

Obstructive Sleep Apnea: A Prosthodontic Overview

Deepali Neeraj

Assistant Professor, School of Dental Sciences, Sharda University

1. Introduction

Section 1: Fundamentals of Sleep Physiology and Sleep Disorders

Sleep is a complex, regulated, and essential physiological process that plays a critical role in systemic restoration, neurocognitive functioning, metabolic homeostasis, and immunological balance. Sleep is divided into two main phases: non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep. These phases occur in cycles, each lasting about 90 to 110 minutes, and repeat throughout the night. The dynamic alternation between these phases contributes to overall sleep quality and health maintenance.

NREM sleep is subdivided into three distinct stages—N1, N2, and N3. Stage N1, the lightest stage of sleep, marks the transition from wakefulness to sleep. It is characterized by a decrease in muscle tone, slow eye movements, and reduced brain wave activity. Stage N2 represents a deeper phase of light sleep and constitutes approximately 50% of total sleep time in adults. Sleep spindles and K-complexes are hallmark features of this stage and are believed to be involved in sensory processing and memory consolidation. Stage N3, also referred to as slow-wave sleep or deep sleep, is marked by the presence of high-amplitude, low-frequency delta waves on electroencephalographic (EEG) recordings. This stage is crucial for tissue repair, immune function, and the release of growth hormone.

REM sleep comprises about 20–25% of total sleep time and is distinguished by rapid eye movements, vivid dreaming, increased brain metabolic activity, and muscle atonia—an adaptive mechanism preventing individuals from physically acting out their dreams. REM sleep is critical for cognitive processes such as learning, memory consolidation, and emotional regulation.

The sleep-wake cycle is governed by circadian rhythms, which are endogenous biological cycles that approximate 24 hours. These rhythms are regulated by the suprachiasmatic nucleus (SCN) in the hypothalamus. The SCN synchronizes bodily functions with environmental cues, primarily light and darkness. Light exposure inhibits melatonin secretion by the pineal gland, promoting wakefulness, while darkness stimulates its release, facilitating sleep onset. Disruptions to circadian rhythms, as seen in shift workers, travelers across time zones, or individuals with irregular sleep patterns, can lead to circadian rhythm sleep-wake disorders.

The International Classification of Sleep Disorders, Third Edition (ICSD-3), classifies sleep disorders into six major categories: insomnia, sleep-related breathing disorders, central disorders of

hypersomnolence, circadian rhythm sleep-wake disorders, parasomnias, and sleep-related movement disorders. Each category encompasses specific diagnoses based on clinical criteria, duration, and impact on daytime function. Among these, sleep-related breathing disorders, particularly obstructive sleep apnea (OSA), have garnered considerable clinical and research interest due to their high prevalence and association with systemic comorbidities.

OSA is characterized by repetitive episodes of partial or complete upper airway obstruction during sleep. These episodes lead to intermittent hypoxemia, hypercapnia, arousals, and sleep fragmentation. The resulting physiological stress activates the sympathetic nervous system, leading to increased cardiovascular morbidity. The prevalence of OSA is estimated to be 9–38% in the general population, with higher rates observed in males, older adults, and individuals with obesity or craniofacial abnormalities.

The severity of OSA is quantified using the Apnea-Hypopnea Index (AHI), defined as the number of apnea and hypopnea events per hour of sleep. The classification is as follows:

- Mild OSA: AHI of 5–15 events/hour
- Moderate OSA: AHI of 15–30 events/hour
- Severe OSA: AHI >30 events/hour

Polysomnography, the gold standard for diagnosing sleep disorders, involves the simultaneous recording of various physiological parameters including EEG, electrooculography (EOG), electromyography (EMG), nasal airflow, thoracoabdominal movements, oxygen saturation, and electrocardiography (ECG). From this data, various indices are calculated, including the AHI, oxygen desaturation index (ODI), and arousal index. These scoring systems are standardized by the American Academy of Sleep Medicine (AASM) to ensure diagnostic reliability.

Sleep hygiene encompasses behavioral and environmental strategies that promote optimal sleep quality. This includes maintaining a consistent sleep schedule, limiting caffeine and alcohol intake, engaging in regular physical activity, and creating a quiet, dark, and cool sleep environment. Poor sleep hygiene can contribute to the development of insomnia and exacerbate other sleep disorders.

Dental professionals, particularly prosthodontists, are uniquely positioned to identify and manage patients with sleep-disordered breathing. Oral manifestations of OSA may include bruxism, scalloped tongue, high-arched palate, retrognathia, and Class II malocclusion. These findings should prompt further investigation and possible referral for polysomnography. Prosthodontists play a vital role in the fabrication and titration of mandibular advancement devices (MADs), which function by repositioning the mandible anteriorly, thereby increasing upper airway patency during sleep.

MADs are custom-fitted oral appliances that have demonstrated efficacy in the treatment of mild to moderate OSA and in patients intolerant to continuous positive airway pressure (CPAP) therapy. Their success depends on proper patient selection, appliance design, and adherence to follow-up protocols. Side effects such as temporomandibular joint discomfort, excessive salivation, or occlusal changes may occur, requiring prosthodontic management.

This section provides a foundational understanding of sleep physiology, classification of sleep disorders, the clinical significance of the AHI index, and the role of dental professionals in the management of OSA. Subsequent sections will delve into the epidemiology, risk factors, diagnostic criteria, and therapeutic modalities used in the management of obstructive sleep apnea from a prosthodontic perspective.

2 Sleep Architecture and Stages

Sleep comprises two principal stages:

- **Non-Rapid Eye Movement (NREM) Sleep**
- **Rapid Eye Movement (REM) Sleep**

These stages alternate cyclically during a typical night's sleep in approximately **4–6 cycles**, each lasting **90–110 minutes**.

1.2.1 NREM Sleep

NREM sleep is divided into three stages based on EEG patterns:

- **Stage N1:** Transition phase from wakefulness to sleep. EEG shows theta wave activity (4–7 Hz). Muscle tone reduces, and slow rolling eye movements are observed. People awakened from N1 often claim they were not asleep.
- **Stage N2:** Occupies about 45–55% of total sleep in adults. EEG shows sleep spindles (short bursts of 12–14 Hz activity) and K-complexes (high voltage biphasic waves). This stage supports memory consolidation and sensory disconnection.
- **Stage N3 (Slow Wave Sleep):** Deepest sleep stage with dominant delta wave activity (<4 Hz). This stage is critical for physical restoration, immune system modulation, and secretion of growth hormone. Arousal from N3 is difficult and often leads to grogginess or sleep inertia.

1.2.2 REM Sleep

REM sleep accounts for **20–25%** of total sleep and is associated with:

- Rapid eye movements
- Dreaming activity
- Cortical activation
- Muscle atonia (paralysis of voluntary muscles)
- Irregular heart rate and respiration

REM sleep enhances emotional and procedural memory consolidation, creative thinking, and neuroplasticity.

1.3 Regulation of Sleep-Wake Cycles

The sleep-wake cycle is orchestrated by two major biological systems:

1.3.1 Circadian Rhythm

The circadian rhythm is an endogenous, entrainable rhythm synchronized by the **suprachiasmatic nucleus (SCN)** of the hypothalamus. Environmental light signals received by the retina modulate the SCN, which in turn regulates melatonin secretion from the **pineal gland**. Melatonin promotes sleep onset and stabilizes circadian timing.

Disruptions in circadian rhythm, such as **jet lag**, **shift work**, or **delayed sleep phase syndrome**, result in poor sleep quality, reduced cognitive performance, and metabolic imbalances.

1.3.2 Homeostatic Sleep Drive

The longer a person remains awake, the stronger the pressure to sleep. This process is regulated by adenosine accumulation in the brain. Sleep restores the balance by clearing adenosine and reducing homeostatic pressure.

1.4 Neurobiology of Sleep

Multiple neurotransmitters and brain structures are involved in sleep regulation:

- **Wake-promoting neurotransmitters:** Dopamine, norepinephrine, serotonin, histamine, acetylcholine, and orexin.
- **Sleep-promoting neurotransmitters:** GABA and galanin, primarily from the ventrolateral preoptic nucleus (VLPO).

The **flip-flop switch** model of sleep regulation proposes that sleep and wake states inhibit each other, ensuring stability and avoiding intermediate states.

1.5 Classification of Sleep Disorders

According to the **International Classification of Sleep Disorders (ICSD-3)**, sleep disorders are divided into six broad categories:

1.5.1 Insomnia Disorders

- Chronic insomnia
- Short-term insomnia
- Paradoxical insomnia

1.5.2 Sleep-Related Breathing Disorders

- **Obstructive Sleep Apnea (OSA)**
- Central sleep apnea
- Sleep-related hypoventilation syndromes

1.5.3 Central Disorders of Hypersomnolence

- Narcolepsy type 1 and 2

- Idiopathic hypersomnia

1.5.4 Circadian Rhythm Sleep-Wake Disorders

- Shift work disorder
- Jet lag disorder
- Irregular sleep-wake rhythm disorder

1.5.5 Parasomnias

- Sleepwalking (somnambulism)
- Night terrors
- REM sleep behavior disorder

1.5.6 Sleep-Related Movement Disorders

- Restless legs syndrome (RLS)
- Periodic limb movement disorder

Each category has distinct diagnostic criteria and management protocols.

1.6 Obstructive Sleep Apnea: A Clinical Overview

OSA is the most prevalent form of sleep-related breathing disorder, involving repeated upper airway collapses during sleep. These result in apneas (complete obstruction) and hypopneas (partial obstruction).

1.6.1 Clinical Consequences

- Intermittent hypoxia
- Fragmented sleep
- Sympathetic nervous system overactivation
- Daytime sleepiness, cognitive decline, mood changes

1.6.2 Apnea-Hypopnea Index (AHI) Classification

- Mild: 5–15 events/hour
- Moderate: 15–30 events/hour
- Severe: >30 events/hour

1.7 Diagnostic Tools in Sleep Medicine

1.7.1 Polysomnography (PSG)

The gold standard diagnostic test, PSG records:

- EEG (brain waves)
- EOG (eye movement)

- EMG (muscle activity)
- Airflow (nasal/oral)
- Respiratory effort
- Oxygen saturation
- ECG

1.7.2 Portable Sleep Monitors

Level III or IV devices used in home sleep studies offer practical alternatives for diagnosis in uncomplicated OSA cases.

1.7.3 Key Diagnostic Indices

- **Apnea-Hypopnea Index (AHI)**
- **Oxygen Desaturation Index (ODI)**
- **Arousal Index**

1.8 Sleep Hygiene and Behavioral Interventions

Sleep hygiene practices aim to enhance sleep efficiency. Key strategies include:

- Going to bed and waking up at the same time daily
- Avoiding screens and bright lights before bedtime
- Limiting caffeine, nicotine, and alcohol
- Creating a dark, cool, quiet bedroom environment
- Engaging in relaxing pre-bed routines (e.g., reading or meditation)

Cognitive Behavioral Therapy for Insomnia (CBT-I) remains the most effective treatment for chronic insomnia.

1.9 Prosthodontic Relevance in OSA Management

Dental professionals play a crucial role in sleep medicine, particularly in the diagnosis and management of OSA.

1.9.1 Orofacial Indicators of OSA

- Retrognathia
- Narrow palate
- Macroglossia
- High-arched palate
- Bruxism
- Enlarged tonsils

1.9.2 Mandibular Advancement Devices (MADs)

MADs are oral appliances designed to reposition the mandible and tongue anteriorly, maintaining airway patency during sleep. They are indicated for:

- Mild to moderate OSA
- Severe OSA intolerant to CPAP therapy

1.9.3 Clinical Considerations

- Custom fitting is essential
- Periodic titration and follow-up required
- Common side effects: TMJ discomfort, occlusal changes, dry mouth

1.9.4 Interdisciplinary Collaboration

Prosthodontists work in tandem with sleep physicians, ENT specialists, and pulmonologists to ensure comprehensive care. Referral pathways and co-management protocols are essential.

1.10 Emerging Trends and Research in Sleep Medicine

Recent advances in sleep medicine include:

- **AI-based PSG analysis** for automated scoring
- **Wearable devices** for long-term sleep monitoring
- **Genetic biomarkers** for sleep disorders
- **Neuromodulation therapies** (e.g., hypoglossal nerve stimulation)
- **Customized MADs** using 3D printing and digital impressions

Conclusion

Sleep is a complex, cyclical, and dynamic physiological process regulated by neurochemical, hormonal, and environmental influences. The foundational understanding of sleep architecture—including the progression through NREM and REM stages—is essential for recognizing disruptions in normal sleep patterns. These stages serve distinct but interdependent roles in physical restoration, memory consolidation, emotional regulation, and immunologic balance. The integrity of circadian rhythms, homeostatic mechanisms, and the orchestration of various neurotransmitter systems ensures optimal sleep quantity and quality. Disruptions in any of these systems contribute to the development of sleep disorders.

The classification of sleep disorders, as outlined in the ICSD-3, enables clinicians and researchers to systematically diagnose and manage a wide range of conditions, from insomnia to parasomnias and sleep-disordered breathing such as obstructive sleep apnea (OSA). Among these, OSA holds particular clinical significance, not only due to its high prevalence but also due to its extensive systemic effects—including cardiovascular, neurocognitive, and metabolic consequences. The measurement of apnea-hypopnea index (AHI), desaturation indices, and arousal frequencies offers objective criteria for both diagnosis and treatment planning.

From a prosthodontic standpoint, the relevance of sleep disorders—particularly OSA—cannot be overstated. The prosthodontist's role has evolved beyond the boundaries of conventional dental care to include contributions in sleep medicine through the fabrication and adjustment of mandibular advancement devices (MADs), recognition of orofacial anatomical risk factors, and interdisciplinary collaboration with physicians. With the advent of precision dentistry and digital technologies, prosthodontists are now uniquely positioned to offer patient-specific interventions that improve not only oral function but also sleep quality and systemic health.

Understanding sleep physiology is not merely academic—it serves as the clinical bedrock for evaluating patients with snoring, disrupted sleep, and related comorbidities. As dental professionals are often the first point of contact for patients presenting with signs of sleep-disordered breathing (e.g., bruxism, dry mouth, or retrognathia), early screening and referral protocols become essential. Incorporating sleep assessments into routine prosthodontic evaluations has the potential to greatly enhance patient outcomes.

Furthermore, with the increasing integration of dental sleep medicine into academic curricula, clinical guidelines, and regulatory frameworks, the knowledge of sleep science is becoming indispensable for every practicing dentist. This chapter lays the physiological and clinical groundwork upon which more advanced topics—such as the prosthodontic management of OSA, epidemiological patterns, and evidence-based treatment strategies—will be built in subsequent chapters.

In summary, mastering the physiology of sleep and the nuances of sleep disorders provides a necessary framework for understanding how these conditions intersect with prosthodontic practice. It enables clinicians to develop a holistic approach to patient care that not only addresses dental needs but also contributes to improved systemic and sleep health—ultimately aligning with the broader goals of multidisciplinary, patient-centered healthcare.

This section has provided an in-depth exploration of sleep physiology, classification of sleep disorders, OSA pathophysiology, diagnostic modalities, and the critical role of prosthodontists in sleep disorder management. A sound understanding of these principles is essential for dental professionals involved in treating sleep-disordered breathing.

In the next section, we will explore the epidemiology, risk factors, and pathogenesis of OSA in greater detail, focusing on prosthodontic perspectives and population-based research findings.