Review Article

University J Dent Scie 2026; Vol. 12, Issue 2

Sleep-Disordered Breathing Across the Lifespan: An Integrated Review of Definitions, Craniofacial and Airway Anatomy, OrofacialMyofunction, Pathophysiology, Diagnosis, and Management

Abstract:

Sleep-disordered breathing (SDB) comprises a spectrum of conditions characterized by abnormal respiration during sleep, ranging from primary snoring to obstructive and central sleep apnea. Affecting individuals across all age groups, SDB is associated with adverse cardiovascular, metabolic, neurocognitive, and craniofacial outcomes. Contemporary understanding recognizes SDB as a heterogeneous disorder arising from the interaction of upper airway anatomy, craniofacial morphology, neuromuscular control, and ventilatory regulation. Increasing evidence highlights the contributory role of orofacialmyofunctional patterns and altered craniofacial growth in both the development and persistence of disease. This review provides a comprehensive, age-independent synthesis of sleep-disordered breathing, integrating validated definitions and classifications from standard sleep medicine texts with contemporary endotype-based pathophysiological models. Upper airway anatomy, craniofacial growth, and orofacialmyofunction are discussed as interrelated determinants of airway stability. Diagnostic approaches, including clinical screening, dental evaluation, and polysomnography, are reviewed. Management strategies are presented in a stratified manner, emphasizing phenotype- and endotype-specific interventions across pediatric and adult populations. A multidisciplinary, airway-centered framework involving dentistry, sleep medicine, and allied specialties is essential for effective lifelong management of sleep-disordered breathing.

Key-words: Craniofacial Abnormalities, OrofacialMyofunctional Therapy, Polysomnography, Sleep Apnea Syndromes, Sleep-Disordered Breathing

Introduction:

Sleep-disordered breathing (SDB) represents one of the most prevalent chronic sleep-related disorders worldwide. Epidemiological data suggest that habitual snoring affects up to 40% of adults, while obstructive sleep apnea (OSA) occurs in approximately 9–38% of the adult population and 1–5% of children, depending on diagnostic criteria and age group.[1-3]Despite its high prevalence, SDB remains underdiagnosed, particularly in pediatric populations and in adults without classical symptoms.

Historically, SDB was conceptualized primarily as a disorder of upper airway obstruction managed by sleep physicians and otolaryngologists. However, advances in craniofacial biology, dental sleep medicine, and respiratory physiology have reshaped this perspective. The patency of the upper airway during sleep depends on a dynamic balance between anatomical constraints, neuromuscular tone, and ventilatory control. Craniofacial morphology, dental arch form, tongue posture, and orofacial muscle function significantly influence airway size and collapsibility.

Dentists and orthodontists routinely assess craniofacial structures and oral function across all age groups, placing them in a pivotal position for early identification of SDB risk.

Access this article online

Website:
www.ujds.in

DOI:
https://doi.org/10.21276/ujds.2026.12.2.31

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Received : 9 March, 2026, **Published :** 30 June, 2026

How to cite this article: Jain Choudhary, P., & Ganiga Channaiah Shivkumar. (2026). Sleep-Disordered Breathing Across the Lifespan: An Integrated Review of Definitions, Craniofacial and Airway Anatomy, Orofacial Myofunction, Pathophysiology, Diagnosis, and Management. UNIVERSITY JOURNAL OF DENTAL SCIENCES, 12(2).

Integrating dental and medical perspectives is therefore critical for comprehensive understanding and management of sleep-disordered breathing.

The **International Classification of Sleep Disorders, Third Edition (ICSD-3)** defines sleep-disordered breathing as a group of disorders characterized by abnormal respiratory patterns or insufficient ventilation during sleep.[4]

The SDB spectrum includes:

1.Primary Snoring

Primary snoring represents the mildest form of sleep-disordered breathing and is characterized by vibratory noise generated by turbulent airflow through a partially narrowed upper airway during sleep, without associated apnea, hypopnea, oxygen desaturation, or sleep fragmentation. [5]Snoring typically arises from vibration of the soft palate, uvula, tonsillar pillars, or pharyngeal walls and is influenced by airway compliance, neuromuscular tone, and craniofacial morphology.

Although traditionally considered benign, longitudinal studies suggest that habitual snoring may represent an early stage in the SDB continuum and may progress to obstructive sleep apnea in susceptible individuals.[6] In children, habitual snoring has been associated with neurobehavioral deficits, impaired attention, and altered craniofacial growth patterns. [7]

2. Upper Airway Resistance Syndrome (UARS):

Upper airway resistance syndrome is characterized by increased inspiratory effort due to partial upper airway narrowing, leading to repetitive respiratory effort–related arousals (RERAs) without meeting standard apnea–hypopnea criteria. [8] Patients typically present with excessive daytime sleepiness, fatigue, and non-restorative sleep despite a normal AHI.

UARS is frequently associated with subtle craniofacial abnormalities, including narrow maxillary arches, retrognathia, and high-arched palates. Unlike classic OSA, oxygen desaturation is minimal, but chronic sleep fragmentation may result in significant autonomic and neurocognitive consequences.[9]

3. Obstructive Sleep Apnea (OSA):

Obstructive sleep apnea is defined by recurrent episodes of partial or complete collapse of the upper airway during sleep, resulting in intermittent hypoxemia, hypercapnia, sleep fragmentation, and sympathetic activation.[4,5] OSA pathogenesis is multifactorial, involving anatomical vulnerability, neuromuscular dysfunction, and ventilatory instability.

Untreated OSA is associated with hypertension, coronary artery disease, stroke, insulin resistance, depression, and impaired quality of life.[10,11] In children, OSA is linked to impaired growth, learning difficulties, and behavioral disorders. [12]

4. Central Sleep Apnea (CSA):

Central sleep apnea is characterized by recurrent cessation of airflow due to absent or diminished central respiratory drive, resulting in apneas without respiratory effort.[13] CSA commonly occurs in association with heart failure, stroke, opioid use, and high-altitude exposure.

The underlying mechanism involves instability of the ventilatory control system, often described as elevated loop gain, leading to oscillations in ventilation.[14] Cheyne–Stokes respiration represents a classic CSA pattern seen in congestive heart failure.

5. Sleep-Related Hypoventilation Disorders:

Sleep-related hypoventilation is characterized by sustained elevation of arterial carbon dioxide levels during sleep. Etiologies include obesity hypoventilation syndrome, neuromuscular disease, chest wall disorders, and congenital central hypoventilation syndrome.

Chronic nocturnal hypoventilation leads to pulmonary hypertension, right heart failure, and reduced survival if untreated. [14]

Table 1. Classification of Sleep-Disordered Breathing

Disorder	Key Features	Gas Exchange Abnormality
Primary snoring	Airway vibration only	Absent
UARS	Increased resistance, arousals	Minimal
OSA	Apneas/hypopneas with effort	Present
CSA	Apneas without effort	Present
Hypoventilation	Sustained hypoventilation	Hypercapnia

Pathophysiology of Obstructive Sleep Apnea:

OSA is now understood as a **heterogeneous disorder** resulting from interaction between anatomical and non-anatomical traits.[15]

Anatomical Collapsibility:

Upper airway collapsibility is quantified by **critical closing pressure (Pcrit)**. A more positive Pcrit indicates a highly collapsible airway.

Upper Airway Muscle Responsiveness:

Reduced activation of pharyngeal dilator muscles, particularly the genioglossus, contributes to airway collapse during sleep.

Arousal Threshold:

A low arousal threshold leads to premature awakening before airway stabilization, while excessively high thresholds prolong hypoxemia.

Ventilatory Control Instability (Loop Gain):

High loop gain reflects an unstable ventilatory control system, promoting cyclical apnea–hyperpnea patterns.

Pathophysiology: Endotype-Based Model:

Modern models conceptualize OSA as the interaction of four key traits: Anatomical collapsibility (Pcrit); Upper airway muscle responsiveness; Arousal threshold; Ventilatory control stability (loop gain). This framework explains interindividual variability in disease severity and treatment response and forms the basis for personalized therapy.[16]

Craniofacial Growth and Airway Development

Craniofacial growth plays a critical role in determining airway size and shape, particularly during childhood and adolescence.[17]Maxillary transverse deficiency, mandibular retrognathia, and vertical growth patterns reduce the skeletal enclosure of the airway, increasing collapsibility.Functional factors such as mouth breathing, abnormal tongue posture, and altered swallowing patterns can redirect craniofacial growth through altered muscle forces acting on developing bone. [18,19]

Upper Airway Anatomy and Craniofacial Framework:

The upper airway extends from the nasal cavity to the larynx and comprises the nasopharynx, oropharynx, and hypopharynx. Unlike the lower airway, the pharynx lacks rigid structural support, rendering it vulnerable to collapse during sleep.[20]

Craniofacial Determinants of Airway Patency:

Craniofacial features associated with increased SDB risk include: Maxillary constriction and high-arched palate; Mandibular retrognathia; Increased lower anterior facial height; Steep mandibular plane angle; Inferiorly positioned hyoid bone; Reduced dental arch perimeter.[21-23]

Orofacial Myofunctional Factors:

Orofacialmyofunctional disorders involve dysfunction of the tongue, lips, cheeks, and jaw muscles at rest or during function.[24] Low tongue posture reduces palatal stimulation during growth, contributing to maxillary constriction and reduced nasal airway volume.

Common indicators include:Mouth breathing; Low or forward tongue posture; Lip incompetence; Mentalis muscle strain; Atypical swallowing patterns. In children, persistent OMDs may influence craniofacial growth and airway size. In adults, these functional abnormalities may perpetuate airway collapse and reduce treatment efficacy.

Diagnosis:

Overview of Diagnostic Principles:

The diagnosis of sleep-disordered breathing (SDB) is based on the integration of clinical symptoms, anatomical and functional risk factors, and objective sleep testing rather than reliance solely on apnea–hypopnea index (AHI) values. Contemporary understanding recognizes SDB as a heterogeneous disorder influenced by upper airway anatomy, craniofacial morphology, neuromuscular control, and ventilatory stability mechanisms. [25-27]

Oral physicians and dental specialists play an important role in early identification because several anatomical indicators of airway compromise are detectable during routine oral examination before patients seek sleep-related medical consultation.[28] Importantly, many of these craniofacial and functional characteristics overlap with risk factors implicated in temporomandibular disorders (TMD), suggesting shared pathophysiological pathways involving altered mandibular posture, muscle activity, and airway compensation.[29,30]

Clinical History and Symptom Assessment:

General Medical History:

A comprehensive history should evaluate classical nocturnal and daytime symptoms including snoring, witnessed apneas, gasping during sleep, excessive daytime sleepiness, morning headaches, nocturnal xerostomia, and impaired concentration.[31,32]

Patients presenting with chronic orofacial pain or TMD symptoms frequently report poor sleep quality and fragmented sleep, which may represent undiagnosed SDB rather than isolated pain-related insomnia.[30,33]Therefore, sleep screening is recommended in patients with persistent TMD complaints.In children, manifestations may include hyperactivity, behavioral disturbances, learning difficulties, enuresis, and failure to thrive.[34,35]

Oral Physician–Focused History:

Targeted questioning should include mouth breathing, recurrent tonsillitis, bruxism, temporomandibular discomfort, xerostomia, and prior orthodontic treatment.[36] Sleep bruxism, commonly observed in dental practice, has been associated with respiratory arousal responses during obstructive events and may represent a protective neuromuscular reaction aimed at reopening the airway rather than a purely parafunctional habit.[37] Recurrent muscle hyperactivity may contribute to development or exacerbation of TMD symptoms.

Validated Screening Questionnaires:

Screening tools such as STOP-BANG and ESS remain valuable for identifying adults at risk for OSA,[38,39] while the Pediatric Sleep Questionnaire aids pediatric risk assessment.[40] In patients presenting primarily with TMD or chronic facial pain, incorporation of sleep questionnaires improves detection of coexisting SDB, which may otherwise remain clinically unrecognized.

Clinical Examination:

General examination should include BMI, neck circumference, blood pressure, craniofacial profile, and nasal patency assessment.[26,41] Mandibular retrusion and vertical facial growth patterns are recognized risk factors for both airway collapse and temporomandibular joint biomechanical stress, reinforcing the need for simultaneous airway and TMJ evaluation during clinical examination.[42]

Oral Physician Perspective:

Extra-Oral Assessment

Findings such as mandibular retrusion, increased lower facial height, lip incompetence, mentalis strain, and forward head posture reflect compensatory adaptations to airway obstruction.[43] These postural adaptations may alter mandibular loading patterns and contribute to myofascial TMD symptoms over time.[44]

Intra-Oral Examination:

Tongue Assessment

Macroglossia, scalloping, and low tongue posture contribute to airway obstruction.[45] Increased tongue pressure and nocturnal repositioning efforts may also elevate masticatory muscle activity, linking airway instability with TMD-related muscle fatigue.

Palatal Morphology:

High-arched palates and reduced maxillary width are associated with decreased nasal airflow and altered occlusal relationships that may influence TMJ biomechanics.[42,46]

Tonsillar Evaluation and Mallampati Classification:

Higher Mallampati scores correlate with airway crowding and increased OSA risk,[47] conditions frequently accompanied by altered mandibular positioning during sleep.

Functional Assessment:

Chronic oral breathing and altered swallowing patterns influence craniofacial growth and airway stability.[48] Persistent orofacialmyofunctional dysfunction may simultaneously predispose individuals to airway collapse and muscular imbalance implicated in TMD pathogenesis.[49]

Imaging in Diagnosis:

CBCT and lateral cephalometry assist evaluation of airway dimensions and skeletal relationships.[48] Reduced posterior airway space and inferior hyoid positioning observed in OSA patients are also commonly reported in individuals with TMD, suggesting shared structural vulnerability.[49]

Polysomnography and Objective Sleep Testing:

Polysomnography remains the diagnostic gold standard.[25] Recognition of sleep fragmentation patterns is clinically relevant in TMD patients, as recurrent arousals and hypoxic stress may amplify pain perception through central sensitization mechanisms.[50]

Drug-Induced Sleep Endoscopy (DISE):

DISE enables identification of dynamic airway collapse patterns.[51] Understanding obstruction sites is particularly relevant when mandibular advancement therapy is considered, as mandibular repositioning influences both airway patency and temporomandibular joint loading dynamics.

Diagnostic Role of the Oral Physician:

Oral physicians function as gatekeepers by identifying anatomical risk indicators, initiating referrals, and monitoring treatment outcomes. Evaluation of TMJ status before initiating oral appliance therapy is essential, as pre-existing TMD may influence treatment tolerance and appliance selection.

Management : Pediatric SDB

Adenotonsillectomy, maxillary expansion, and myofunctional therapy remain primary treatments. [34,46,49] Early correction of airway dysfunction may also reduce development of maladaptive oral habits and functional patterns associated with future TMD risk.[52]

Adult OSA:

CPAP remains first-line therapy.[25] Oral appliance therapy advances the mandible to enlarge airway space;however, clinicians should monitor for transient TMJ discomfort or occlusal changes, particularly in patients with pre-existing TMD.[53]

Weight reduction, positional therapy, and surgery remain adjunctive options.

Endotype-Targeted Therapies:

Emerging therapies targeting loop gain, arousal threshold, and muscle responsiveness further support individualized treatment approaches,[53]which may indirectly improve pain modulation and sleep quality in patients with coexisting TMD.

Multidisciplinary Model of Care:

Optimal management requires collaboration among sleep physicians, dentists, orthodontists, otolaryngologists, and myofunctionaltherapists. Integration of airway and temporomandibular assessment allows comprehensive management of overlapping functional disorders affecting breathing, sleep quality, and orofacial health.[29,30]

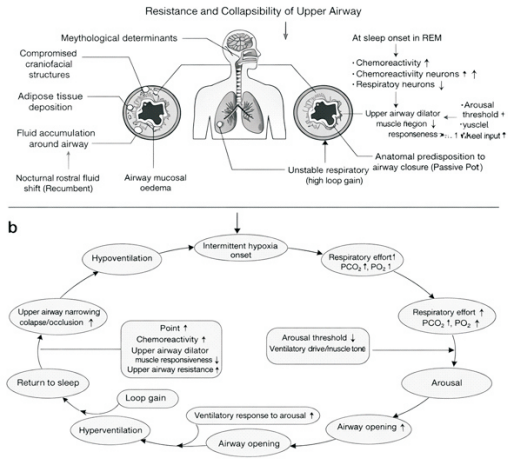

Figure 1- Conceptual diagram of sleep-disordered breathing showing **(a)** anatomical and neuromuscular factors contributing to upper airway collapse and **(b)** the ventilatory control cycle linking intermittent hypoxia, arousal, airway reopening, and recurrent respiratory events.

Conclusion:

Sleep-disordered breathing is a multifactorial condition affecting individuals across all age groups. Integration of airway anatomy, craniofacial growth, and orofacialmyofunction provides a comprehensive framework for diagnosis and individualized management. Recognition of the close interaction between SDB and temporomandibular disorders further emphasizes the importance of a multi disciplinary, phenotype-driven approach to improve detection, treatment outcomes, and long-term patient health.

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