depth, duration and frequency of oxygen desaturations during sleep, has emerged as a superior predictor of cardiovascular morbidity and mortality [13]. Unlike AHI, hypoxic burden accounts for the physiological “load” imposed on the cardiovascular system, better reflecting interindividual risk. Recent cohort studies demonstrate that hypoxic burden correlates more consistently with incident hypertension, atrial fibrillation and mortality than AHI [14]. Incorporating this metric into clinical practice may refine risk stratification, identify patients at highest risk, and guide therapeutic intensity. However, consensus on thresholds and standardisation across devices is still lacking.

Table 1 summarizes established and emerging diagnostic approaches for obstructive sleep apnoea (OSA), outlining methodology, clinical applications, advantages, limitations, and supporting evidence from recent guidelines and studies [5–18]

Table 1 Diagnostic Approaches in Obstructive Sleep Apnoea (OSA)

Diagnostic Method	Description	Advantages	Limitations
Polysomnography (PSG)	Overnight in-laboratory study measuring sleep stages, arousals, respiratory events, oxygenation.	Gold standard; comprehensive assessment; identifies comorbid sleep disorders.	Expensive; limited accessibility; burdensome for patients.
Home Sleep Apnoea Testing (HSAT)	Portable multi-channel recording used in suspected moderate-to-severe OSA without major comorbidities.	More accessible; lower cost; higher patient convenience.	Less sensitive for mild OSA; cannot detect other sleep disorders; no EEG for sleep staging.
Overnight Oximetry	Monitors oxygen saturation trends overnight to derive desaturation indices (ODI).	Low cost; useful triage tool; high sensitivity for moderate-to-severe OSA.	Limited specificity; underestimates severity; not standalone diagnostic.
Screening Questionnaires (STOP-Bang, NoSAS)	Clinical questionnaires assessing OSA risk based on symptoms and anthropometrics.	Simple; inexpensive; high sensitivity for case-finding.	Low specificity; not diagnostic; risk of false positives.
Wearables & Consumer Devices	Devices with photoplethysmography and accelerometry to estimate sleep and breathing patterns.	Scalable; convenient; potential for population screening.	Variable accuracy; limited validation; not standard of care.
Hypoxic Burden Metric	Quantifies cumulative depth, duration, frequency of oxygen desaturations.	Better predictor of cardiovascular risk vs AHI; useful for prognosis.	No universal threshold; limited standardisation; not yet routine.

THERAPEUTIC STRATEGIES

POSITIVE AIRWAY PRESSURE (PAP)

Continuous positive airway pressure (CPAP) remains the cornerstone of OSA therapy. Large randomized controlled trials and meta-analyses confirm its effectiveness in normalising AHI, improving daytime sleepiness, and reducing blood pressure, particularly in resistant hypertension [15-18]. However, the cardiovascular protection expected from CPAP has been inconsistent, with some trials failing to show reduction in hard outcomes such as myocardial infarction or stroke [16]. Importantly, individual patient data meta-analyses indicate that cardiovascular benefit is strongly dependent on adherence, with protective effects observed in patients using CPAP for more than 4 hours per night [38]. Auto-titrating PAP (APAP) offers greater comfort and is widely used in routine practice, while bilevel PAP (BiPAP) is reserved for those requiring higher pressures or with hypoventilation syndromes. Despite proven efficacy, adherence remains the major barrier, highlighting the role of behavioural interventions, patient education, and telemonitoring [32].

ORAL APPLIANCES

Mandibular advancement devices (MADs) represent the most widely studied alternative to CPAP. Randomised trials and systematic reviews show that MADs significantly improve daytime sleepiness and quality of life in patients with mild-to-moderate OSA, though they typically produce smaller reductions in AHI compared to CPAP [19,20]. Long-term adherence tends to be superior to CPAP, and some patients prefer MADs because of their portability and comfort. Their effectiveness depends on craniofacial morphology and dental health, requiring careful patient selection.

POSITIONAL THERAPY

Positional OSA, where events occur predominantly in the supine position, affects up to one-third of patients. Interventions that prevent supine sleep- ranging from simple tennis-ball techniques to advanced vibration-based devices- reduce AHI significantly in positional OSA [20,34-37]. Comparative studies suggest that while positional therapy is less effective than CPAP at reducing AHI, improvements in sleepiness and quality of life can be comparable in selected patients [35,36]. However, long-term adherence is a major challenge, with many patients abandoning therapy after several months [38]. Integration of positional therapy into multimodal treatment may be optimal.

HYPOGLOSSAL NERVE STIMULATION (HNS)

HNS has emerged as a promising therapy for PAP-intolerant patients with moderate-to-severe OSA who lack complete concentric palatal collapse. Randomized trials demonstrate significant reductions in AHI, improvements in daytime symptoms, and high nightly adherence (often exceeding that of CPAP) [21,22]. The durability of benefit over several years has been established, and real-world registries confirm sustained efficacy. Limitations include surgical invasiveness, high cost, and restricted eligibility criteria, which currently limit widespread use.

SURGICAL INTERVENTIONS

Upper airway and skeletal surgeries remain options for selected patients. Uvulopalatopharyngoplasty (UPPP) and its modifications show variable efficacy, with long-term results often limited by relapse [31]. Expansion pharyngoplasty has shown improved outcomes compared to traditional UPPP in carefully selected patients. Maxillomandibular advancement (MMA) demonstrates the highest success rates, with meta-analyses confirming long-term improvements in AHI and oxygenation, particularly in patients with craniofacial risk factors [30]. Cohort studies further suggest potential systemic benefits following surgical treatment, including improvements in cardiovascular and metabolic outcomes [31].

PHARMACOTHERAPY

Pharmacological approaches to OSA are rapidly evolving.

Wake-promoting agents: Solriamfetol and pitolisant are approved for treating residual excessive daytime sleepiness despite PAP, improving functional outcomes without reducing AHI [24-26].

Weight-loss pharmacotherapy: GLP-1 receptor agonists and dual agonists such as tirzepatide offer the most promising disease-modifying approach. Phase 3 SURMOUNT-OSA trials showed tirzepatide reduced AHI by more than 50% in obese adults with OSA, alongside substantial weight loss [23,27,28]. These findings support pharmacological weight management as a key adjunct or alternative to traditional therapies in obesity-driven OSA.

Trait-targeted drugs: Carbonic anhydrase inhibitors lower loop gain and show promise in early studies, but are not yet standard practice [27].

LIFESTYLE AND WEIGHT MANAGEMENT

Lifestyle interventions remain foundational. Sustained weight loss through diet, exercise, or bariatric surgery consistently reduces AHI and improves cardiometabolic risk. Even modest weight loss of 5-10% can reduce OSA severity. Avoidance of alcohol, sedatives, and smoking further improves airway stability [37]. These approaches are essential adjuncts to device- or drug-based treatments.

TELEMEDICINE AND INTEGRATED CARE

Telemonitoring of CPAP adherence provides real-time feedback to patients and clinicians, improving long-term usage and reducing healthcare burden [32]. Virtual care pathways integrating screening questionnaires, HSAT, remote initiation, and digital adherence support are increasingly feasible, especially in resource-constrained or rural settings. The future likely lies in hybrid models combining telemedicine with precision phenotyping to tailor treatment.

TOWARD PERSONALISED THERAPY

The growing range of therapeutic options underscores the need for personalised, phenotype-driven care. For example, patients with favourable mandibular profiles may benefit from MADs, those with obesity may respond to GLP-1 agonists, while anatomical collapse without PAP tolerance may indicate HNS or MMA. Patient-centred therapy frameworks emphasise shared decision-making, incorporating patient values and treatment preferences to improve adherence and outcomes [37].

Table 2 summarizes established and emerging treatment modalities for obstructive sleep apnoea (OSA), highlighting mechanisms, clinical indications, benefits, limitations, and supporting evidence from recent trials, reviews, and guidelines [15-37].

Table 2: Treatment approaches for Obstructive Sleep Apnoea (OSA)

Therapy	Key Features	Efficacy	Limitations/ Considerations	References
Positive airway pressure (PAP)	CPAP is cornerstone; APAP improves comfort; BiPAP for hypoventilation.	Normalises AHI, reduces BP, improves symptoms; CV benefit with adherence >4h/ night.	Adherence remains major barrier; requires education, telemonitoring.	[15-18,32,38]
Oral appliances (MADs)	Mandibular advancement devices advance jaw to maintain airway patency.	Effective in mild-to-moderate OSA; improve QoL; adherence often superior to CPAP.	Less reduction in AHI than CPAP; dependent on dental health and craniofacial anatomy.	[19,20]
Positional therapy	Prevents supine sleep (tennis-ball, vibration-based devices).	Reduces AHI in positional OSA; comparable QoL to CPAP in selected patients.	Less effective than CPAP; poor long-term adherence common.	[20,35-37]
Hypoglossal nerve stimulation (HNS)	Implanted device stimulates tongue muscles to maintain airway patency.	Significant AHI reduction, improved symptoms, high adherence, durable benefit.	High cost; surgical procedure; limited eligibility (excludes concentric palatal collapse).	[21-23]
Surgical interventions	Includes UPPP, expansion pharyngoplasty,	MMA most effective; long-term	UPPP has variable efficacy; invasiveness and	[30,31]

	maxillomandibular advancement.	improvements in AHI, oxygenation, and systemic outcomes.	relapse risk limit broader use.	
Pharmacotherapy	Wake-promoting agents (solriamfetol, pitolisant); GLP-1 agonists (liraglutide, tirzepatide); trait-targeted drugs (CAIs).	Wake-promoting agents improve sleepiness; tirzepatide reduces AHI >50% in obesity-related OSA.	Wake agents do not treat underlying OSA; pharmacotherapy costly and long-term safety still under study.	[24-29]
Lifestyle and weight management	Diet, exercise, bariatric surgery; avoidance of alcohol, sedatives, smoking.	5-10% weight loss reduces AHI; bariatric surgery and pharmacological weight loss highly effective.	Difficult to sustain lifestyle changes; relapse possible without ongoing support.	[37]
Telemedicine and integrated care	Remote monitoring, digital adherence support, virtual pathways.	Improves adherence, enables scalable care in rural/low-resource settings.	Requires access to technology; disparities remain.	[32]
Toward personalised therapy	Phenotype-driven care, shared decision-making.	Aligns treatment with patient anatomy, comorbidities, and preferences.	Requires multidisciplinary input; evidence base still evolving.	[27,30,37]

DISCUSSION

Obstructive sleep apnoea is increasingly recognized as a systemic disorder rather than an isolated sleep-related breathing problem. The literature emphasizes that its pathophysiology is heterogeneous, with contributions from anatomical, physiological, and metabolic factors that vary between individuals [7-10]. This heterogeneity is mirrored in clinical presentation and therapeutic response, highlighting the limitations of uniform treatment strategies.

In diagnostics, polysomnography remains the reference standard [7], but limitations of cost and accessibility have promoted the use of home sleep apnoea testing [8] and simplified triage tools such as oximetry [12]. Screening questionnaires including STOP-Bang and NoSAS [9,10] are widely adopted, particularly in perioperative and primary care settings, though their low specificity restricts their standalone use.

More recently, hypoxic burden has emerged as a promising marker for cardiovascular risk, outperforming the traditional apnoea-hypopnoea index [13,14]. By incorporating not only event frequency but also the depth and duration of desaturations, hypoxic burden may allow more accurate risk stratification, though further standardization is required before routine clinical use.

Treatment remains complex and highly individualized. Continuous positive airway pressure (CPAP) remains the most effective intervention for reducing respiratory events and improving daytime symptoms [15-18], yet adherence is a persistent challenge, with sustained cardiovascular benefits largely dependent on consistent nightly use [38]. Telemonitoring and behavioural support [32] have been shown to improve adherence and should be incorporated

into clinical practice where possible. Alternatives such as oral appliances [19,20], positional therapy [34-37], hypoglossal nerve stimulation [21,22], and surgical procedures [30,31] provide meaningful options for patients

unable to tolerate CPAP, but each comes with limitations in terms of efficacy, cost, or invasiveness. The expansion of these modalities supports a transition from a single gold standard toward a multimodal model of care.[39]

Pharmacological innovation has added a new dimension to OSA management. Wake-promoting agents such as solriamfetol and pitolisant [24-26] effectively treat residual sleepiness but do not address the underlying disorder.

In contrast, GLP-1 receptor agonists and dual agonists such as tirzepatide demonstrate the ability to reduce both body weight and AHI [27-29], representing a potential disease-modifying therapy in obesity-related OSA. Recent meta-analytic evidence further supports the efficacy of incretin-based therapies for obesity-related OSA [41]. When combined with lifestyle modification and bariatric surgery, these approaches form a spectrum of weight-management strategies that may fundamentally alter disease progression [37].

Across all these developments, the guiding principle is the movement toward precision and integrated care. Patients with positional disease may benefit from targeted positional therapy, those with craniofacial abnormalities may respond best to maxillomandibular advancement, and obese patients may gain the most from pharmacological weight-loss interventions [20,27,30].

Telemedicine [32,40] provides opportunities to improve efficiency, adherence monitoring, and patient education, though disparities in access remain a concern. Collectively, these advances emphasize that treatment selection must be individualized, informed by phenotype, patient preference, and long-term risk considerations.

CRITICAL ANALYSIS AND STUDY LIMITATIONS

Recent literature shows important advances in OSA diagnostics and therapy, but several limitations remain. Many diagnostic innovations-HSAT, overnight oximetry, and wearables -improve accessibility but often underestimate disease severity compared with PSG, particularly in mild OSA or patients with comorbidities [7-12,33]. Hypoxic burden is a promising prognostic metric, yet standardisation and validation across populations and devices are still needed [13,14]. For treatment, CPAP is highly effective, but adherence is variable and long-term cardiovascular benefits inconsistent [15-18,38]. Non-CPAP alternatives, including MADs, positional therapy, HNS, and surgery, are effective in selected patients [19-23,29-31,34,35], but study populations are often small or highly selected, and long-term durability remains uncertain. Pharmacological strategies, such as GLP-1 agonists and tirzepatide, are promising for obesity-driven OSA [23,27,29,41], though evidence is mostly short-term and costly, and applicability to non-obese patients is unclear.

Finally, most evidence comes from high-income countries, limiting generalisability to low-resource settings and highlighting the need for research on equity and accessibility [32,37,40].

In summary, despite significant progress in understanding and managing OSA, substantial uncertainties remain regarding long-term outcomes, optimal patient stratification, and integration of emerging pharmacological and digital approaches into routine care. Future research should prioritise standardisation of novel diagnostic metrics, evaluation of multimodal therapeutic strategies, and equitable access to effective treatments across healthcare systems[42].

CONCLUSION

Obstructive sleep apnoea is a prevalent multifaceted disorder with significant health consequences CPAP remains the cornerstone therapy but adherence limits effectiveness [38]. Expanding options including oral appliances [19 20] positional strategies [34-37] surgical interventions [29-30] hypoglossal nerve stimulation [21-22] and weight-loss therapies [23,27,28,37] supports a shift toward personalized management. Diagnostic innovations particularly hypoxic burden [13,14] and home-based testing [8,11,33] improve risk stratification and prognostication while pharmacological advances especially tirzepatide show promise as disease-modifying therapy in obesity-related OSA [23,27,28,41].

Future research should focus on: standardization of diagnostic metrics for reliable assessment; long-term evaluation of emerging therapies including pharmacological and device-based interventions;equitable access to care to reduce disparities across healthcare systems[42].

DISCLOSURES

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CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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