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REVIEW

Hyperbaric Oxygen Therapy and Its Physio-Mechanical Effects on Sleep Breathing Disorder: A Systematic Review

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ABSTRACT

Introduction: Hyperbaric oxygen therapy (HBOT), traditionally used for decompression sickness and chronic wounds, has recently attracted interest for its potential role in sleep breathing disorders (SBD). Because sleep is highly sensitive to oxygen homeostasis, altered oxygenation can significantly disrupt sleep architecture and efficiency.

Method: This review examines emerging evidence on HBOT's effects in modulating sleep physiology and alleviating major SBD phenotypes, including obstructive sleep apnea (OSA), central sleep apnea (CSA), and high-altitude-related breathing disorders. HBOT may exert therapeutic benefits through several mechanisms—reducing inflammation and oxidative

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stress, reversing tissue hypoxia, improving pulmonary function and oxygenation, enhancing neurocognitive function, modulating arousal threshold and loop gain, and influencing brain regions involved in sleep regulation. These physiological pathways provide a rationale for considering HBOT as an adjunctive or alternative therapy, especially for patient's intolerance of conventional treatments. This systematic review aims to synthesize existing evidence on the effects of HBOT in SBD, particularly OSA, CSA, and altitude-related sleep disturbances.

***Conclusion*:** Current studies offer promising but preliminary evidence supporting HBOT's role in selected SBD populations. However, heterogeneity in protocols, small sample sizes, and limited long-term follow-up constrain interpretation. Future multicenter trials should focus on optimizing treatment pressure, duration, and patient selection, while ensuring safety across vulnerable populations. Understanding the interactions among hyperoxia, neurophysiology, and sleep regulation could unlock novel therapeutic directions for refractory or comorbid SBD.

Keywords: Hyperbaric oxygen therapy; HBOT; OSA; CSA; Sleep breathing disorders; SBD

Key Summary Points

Hyperbaric oxygen therapy (HBOT) has emerged as a potential adjunctive approach for sleep breathing disorders (SBD), including obstructive sleep apnea (OSA), central sleep apnea (CSA), and altitude-related syndromes.

By enhancing tissue oxygenation, reducing inflammation and oxidative stress, and modulating neural and immune responses, HBOT may improve sleep architecture and physiological stability during sleep.

Preliminary studies suggest potential benefits in alleviating hypoxia-related arousals and improving sleep efficiency; however, evidence remains limited by small sample sizes, heterogeneous protocols, and lack of long-term follow-up.

Further well-designed clinical trials are warranted to clarify optimal patient selection, dosing, and safety.

INTRODUCTION

Sleep disorders encompass a broad spectrum of conditions, including insomnia, sleep breathing disorders (SBD), hypersomnolence, circadian rhythm disturbances, parasomnias, and movement-related sleep disorders. Accurate diagnosis is essential for identifying clinical phenotypes and tailoring treatment approaches. As the complexity of sleep disorders has become better understood, individualized or precision-based management has become increasingly important [1, 2].

Among SBD, obstructive sleep apnea (OSA) and central sleep apnea (CSA) represent major clinical challenges. OSA arises from upper airway obstruction influenced by anatomical, neuromuscular, and inflammatory factors, whereas CSA results from central dysregulation of respiratory drive, often associated with heart failure, opioid use, or neurological disease. Continuous positive airway pressure (CPAP) remains the gold standard for OSA,

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while adaptive servo-ventilation (ASV), bilevel positive airway pressure (BiPAP), and supplemental oxygen are recommended for CSA management. The 2025 American Academy of Sleep Medicine (AASM) guidelines underscore the importance of individualized strategies in treating CSA [3].

Hyperbaric oxygen therapy (HBOT)—involving inhalation of 100% oxygen under elevated atmospheric pressure—has long been used for decompression sickness and wound healing but is now gaining attention as a potential adjunct in sleep medicine. By improving oxygen delivery, mitigating intermittent hypoxia, and modulating oxidative and inflammatory pathways, HBOT may alleviate key pathophysiological mechanisms underlying SBD. Preliminary evidence suggests that HBOT can enhance cerebral oxygenation, neuroplasticity, and autonomic regulation, leading to improved ventilatory stability and sleep quality [4]. Moreover, by reducing oxidative stress and optimizing cerebral blood flow, HBOT may support sleep onset and maintenance [5]. Although early studies indicate potential benefits, the clinical use of HBOT for SBD remains experimental. Data are limited by small sample sizes, heterogeneous protocols, and short-term follow-up. Further research is needed to define optimal treatment parameters and evaluate long-term outcomes.

Therefore, this systematic review aims to synthesize existing evidence on the effects of HBOT in SBD, particularly OSA, CSA, and altitude-related sleep disturbances. The review focuses on underlying physiological mechanisms, potential therapeutic roles, and future directions for integrating HBOT into personalized sleep medicine. Searches were conducted across PubMed/MEDLINE, Embase, Web of Science, Scopus, Cochrane CENTRAL, and ClinicalTrials.gov, using controlled terms related to HBOT and sleep disorders. Eligible studies included human clinical trials reporting sleep-related outcomes following HBOT, while non-HBOT oxygen studies, animal data, and incomplete abstracts were excluded. This article is based on previously conducted studies and does not contain any new studies with human participants or animals performed by any of the authors.

OVERVIEW ON MECHANISMS OF HYPERBARIC OXYGEN THERAPY

HBOT originated in the seventeenth century [6], starting with early trials in pressurized chambers, but it was not until the twentieth century that it was recognized for medical applications [7]. In 1962, Sharp and Smith (Scotland) were the first to employ HBOT in the treatment of carbon monoxide poisoning [8]. The following year, in 1963, Hitchcock testified before the US House Committee on Labor, Health, Education, and Welfare, advocating for the integration of hyperbaric chambers into surgical practice, which led to congressional appropriation of funds for the construction of approximately 20 hyperbaric chambers [9, 10].

HBOT is provided in a specially designed chamber that may be either monoplace (for one individual) or multiplace (for several patients). These chambers are pressurized to levels generally ranging from 1.5 to 3 times the standard atmospheric pressure at sea level [11]. Throughout the treatment, patients inhale 100% oxygen, which significantly boosts the amount of oxygen that dissolves in the blood plasma. These mechanisms highlight HBOT's role in facilitating tissue regeneration, reducing inflammation, and fighting infections in a variety of clinical contexts. In sleep disorders, HBOT may improve sleep disorders by enhancing oxygenation in the brain, reducing inflammation, and potentially stabilizing autonomic function, which can benefit conditions like OSA and insomnia (Fig. 1). Research has also revealed that HBOT exerts its effects on SBD through multiple physiological mechanisms, including the reduction of inflammation and oxidative stress, reversal of tissue hypoxia, modulation of cytokines and immune response, improvement in sleep architecture, reduction of sleep-related pain and fatigue, effects on brain regions involved in sleep regulation, improvement in pulmonary function and oxygenation, reduction of arousal threshold and loop gain, and effects on psychological symptoms [5, 12–17]. These mechanisms contribute to improved sleep quality and duration, making HBOT a promising therapeutic option for various SBD.

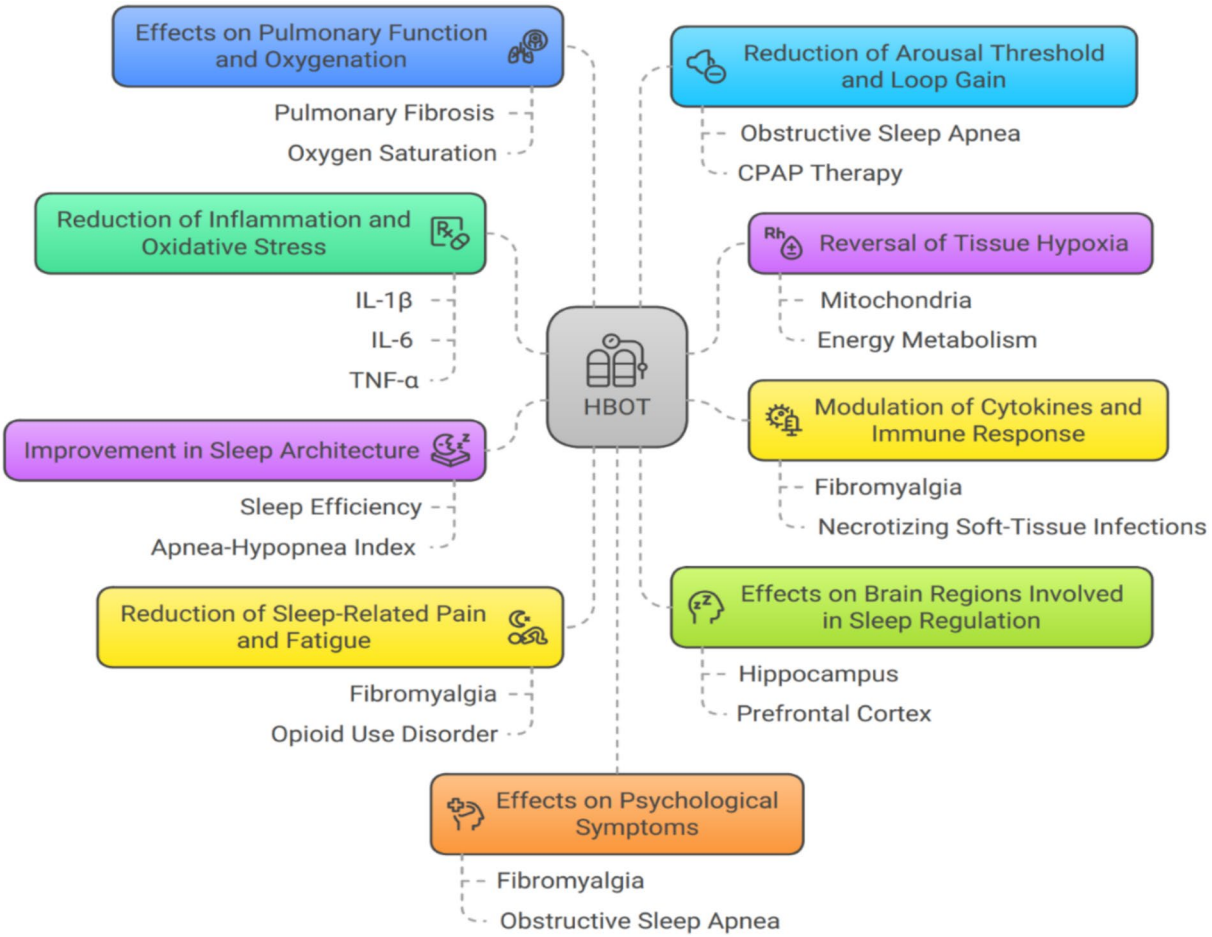

Fig. 1 Mechanisms of HBOT in improving sleep breathing disorders. HBOT may improve sleep by boosting oxygenation and stabilizing ventilatory control, reducing inflammation/oxidative stress, and reversing tissue hypoxia to support mitochondrial energy metabolism; HBOT may also modulate immune signaling and enhance perfusion/neuroplasticity in sleep-related brain regions, which can translate into better sleep architecture, less pain/fatigue, and improved psychological symptoms in select conditions. *CPAP* continuous positive airway pressure, *HBOT* hyperbaric oxygen therapy, *IL-1β* interleukin-1 beta, *IL-6* interleukin-6, *TNF-α* tumor necrosis factor alpha

HBOT is generally safe, well tolerated, and has minimal side effects. In a study on patients with fibromyalgia, HBOT was associated with mild side effects like middle-ear barotrauma and new-onset myopia, but these were not severe enough to discontinue therapy [14, 15]. In another study on patients with opioid use disorder, HBOT was found to be feasible and acceptable, with high patient satisfaction and minimal side effects [18].

HYPERBARIC OXYGEN THERAPY AND OBSTRUCTIVE SLEEP APNEA

OSA is a common sleep disorder that affects nearly billions globally, frequently without a diagnosis [19]. It is characterized by recurrent episodes of partial or complete upper airway collapse during sleep, leading to intermittent hypoxia and sleep fragmentation [20]. OSA has

been correlated with a heightened prevalence of hypertension, type 2 diabetes mellitus, atrial fibrillation, heart failure, coronary artery disease, cerebrovascular accidents, and mortality [21–24].

Pathophysiology of Obstructive Sleep Apnea

OSA is a complex sleep disorder characterized by recurrent episodes of upper airway obstruction during sleep, leading to respiratory pauses, oxygen desaturation, and sleep fragmentation [1]. The pathophysiology of OSA involves a combination of anatomical, neurological, and genetic factors, which interact to predispose individuals to airway collapse. Duong-Quy et al. [1] suggested that the pathophysiology of OSA involves both anatomical and non-anatomical factors contributing to upper airway obstruction during sleep. The primary cause of OSA, in terms of its physiopathology, is generally attributed to anatomical abnormalities of the laryngopharynx. These abnormalities induce the narrowing or complete obstruction of the upper airway while a person is sleeping.

The upper airway anatomy plays a critical role in the pathophysiology of OSA (Fig. 2). A narrow oropharynx, often due to abnormal craniofacial anatomy or soft tissue accumulation in the neck, increases the likelihood of airway collapse during sleep [25]. Obesity, a major risk factor for OSA, contributes to fat deposition around the upper airway, further narrowing the airway lumen [26]. Furthermore, excess soft tissue in the neck and tongue can encroach on the upper

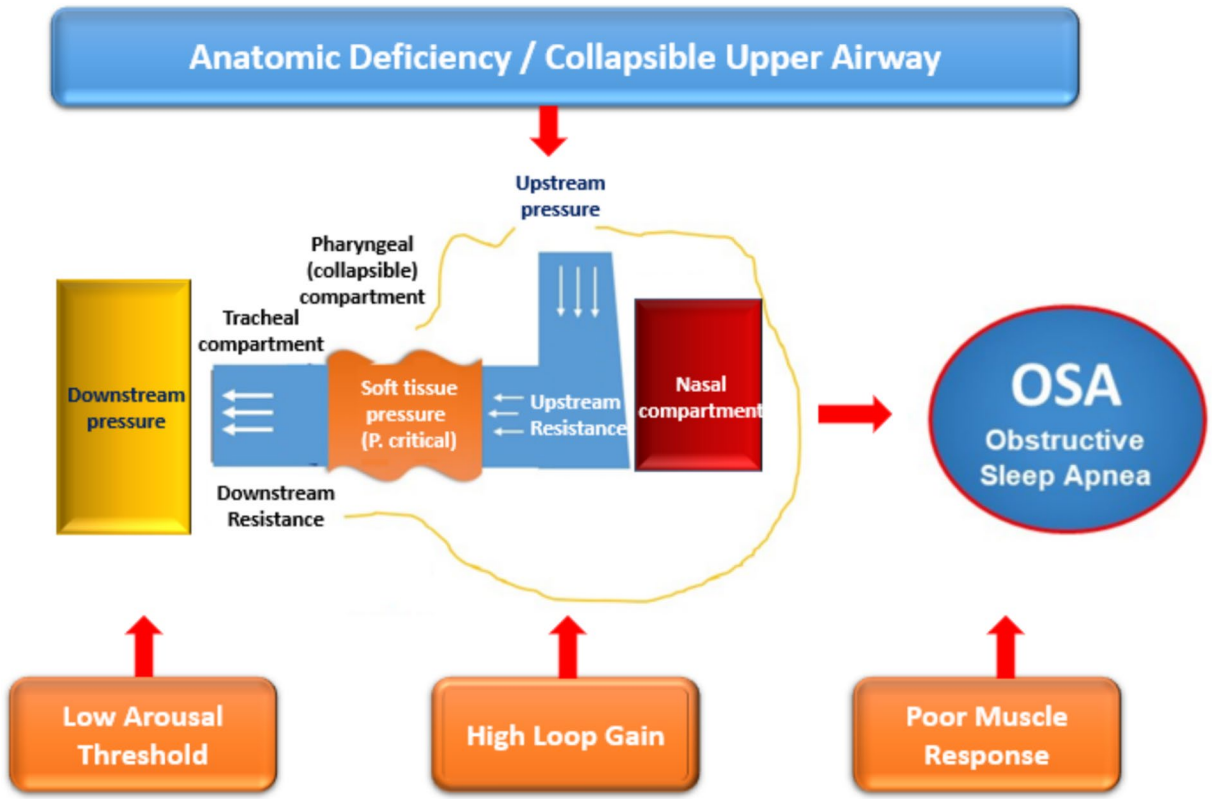

Fig. 2 Anatomical and non-anatomical factors of OSA. OSA arises when a collapsible upper airway—influenced by nasal/pharyngeal upstream resistance and soft-tissue critical closing pressure (Pcrit)—narrows under inspiratory load, while non-anatomical traits (low arousal threshold, high loop gain/unstable ventilatory control, and poor pharyngeal muscle responsiveness) further destabilize breathing and precipitate obstructive events. *OSA* obstructive sleep apnea

airway, reducing its cross-sectional area (Fig. 2). This is particularly evident in individuals with obesity, where fat accumulation in the pharyngeal region exacerbates airway collapsibility [25, 26]. Moreover, in the recumbent position, fluid shifts from the lower extremities to the neck, increasing soft tissue volume around the upper airway. This rostral fluid shift is a significant contributor to airway narrowing and collapse during sleep [25]. The genioglossus muscle, a key upper airway dilator, plays a crucial role in maintaining airway patency. However, during sleep, the responsiveness of this muscle is reduced, impairing its ability to counteract collapsing forces [25]. Additionally, ventilatory instability, loop gain, and inadequate muscle compensation further destabilize the upper airway [25, 27]. The arousal threshold, or the level of respiratory disturbance required to awaken from sleep, is another important factor. A low arousal threshold can lead to frequent awakenings, disrupting sleep quality and perpetuating the cycle of airway obstruction and arousal [27]. Besides, ventilatory control instability, often manifesting as high loop gain, can lead to cyclical breathing patterns. This instability amplifies the severity of apneas and hypopneas, creating a vicious cycle of airway collapse and respiratory effort [25, 27] (Fig. 2).

Recurrent episodes of upper airway obstruction lead to intermittent hypoxia, a hallmark of OSA. Intermittent hypoxia triggers systemic inflammation, oxidative stress, and sympathetic activation (Fig. 3), contributing to the development of comorbidities such as cardiovascular and metabolic disorders [26, 28]. Frequent

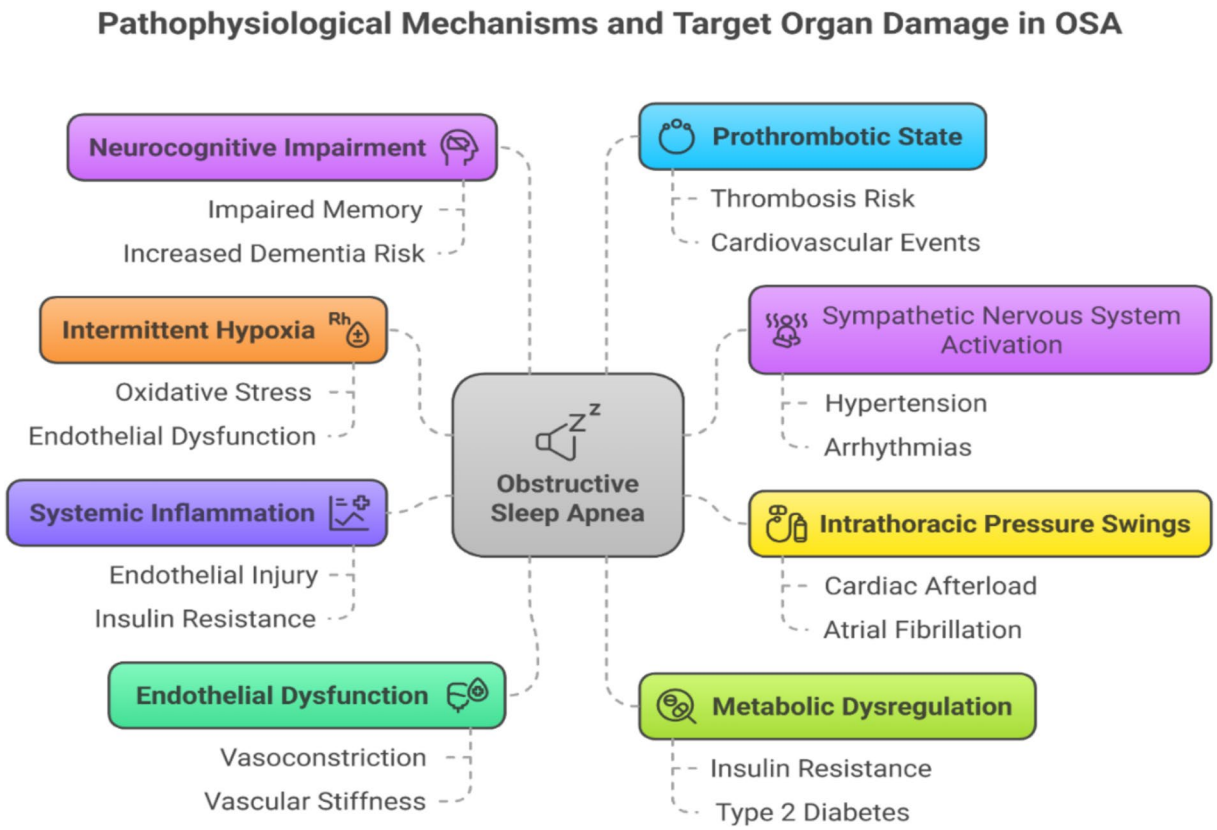

Fig. 3 Pathophysiological mechanism and target organ damage in OSA. Recurrent airway obstruction in OSA triggers intermittent hypoxia, sympathetic surges, inflammation, prothrombotic state, endothelial dysfunction, metabolic dysregulation, and large intrathoracic pressure swings; these pathways converge to drive hypertension/arrhythmias, thrombosis and cardiovascular events, insulin resistance/type 2 diabetes, and neurocognitive impairment—explaining the multisystem harm of untreated OSA. *OSA* obstructive sleep apnea

arousals disrupt normal sleep architecture, leading to sleep fragmentation. This fragmentation impairs sleep quality and exacerbates daytime sleepiness, a common symptom of OSA (Fig. 3). Recent evidence highlights the role of inflammatory pathways in the pathogenesis of OSA. Chronic intermittent hypoxemia and mechanical stress on the upper airway induce low-grade inflammation, further destabilizing the airway and perpetuating the disease [29, 30].

Physiological Mechanisms of Hyperbaric Oxygen Therapy in Obstructive Sleep Apnea

Intermittent hypoxia is a hallmark of OSA, leading to oxidative stress, systemic inflammation, and cardiovascular dysfunction [23]. HBOT, by increasing oxygen availability, can mitigate the effects of hypoxia (Fig. 4). Studies have shown that supplemental oxygen therapy reduces the severity of OSA by decreasing the apnea–hypopnea index (AHI) and improving oxygen saturation levels during sleep [3, 31, 32]. Similarly, HBOT may enhance oxygen delivery to the upper airway muscles, potentially reducing the frequency of apneic events (Fig. 4). On the other hand, loop gain is a key physiological trait in OSA, representing the ventilatory response to changes in oxygen and carbon dioxide levels. Elevated loop gain can exacerbate ventilatory instability, leading to more severe apneas.

Research indicates that hyperoxia, such as that achieved through HBOT, can reduce loop gain by stabilizing breathing patterns and decreasing the ventilatory overshoot following an apnea [11, 33]. This mechanism suggests that HBOT could help reduce the severity of OSA by improving ventilatory stability (Fig. 4). Moreover, upper airway collapsibility is another critical factor in OSA. HBOT may influence this by increasing the stiffness of the upper airway muscles, thereby reducing the likelihood of collapse during sleep. While direct

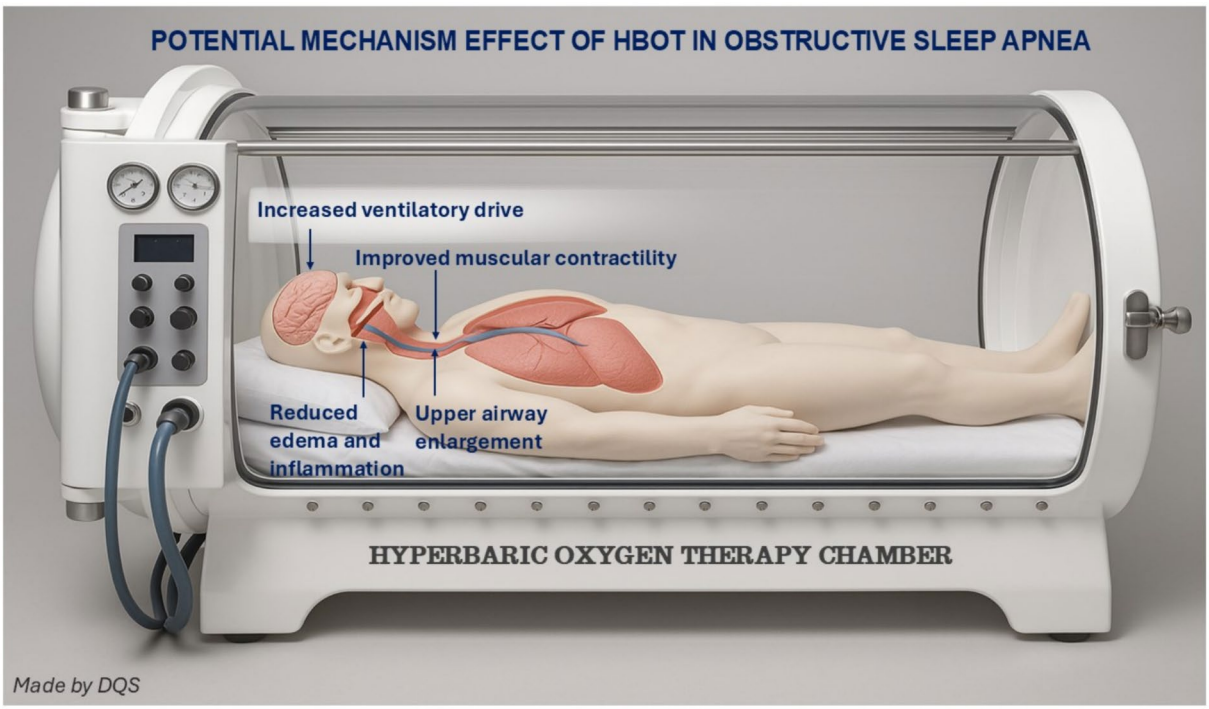

Fig. 4 Proposed mechanisms of hyperbaric oxygen therapy in alleviating OSA. HBOT may reduce upper airway collapsibility: higher dissolved oxygen may stabilize ventilatory drive, enhance upper airway dilator muscle contractility, reduce mucosal edema and inflammation, and transiently increase airway caliber. Together these effects could lessen apnea–hypopnea burden. *HBOT* hyperbaric oxygen therapy, *OSA* obstructive sleep apnea

evidence on HBOT's effects on upper airway collapsibility is limited, studies on supplemental oxygen therapy have shown that improved oxygenation can enhance upper airway muscle tone, potentially reducing collapsibility [33, 34]. HBOT may increase the arousal threshold by reducing the frequency of hypoxia-induced arousals, allowing for more stable sleep and fewer interruptions (Fig. 4). This effect has been observed in studies using nocturnal oxygen therapy, where improved oxygenation led to fewer arousals and better sleep quality [11, 35].

Effects of Hyperbaric Oxygen Therapy on Obstructive Sleep Apnea

First, studies on oxygen therapy have consistently shown a significant reduction in AHI with supplemental oxygen administration [31–33]. HBOT, by providing a higher concentration of oxygen, may further enhance this effect and might mitigate the adverse effects of hypoxia on the cardiovascular system. By reducing the frequency of apneic events and improving oxygenation, HBOT may lead to more consolidated sleep and improved sleep quality, similar to its effects on sleep disorders in other diseases [34–36]. Research on oxygen therapy has identified specific physiological traits such as elevated loop gain, increased upper airway collapsibility, and reduced compensation (the ability to maintain ventilation in response to upper airway obstruction) that predict responsiveness to treatment of OSA [37–40]. Patients with these traits may be more likely to experience a significant reduction in AHI and improvement in sleep quality with HBOT. Thus, HBOT might offer a potential alternative for patients who cannot tolerate CPAP, or are refractory to other therapies, owing to its effect on improving oxygenation and modulating respiratory control together with the use of nocturnal oxygen therapy [39].

HYPERBARIC OXYGEN THERAPY AND CENTRAL SLEEP APNEA

Although HBOT is not a proven treatment for CSA, it can reduce hypoxemia during treatment sessions and raise dissolved oxygen. The optimization of guideline-directed medical therapy for heart failure, the identification and treatment of underlying drivers (such as heart failure, stroke, and opioids), and the consideration of positive airway pressure modalities (CPAP, bilevel, adaptive servo-ventilation) or nocturnal supplemental oxygen in certain cases are the main goals of in-center management of CSA in clinical practice [23, 41]. HBOT is still an unconventional treatment for CSA and can be argued as a physiological justification for oxygenation and respiratory control.

Although HBOT is not a proven treatment for CSA, it can reduce hypoxemia and raise dissolved oxygen levels during treatment sessions. In clinical management, the primary goals include optimizing therapy for underlying causes (heart failure, stroke, opioids) and considering positive airway pressure (CPAP, bilevel, adaptive servo-ventilation) or nocturnal oxygen in specific cases [23, 41]. HBOT remains unconventional but provides physiological justification for improving oxygenation and respiratory control.

Pathophysiology of Central Sleep Apnea

CSA is characterized by recurrent episodes of airflow cessation due to a temporary reduction or absence of central respiratory drive. The underlying physiological mechanisms of CSA involve a complex interplay of factors, including ventilatory control instability, chemoreflex sensitivity, and the balance between excitatory and inhibitory influences on respiration. These mechanisms can be broadly categorized into those related to excessive respiratory drive and those associated with inadequate drive.

The pathophysiology of CSA is multifactorial, involving complex interactions between chemoreflex sensitivity, ventilatory control stability, circulatory dynamics, and neurological integrity. CSA is fundamentally linked to ventilatory control instability, where fluctuations in respiratory drive lead to periods of apnea and hyperventilation [42]. The transition from wakefulness to sleep involves changes in respiratory control, which can destabilize breathing patterns and contribute to CSA [42]. Moreover, chemoreflex

sensitivity plays a critical role in CSA, particularly the sensitivity to carbon dioxide levels. A drop in arterial carbon dioxide below the apneic threshold can trigger CSA [43]. The concept of the carbon dioxide reserve quantifies the propensity to develop CSA, with a lower reserve indicating a higher risk.

Loss of wakefulness drive to breathe and diminished chemosensitivity during sleep, especially during rapid eye movement (REM) sleep, can exacerbate CSA [43]. CSA and OSA often coexist, with each condition potentially predisposing to the other. This overlap in ventilatory instability, high loop gain, and arousal-threshold variability also appears in high-altitude periodic breathing, linking the central and environmental forms of SBD [44]. CSA can lead to significant cardiorespiratory dysfunction and sleep disruption [45], necessitating a comprehensive approach to management that addresses both the underlying pathophysiology and the resultant health consequences.

Mechanism and Efficacy of Hyperbaric Oxygen Therapy in Central Sleep Apnea

HBOT has shown potential in treating CSA through its neuromodulatory and oxygen-stabilizing effects, although direct evidence specific to CSA is limited (Fig. 5). HBOT is known to enhance neuroplasticity and brain recovery by modulating cellular and molecular mechanisms [46–49], which could theoretically benefit patients with CSA by improving brain function and reducing apnea events. Clinical trials have demonstrated that HBOT can improve cognitive, psychiatric, fatigue, and sleep symptoms in patients with long COVID, highlighting its neuromodulatory potential and relevance to sleep regulation [50].

HBOT promotes mitochondrial biogenesis and function, stimulates neurogenesis and synaptogenesis, and reduces inflammation [49]. These effects may enhance neural control of respiration and complement the ventilatory-stabilizing

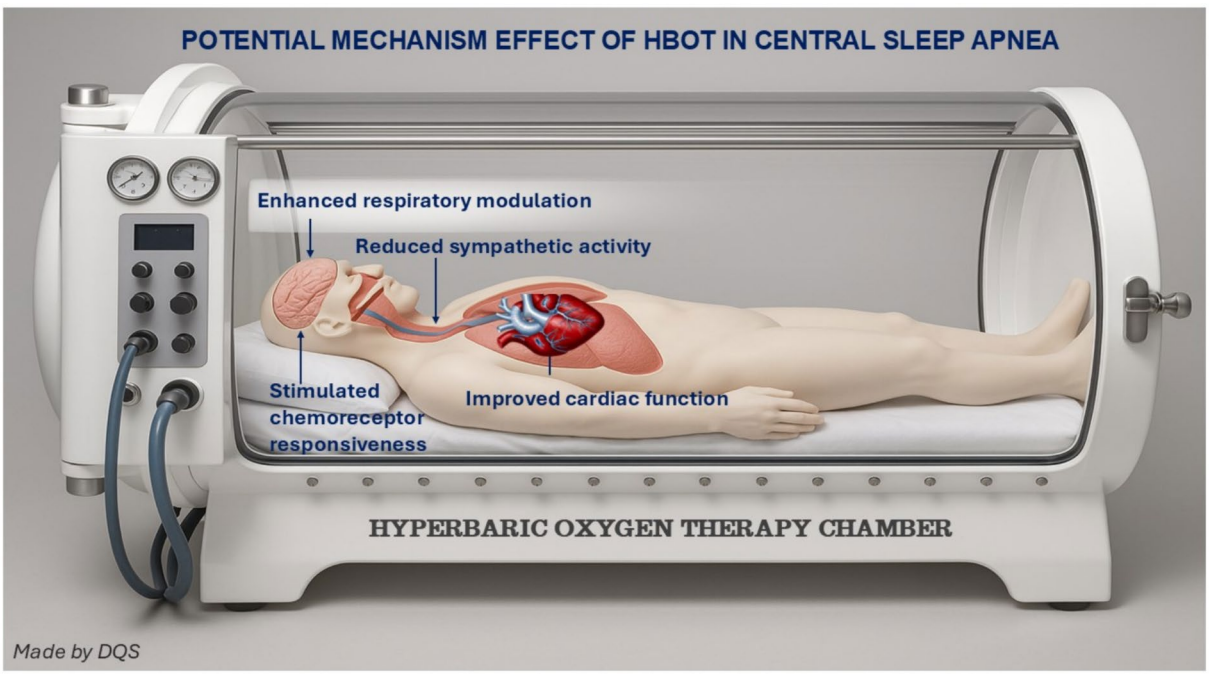

Fig. 5 Mechanism of OSA in treating central sleep apnea. HBOT might stabilize breathing in CSA by enhancing respiratory modulation and smoothing of ventilatory oscillation, stimulating peripheral/central chemoreceptor responsiveness, improving feedback control, reducing sympathetic activity, and improving cardiac function. *CSA* central sleep apnea, *HBOT* hyperbaric oxygen therapy

role of oxygen therapy observed in OSA and altitude-related breathing disorders. By improving oxygenation, reducing loop-gain-related oscillations, and decreasing sympathetic overactivity, HBOT may help maintain a steadier ventilatory pattern and higher arousal threshold (Fig. 5).

HYPERBARIC OXYGEN THERAPY AND SLEEP BREATHING DISORDERS AT HIGH ALTITUDE

Currently, several million people permanently reside in high-altitude mountain regions, and an increasing number of people travel to higher altitudes for work and pleasure worldwide. Exposure to high altitudes (generally exceeding 2500 m) can elicit considerable sleep disruptions primarily because of hypobaric hypoxia. Frequently observed phenomena include sleep fragmentation, periodic breathing, and diminished slow-wave sleep [51]. When at high altitude, lowlanders will not be able to exert themselves as much as they could at sea level. Fortunately, a series of physiological adjustments known as acclimatization allow the human body to adapt to this hypoxia [52]. HBOT plays a significant role in addressing SBD at high altitudes by improving oxygen saturation and sleep quality.

Adaptation of Oxygenation at High Altitude

The barometric pressure exhibits an exponential decline as altitude increases. The inspired and arterial partial pressures of oxygen consequently drop, affecting nearly all organ systems [53]. At high altitude, the body undergoes physiological adjustments to counter hypobaric hypoxia: breathing becomes deeper and faster, cardiac output rises, and red blood cell production increases to enhance oxygen transport. Over time, capillary density and mitochondrial efficiency improve, allowing tissues to better utilize limited oxygen. Yet, despite these adaptations, arterial oxygen saturation seldom returns to sea-level levels, resulting in periodic breathing, sleep fragmentation, and reduced restorative sleep. Persistent hypoxemia may provoke CSA or altitude-related breathing instability—mechanistically similar to the ventilatory instability and loop-gain fluctuations observed in CSA. Understanding these adaptive and maladaptive processes provides a physiological rationale for applying hyperbaric or oxygen-enriched therapies to restore sleep stability and oxygen homeostasis.

High-Altitude Periodic Breathing During Sleep

Periodic breathing is a hallmark of high-altitude sleep, yet the same waxing–waning ventilatory cycles can also emerge during wakefulness and exertion. The apneic intervals associated with periodic breathing may manifest as an alarming sensation of asphyxiation, succeeded by several deep inhalations characterized by gasping for oxygen [54, 55]. Although it diverges from the typical crescendo–decrescendo pattern of Cheyne–Stokes respiration [55], periodic breathing at high altitudes resembles the erratic respiratory patterns associated with CSA and other high-loop-gain states [56]. The absence of the stabilizing wakefulness drive renders periodic breathing more prevalent during non-REM sleep. Additionally, hypercapnia is linked to a decreased ventilatory response compared with wakefulness [57–59]. Thus, altitude-related periodic breathing shares key physiological mechanisms—loop-gain elevation, ventilatory instability, and arousal-threshold variability—with CSA.

Benefits of HBOT on SBD at High Altitudes

Oxygen enrichment at high altitude increases the time spent in slow-wave sleep and improves arterial oxygen saturation (from 84.3% to 92.7%), reducing the oxygen-desaturation index and improving sleep quality [60]. At extreme simulated altitudes, HBOT helped maintain higher arterial oxygen saturation levels, which correlated with fewer sleep arousals and improved sleep continuity [61].

Simulated descent in a hyperbaric chamber decreased periodic breathing and body position changes, further indicating improved sleep quality [60]. While HBOT shows promise in

alleviating SBD at high altitudes, it is essential to consider individual variability in response to therapy. Factors such as acclimatization, individual health conditions, and the specific altitude may influence the effectiveness of HBOT (Fig. 6). Recent reviews also suggest that HBOT preconditioning may enhance systemic oxygenation and resilience to high-altitude hypoxia, providing a theoretical basis for its potential use in SBD at altitude [62]. Further research could explore these variables to optimize treatment protocols for diverse populations.

Mechanisms of HBOT in SBD at High Altitude

HBOT augments the partial pressure of oxygen within plasma, thereby enhancing tissue oxygenation even in conditions of low ambient oxygen pressure [63]. At high altitude, HBOT may improve sleep by stabilizing ventilatory control loops, attenuating hypoxia-related instability in the arousal threshold, and enhancing cerebral oxygenation with downstream benefits for sleep architecture [64].

HBOT offers a promising approach to improving sleep quality in individuals exposed to high altitude. While preliminary evidence supports its effectiveness in reducing sleep fragmentation and breathing disturbances, broader clinical trials are necessary before it can be widely recommended as a standard intervention.

CONCLUSION

HBOT offers a promising approach to managing OSA, CSA, and sleep disturbance at high altitude by addressing the physiological mechanisms underlying the disorder. By reducing intermittent hypoxia, modulating loop gain, and improving oxygen saturation, HBOT may

Fig. 6 The first author pictured next to a patient undergoing HBOT at Lao Cai Traditional Hospital (Lao Cai is a high-altitude mountainous province of Vietnam). *HBOT* hyperbaric oxygen therapy. The photo is used with permission from the Lao Cai Traditional Hospital (license number BVYHCT-LC/24.10/25)

Table 1 Summary of representative studies on hyperbaric oxygen therapy and oxygen-based interventions in sleep breathing disorder

Study (Ref. no.)	Design and sample	Population/setting	Main outcomes	Key conclusion
Przybyłowski et al., 2003 [60]	Observational field study ($n = 24$)	Gold-mine workers (3800–4200 m) undergoing simulated 2000 m descent in hyperbaric chamber	SaO_2 increased from 84.3 ± 3.2% to 92.7 ± 2.8%; reduced ODI and arousals	Simulated descent significantly improved nocturnal oxygenation and sleep quality under hypobaric conditions
Walker et al., 2018 [34]	RCT ($n = 50$)	US service members with post-concussive mTBI	HBOT increased sleep efficiency and total sleep time vs. sham	HBOT enhanced restorative sleep, suggesting neuromodulatory benefit
Shi et al., 2024 [35]	Systematic review/protocol	Post-stroke insomnia	Planned evaluation of HBOT vs. standard care on ISI, PSQI	HBOT expected to improve post-stroke sleep via neurovascular repair
Tan et al., 2024 [36]	Meta-analysis of 7 studies	Parkinson's disease with sleep disorders	HBOT reduced sleep latency; increased sleep efficiency	Adjunct HBOT improved motor and sleep outcomes with good tolerability
Hadanny et al., 2022 [50]	RCT ($n = 73$)	Patients with long COVID	Improved fatigue, cognition, and sleep quality scores	HBOT restored neurocognitive and sleep functions via enhanced oxygenation
Efrati et al., 2013 [46]	Randomized, prospective trial ($n = 74$)	Patients post stroke	HBOT induced late neuroplasticity; increased cognitive and functional scores	Supports mechanism for CSA improvement via brain-stem plasticity
Ahmadi et al., 2021 [48]	Mechanistic review	Preclinical and clinical HBOT data	HBOT increased mitochondrial function and neurogenesis; reduced inflammation	Provides molecular basis for HBOT effects in central apnea
Bin-Alamer et al., 2024 [49]	Narrative review	Neuromodulatory applications of HBOT	Summarized recent neurological findings	Confirms HBOT stabilizes autonomic and respiratory control loops

Table 1 continued

Study (Ref. no.)	Design and sample	Population/setting	Main outcomes	Key conclusion
Phyu et al., 2024 [31]	Systematic review/meta-analysis	OSA; nocturnal oxygen therapy	Oxygen therapy reduced AHI significantly vs. baseline	Oxygen supplementation reduces hypoxic burden; HBOT may potentiate this effect
You et al., 2023 [62]	Review and experimental synthesis	High-altitude exposure and HBOT preconditioning	Increased SaO_2; reduced hypoxia-induced inflammation	HBOT preconditioning mitigates altitude illness and improves acclimatization

AHI apnea–hypopnea index, *COVID* coronavirus disease, *HBOT* hyperbaric oxygen therapy, *ISI* insomnia severity index, *mTBI* mild traumatic brain injury, *ODI* oxygen desaturation index, *PSQI* Pittsburgh sleep quality index, *RCT* randomized controlled trial, SaO_2 arterial saturation of oxygen, *SBD* sleep breathing disorder, *US* United States

lead to significant improvements in AHI, sleep quality, and cardiovascular outcomes, along with reducing sleep fragmentation and breathing disturbances.

Limitations and Future Directions

This systematic review is susceptible to selection and publication bias because it lacked a formal risk-of-bias assessment, meta-analysis, and preregistered protocol [65–73]. In addition to relying on small, single-center or uncontrolled studies, the underlying HBOT literature is diverse in terms of pressure, session length, and treatment duration. The literature also commonly presents surrogate results rather than long-term clinical objectives. Generalizability is limited by co-interventions and patient-status variations; safety reporting is inconsistent, particularly for susceptible populations and related to the equipment.

While the current evidence suggests that HBOT may be beneficial for patients with OSA (Table 1), further research is needed to fully understand its effects. Long-term studies are required to assess the sustainability of benefits and the potential for HBOT to be used as a standalone or adjunct therapy. Additionally, studies should focus on identifying the optimal dosing and duration of HBOT for OSA, CSA, and sleep disturbance at high altitude as well as the patient populations most likely to benefit from this treatment therapy after specific diagnosis of SBD in different populations [74].

Author Contribution. Sy Duong-Quy: conceptualization; methodology; writing—original draft; writing—review and editing; visualization (figure development). Thai Nguyen-Duy: methodology; data curation; literature search; writing—original draft. Tran V. Hoc: methodology; data curation; literature search; writing—original draft. Tram Tang-Thi-Thao: literature search; data curation; references management; writing—review. Toi Nguyen-Van: literature search; evidence synthesis; writing—review. Tuan Huynh-Anh: visualization (schematics/figure refinement); writing—review. Trung Mai-Xuan: critical revision of clinical/physiology

content; writing—review. Phap Tran-Quang: domain expertise (pulmonary/oxygenation); writing—review. Viet Nguyen-Ba: methodology (evidence synthesis); validation; editing. Bang Nguyen-Trong: quality appraisal; references verification; editing. Thu Nguyen-Ngoc-Phuong: clinical implications; writing—review. Hai Doan-Ngoc: clinical implications; language polishing; review and editing. Giap Vu-Van: clinical implications; language polishing; writing—review and editing. Nhung Nguyen-Viet: supervision; validation; final approval of the version to be published. Franck Soyez: supervision; validation; final approval of the version to be published. Francis Martin: supervision; validation; final approval of the version to be published. Thomas Penzel: supervision; validation; final approval of the version to be published. Clete Kushida: supervision; validation; final approval of the version to be published. Timothy Craig: supervision; validation; editing; final approval of the version to be published.

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Declarations

Conflict of Interest. Sy Duong-Quy, Thai Nguyen-Duy, Tran V. Hoc, Tram Tang-Thi-Thao, Toi Nguyen-Van, Tuan Huynh-Anh, Trung Mai-Xuan, Phap Tran-Quang, Viet Nguyen-Ba, Bang Nguyen-Trong, Thu Nguyen-Ngoc-Phuong, Hai Doan-Ngoc, Giap Vu-Van, Nhung Nguyen-Viet, Franck Soyez, Francis Martin, Thomas Penzel, Clete Kushida, and Timothy Craig have not any conflicts of interest related to this study to declare. The authors confirm that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest. Sy Duong-Quy and Timothy Craig are members of the Editorial Board of *Pulmonary Therapy*. They were not involved in the selection of peer reviewers for this manuscript nor in any of the subsequent editorial decisions.

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