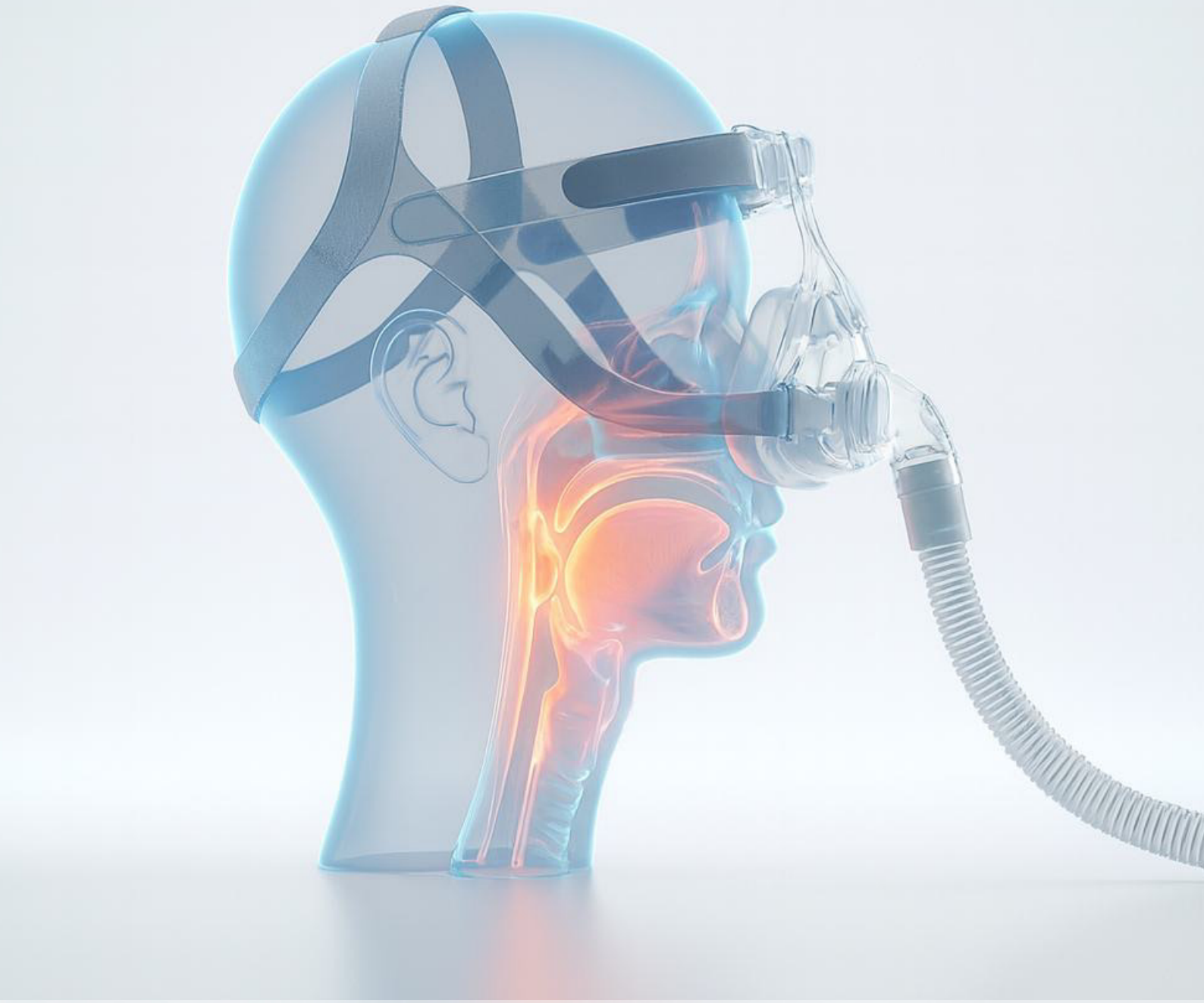
OBSTRUCTIVE SLEEP APNOEA

National Standard Treatment Guideline



Ministry of Health
Republic of Maldives

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Maldives

National Standard Treatment Guidelines

- Acid Peptic Disease
- Acute Anxiety
- Acute Pancreatitis
- Acute Psychosis
- Acute kidney Injury
- Arrhythmia
- Chronic Liver Disease
- Chronic Pancreatitis
- Chronic kidney disease
- Congenital Heart Diseases
- Dementia
- Depression
- Diabetes Mellitus Type 1
- Diabetes Mellitus Type 2
- Gestational Diabetes
- Epilepsy
- Heart Failure
- Hyponatremia
- Hypernatremia
- Hypokalemia
- Hyperkalemia
- Interstitial Lung Disease
- Liver Failure
- Obesity
- Obstructive Sleep Apnoea
- Osteoarthritis
- Ovarian Cancer
- Pneumonia
- Stroke
- Upper Gastrointestinal bleed
- Unstable Angina

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GUIDELINES DEVELOPMENT METHODOLOGY

The development of the Maldives Standard Treatment Guidelines (STGs) followed a structured, evidence-informed, and consensus-driven methodology adapted from internationally accepted guideline-development standards and the Delhi Society for Promotion of Rational Use of Drugs (DSPRUD) model. The process combined systematic evidence retrieval, critical appraisal, contextual adaptation, and multidisciplinary expert review to ensure feasibility, clinical relevance, and national ownership.

1. Determining Scope and Priority Conditions

Priority clinical conditions were identified through consultation with national programme managers, specialty clinicians, and health-system stakeholders. Selection criteria included: (i) major causes of morbidity and mortality, (ii) observed variation in clinical practice or prescribing patterns, (iii) potential to improve patient outcomes, and (iv) the feasibility of implementation across health-facility levels in Maldives. The final list of diseases reflected national epidemiology, service-delivery capacity, and essential-medicine availability.

2. Identification of Existing Evidence and Source Guidelines

A targeted search strategy was used to identify high-quality existing clinical guidelines. Searches were conducted across international guideline repositories (e.g., WHO, NICE, SIGN and other intergovernmental bodies, international and national guideline repositories, specialty societies and professional associations.

3. Quality Appraisal of Source Guidelines

Retrieved guidelines were screened for transparency of development, methodological rigour, clarity of recommendations, applicability to health-system reality, editorial independence. Guidelines were included if they met the Institute of Medicine (IOM) definition of a clinical guideline and addressed treatment or management of priority conditions. Guidelines that did not meet minimum quality standards, review articles, diagnostic criteria, or technical standards were excluded.

4. Adoption, Adaptation, and Contextualization

The guideline-development team employed an adopt–adapt–contextualize model:

- **Adoption**: High-quality recommendations that aligned with Maldivian health-system realities were retained without modification.
- **Adaptation**: Recommendations were modified when local considerations such as diagnostic capacity, medicine availability, workforce skills, referral pathways, or cost constraints affected feasibility.

- **Contextualization**: Where evidence was absent or inconclusive, conditional recommendations were formulated based on expert consensus, with explicit consideration of pragmatism, safety, and local workflows.
  Medicines were selected in alignment with the Maldives National Essential Medicines List (NEML), based on suitability, efficacy, safety, and availability.

5. Expert Consensus and Multidisciplinary Input

Draft recommendations were initially prepared by experts from the DSPRUD, India, providing a strong methodological foundation for the process. Building on this, a collaborative and participatory process brought together clinicians from internal medicine, paediatrics, obstetrics-gynaecology, surgery, emergency medicine, endocrinology, cardiology, general practitioners, and public health representing different levels of healthcare. Consensus was achieved through moderated discussions, iterative revisions, and resolution of divergent views. For topics lacking strong evidence, recommendations were derived from expert clinical judgment grounded in extensive practice experience.

6. Drafting, Peer Review, and Validation

Each guideline section was organized in a standard format including key clinical features, essential investigations, non-pharmacological management, pharmacological therapy (with step-up/step-down options where relevant), referral criteria, paediatric considerations, and follow-up requirements. Drafts were peer-reviewed by senior clinicians and national experts. Reviewer comments were systematically integrated to strengthen clarity, accuracy, and applicability.

7. Addressing Conflicts of Interest

All contributors declared the absence of conflicts of interest. Individuals with potential or perceived conflicts were excluded from authorship or decision-making roles.

8. Updating and Future Revisions

The STGs were conceptualized as a living document. Future updates will incorporate new scientific evidence, changes in essential-medicine availability, national programme priorities, and user feedback from clinicians. Periodic review cycles will ensure the continued relevance and reliability of recommendations.

9. Distinctive Features of the Guidelines

Developed through a collaborative process involving a large group of multidisciplinary experts from different levels of healthcare, the guidelines incorporate the following distinctive features:

- **Diagnostic Assumption and Confirmation**: While assuming that an initial diagnosis has been established by the healthcare provider, the guidelines provide essential information for confirming diagnoses. This includes a comprehensive overview of major signs and symptoms, descriptions of confirmatory tests, and clear guidance on practices that are prohibited, discouraged, or unreliable—promoting evidence-based medicine supported by relevant references.
- **Comprehensive Treatment Approach**: The guidelines offer a systematic, up-to-date framework for managing medical conditions across the continuum of care. They begin at the primary care level and extend to secondary and tertiary care, incorporating protocols for treatment response assessment and referral criteria as integral components.
- **Diverse Treatment Modalities**: Recommendations encompass both non-pharmacological and pharmacological interventions and surgical intervention where applicable, providing flexibility for individualized treatment plans. Cautionary notes are included where necessary to ensure safe and effective use of therapies.
- **Assessment and Referral Criteria**: Clear criteria and goals for evaluating patient response to treatment are provided, along with guidance on when referral to higher levels of care is warranted ensuring continuity and comprehensiveness in patient management.

ACKNOWLEDGEMENTS

The Government of the Republic of Maldives is committed to ensuring universal access to quality health services for all citizens. The Constitution of Maldives mandates the progressive realization of rights, including the right to good standards of health care for the population. In line with this national commitment, standardized quality health services are regarded as the foundation of a strong and equitable healthcare system.

This important work would not have been possible without the cooperation and support of many individuals and institutions. We express our sincere appreciation to the Honourable Minister of Health, Abdullah Nazim Ibrahim, for his leadership, commitment, and continuous guidance throughout the development process. We are grateful to WHO and ADB for their significant contribution, support, and technical assistance.

Our heartfelt gratitude is extended to the technical lead and editor, Dr. Sangeeta Sharma, Professor, Neuropsychopharmacology, IHBAS and President, Delhi Society for Promotion of Rational Use of Drugs (DSPRUD), and her team. We express our deepest appreciation to the Maldivian and DSPRUD experts and contributors who played a pivotal role in this process. Their technical expertise and dedication to adapt the standards to the Maldivian context have been instrumental in the development and finalization of these guidelines. The time, experience, generous sharing of knowledge and insights contributed by all parties have not only enriched the work but also have been invaluable in making these standards practical, locally acceptable, and aligned with the needs of the resident population.

It is important to acknowledge the immense efforts, involvement, timely coordination, collaboration, and dedication of the Quality Assurance and Regulation Division team who made it possible for these Clinical Treatment Guidelines to come into existence.

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OBSTRUCTIVE SLEEP APNOEA

QUICK REFERENCE GUIDE

Obstructive sleep apnoea (OSA) affects about one billion adults globally, with moderate to severe disease most common in middle-aged individuals and those with obesity. It is strongly associated with cardiovascular, metabolic, and neurocognitive morbidity, increasing risks of hypertension, diabetes, heart disease, stroke, and accidents. With timely diagnosis and consistent therapy such as CPAP, outcomes improve significantly, reducing mortality and long-term complications.

Definition

Obstructive sleep apnoea (OSA) is defined as a recurrent upper-airway obstruction during sleep causing apnoeas/hypopnoeas, causing oxygen desaturation, and arousals.
Obstructive sleep apnoea is diagnosed when a polysomnography (PSG)-determined obstructive respiratory disturbance index (RDI) is ≥5 events/hour with symptoms (e.g., unrefreshing sleep, daytime sleepiness, fatigue/insomnia, gasping/choking, loud snoring, or witnessed apnoeas) or ≥15 events/hour regardless of symptoms. The RDI includes apnoeas, hypopnoeas, and respiratory-effort-related arousals (RERAs).
Various phenotypes are positional OSA (supine-predominant), rapid eye movement (REM)-related OSA, craniofacial/anatomical OSA, obesity-related OSA, overlap with chronic obstructive pulmonary disease (COPD), OSA with hypoventilation (e.g., obesity hypoventilation syndrome [OHS]).

Causes, risk factors & triggers

- Anatomy: large tonsils, macroglossia, elongated soft palate, low hyoid, retro/micrognathia, large neck, peripharyngeal fat.
- Non-anatomical: reduced pharyngeal dilator tone in sleep, high arousal threshold, ventilatory control instability (high loop gain).
- Risk factors: obesity (central > general), male sex, post-menopause, ageing, family history.
- Aggravators: supine sleep, evening alcohol/sedatives/opioids, nasal obstruction, smoking, rostral fluid shift (heart failure), uncontrolled hypothyroidism/acromegaly.

Evaluation for diagnosis

- Excessive daytime sleepiness, loud snoring, witnessed apnoeas/gasping, unrefreshing sleep, morning headaches; insomnia (often in women); nocturia; cognitive/mood change; near-misses while driving.
- Body mass index (BMI)/waist; neck circumference (>40 cm men, >37 cm women); Mallampati class 3-4; tonsillar hypertrophy; craniofacial crowding.

Screening tools (risk stratification; not diagnostic)

- **STOP-BANG** (Snoring, Tiredness, Observed apnoeas, high Blood pressure, BMI ≥35 kg/m², Age ≥50 years, Neck >40 cm, male Gender); score ≥5 = high risk.
- **Berlin Questionnaire; NoSAS** (Neck circumference, Obesity, Snoring, Age, Sex; score ≥8 = high risk).
- **Epworth Sleepiness Scale (ESS)**: >10 suggests excessive daytime sleepiness.

Confirmation of diagnosis

- Polysomnography (PSG), Level 1, attended: gold standard.
- Home sleep apnoea testing (HSAT), Level 3: for uncomplicated adults with high pretest probability; negative/ technically inadequate HSAT with persistent suspicion → PSG.
- Score events per American Academy of Sleep Medicine (AASM) rules. Use apnoea-hypopnoea index (AHI) for PSG and respiratory event index (REI) for HSAT.

Confirmation of diagnosis

- **Normal:** apnea-hypopnea index (AHI)/ RDI/REI < 5 events/hour
- **Mild OSA:** 5-14.9 events/hour
- **Moderate OSA:** 15-29.9 events/hour
- **Severe OSA:** ≥30 events/hour

Use AHI (polysomnography), RDI (includes RERAs), or REI (home test) with the same cut-offs. Interpret with symptoms and risk. Adjunct metrics such as hypoxic burden, total sleep time with peripheral oxygen saturation <90% (TST90), and arousal burden refine prognosis.

Differential diagnosis

- Central sleep apnoea; upper airway resistance syndrome (UARS); chronic insomnia; restless legs syndrome and periodic limb movements (PLMs); nocturnal seizures; hypoventilation syndromes (e.g., OHS); narcolepsy; circadian/behavioural sleep disorders.

Management goals & principles

- **Goals:** improve sleepiness and daytime function; reduce AHI/REI and nocturnal hypoxaemia; lower cardiovascular/ metabolic risk; reduce crash risk; optimise comorbidities.
- **Principles:** confirm objectively; address modifiable factors; use continuous positive airway pressure (CPAP) as default for moderate-severe or symptomatic disease; monitor adherence/effectiveness; escalate or de-escalate stepwise.

Approach to management (stepwise)

1. **Lifestyle & risk control for all:** weight loss (target ≥5-10%), side-sleeping if positional, treat nasal disease, avoid evening alcohol/sedatives, smoking cessation, optimise hypothyroidism/ heart failure/COPD.

2. **First-line therapy:**
    - **CPAP** (fixed; or auto-titrating CPAP if no lab titration available) for moderate-severe OSA or symptomatic mild OSA. Early follow-up at 1-3 weeks to check adherence, mask leaks, residual events.
3. **If CPAP intolerance/high pressures/ hypoventilation:**
    - **Bilevel positive airway pressure (BiPAP/BPAP)-** lower expiratory positive airway pressure (EPAP) for comfort; adequate inspiratory positive airway pressure (IPAP) for ventilation; add backup rate (spontaneous/timed [S/T] mode) if hypoventilation.
4. **Oral appliance therapy (OAT):** customized, titratable mandibular advancement device for mild-moderate OSA or CPAP-intolerant patients; fitted by a qualified dentist with sleep-physician oversight; verify with follow-up sleep testing.
5. **Surgery (selected cases):** after drug-induced sleep endoscopy (DISE) mapping- nasal surgery for obstruction/PAP tolerance; uvulopalatopharyngoplasty (UPPP) for palatal collapse; maxillomandibular advancement (MMA) for retrognathia/ multilevel collapse; hypoglossal nerve stimulation (HNS) for eligible CPAP-intolerant moderate-severe OSA; tracheostomy for refractory, life-threatening OSA.
6. **Special situations:**
    - **Overlap (OSA + COPD):** CPAP first; switch to BiPAP if chronic hypercapnia or ventilatory failure.
    - **OHS:** CPAP if hypoventilation is mild; BiPAP if daytime carbon dioxide (CO_2) remains elevated or CPAP fails.
    - **Perioperative/pregnancy:** screen high-risk; prioritize PAP, positional therapy; avoid sedatives.

Non-pharmacological interventions

- Prioritise STOP-BANG in non-communicable disease (NCD) and cardiology clinics and pre-operative areas.
- Use HSAT pathways for uncomplicated high-probability adults; reserve PSG for complex/discordant cases.
- Provide positional therapy devices or low-cost side-sleep training.
- Group education on mask fit, cleaning, humidification, and adherence coaching.
- Tele/phone follow-up for early troubleshooting.

Pharmacological therapy (adjuncts; OSA itself is treated with PAP/OAT/surgery)

1. Nasal obstruction impairing PAP: Fluticasone propionate 1-2 sprays/ nostril daily; saline rinses.
2. Anti-obesity pharmacotherapy (with lifestyle; per local criteria): Semaglutide 2.4 mg subcutaneous [SC] weekly or liraglutide 3 mg SC daily.

2. Residual sleepiness despite effective PAP (verify adherence and low residual AHI/REI; exclude other causes):
    - Modafinil 100-200 mg orally (PO) morning; may add 100 mg midday. Cautions: hypertension, anxiety, drug interactions.

Note: Avoid routine hypnotics/ benzodiazepines/opioids at bedtime unless essential and supervised.

Assessment of response; follow-up & adjustment

- Symptoms: ESS trend; fatigue/ cognition; driving risk.
- Device data: adherence ≥4 h/night on ≥70% nights (prefer all-night use); leaks within target; residual AHI <5/h (or ≥50% reduction from baseline if very severe).
- Comorbidities: blood pressure (BP), weight/waist, glycated haemoglobin (HbA1c)/lipids, atrial fibrillation (AF) burden.
- Adverse effects: dryness, leaks, skin injury, aerophagia - address with humidification, mask refit/change, pressure tweaks.
- Timelines: first review 1-3 months (earlier if issues), then 6-12 monthly if stable.
- When to repeat testing: persistent symptoms despite “adequate” use, discordant device data, ≥10% weight change, new cardiopulmonary/ neurologic issues → HSAT/PSG ± titration.
- Step-up triggers: intolerance or inadequate control on CPAP, persistent hypoventilation, high residual AHI, marked sleepiness → BiPAP, OAT, surgery/HNS evaluation.
- Step-down: only after sustained control, with close monitoring.

Referral (tiered)

- **Primary to Secondary:** uncertain/ complicated diagnosis, comorbid complexity, CPAP intolerance despite coaching, suspected central apnoea/ hypoventilation.
- **Secondary to Tertiary:** refractory OSA, need for advanced titration/ventilation (BiPAP S/T), overlap/OHS with hypercapnia, surgical or HNS evaluation, severe cardiopulmonary comorbidity, pregnancy with moderate-severe OSA.

Complications (unrecognized/ untreated)

- Hypertension; coronary artery disease (CAD)/myocardial infarction (MI); AF/ other arrhythmias; heart failure; stroke; pulmonary hypertension; metabolic syndrome/type 2 diabetes; depression/ cognitive impairment; motor-vehicle crashes/work errors; chronic hypoxaemia/hypercapnia.

Objectives of patient education & instructions to patient/ caregiver

- Explain OSA: repeated airway collapse leads to sleepiness, cardiovascular risk, and cognitive effects; treatment lowers these risks.
- Safety: never drive when drowsy; pull over if sleepy.
- Lifestyle: structured weight-loss plan; side-sleep if positional; avoid evening alcohol/sedatives; treat nasal symptoms; stop smoking.
- Positive airway pressure (PAP) use: wear every night; target ≥4 h/night (ideally all night); use ramp/exhalation relief; humidifier for dryness; do not self-change pressures.
- Troubleshooting: report leaks, discomfort, dryness, aerophagia early; try alternative mask types/sizes.
- Cleaning/maintenance: daily wipe of mask; weekly soap-and-water clean of mask/tube/humidifier tank; replace filters per schedule.
- Follow-up: bring device/app data; first review 1-3 months, then 6-12 monthly; earlier if symptoms recur or weight changes ≥10%.
- Red flags: In case of persistent excessive sleepiness despite use; new/ worsening chest pain, palpitations, severe morning headaches, marked breathlessness seek urgent review.

INTRODUCTION

Obstructive sleep apnea (OSA) is repeated upper-airway collapse during sleep that causes intermittent hypoxia, sleep fragmentation, and sympathetic surges, leading to multisystem harm. It's strongly linked to cardiovascular disease, metabolic dysfunction, neurocognitive deficits, traffic accidents, and higher mortality when untreated. An estimated billion adults have some degree of OSA worldwide, with moderate to severe disease common in middle-aged and people with obesity and rising over the last two decades. South Asians are at higher risk because of central obesity and craniofacial factors; Indian studies show a large undiagnosed burden, and similar cardiometabolic profiles suggest substantial hidden OSA in the Maldives. Risk increases with age; men, post-menopausal women, and those with obesity are most affected, and older adults may present atypically. Most patients can achieve better oxygenation, sleep quality, and lower downstream risk with continuous positive airway pressure (CPAP) or other tailored therapies. Standardized identification and management across levels of care reduce variability, prevent under- or overtreatment, support timely referral, and embed risk-factor modification.

SCOPE OF THE GUIDELINES

These guidelines cover adults with suspected or confirmed obstructive sleep apnea and are designed for use across primary, secondary, and tertiary care, with recommendations scaled to the diagnostic and treatment capacity at each level.

Intended users: primary-care physicians/GPs, internists, pulmonologists, ENT/sleep surgeons, cardiologists, endocrinologists, anesthesiologists (perioperative screening), dentists practicing dental sleep medicine, sleep physicians, nurses, community health workers, respiratory therapists, and sleep technologists; administrators may use them for pathway design.

- Primary care focuses on case finding, risk stratification with validated tools, basic lifestyle and positional advice, and identifying who needs further evaluation
- Secondary care coordinates home sleep apnea testing for uncomplicated high-probability cases, starts CPAP with basic support, manages common comorbidities, and escalates when needed.
- Tertiary centers provide definitive diagnosis with in-lab polysomnography, CPAP titration, specialist evaluation including surgical options, and management of refractory or complex cases.

The pathway assumes polysomnography at higher tiers but prioritizes screening, selective home testing, and structured referral where full studies are not available.

DEFINITION

AASM/ICSD-3 adult definition of OSA: Obstructive sleep apnoea is diagnosed when a polysomnography (PSG) - determined obstructive respiratory disturbance index (RDI) is ≥5 events/hour with symptoms (e.g., unrefreshing sleep,daytime sleepiness, fatigue/ insomnia, gasping/choking, loud snoring, or witnessed apnoeas) or ≥15 events/hour regardless of symptoms. The RDI includes apnoeas, hypopnoeas, and respiratory-effort-related arousals (RERAs).

Event scoring follows the AASM manual: apnoea = ≥90% airflow drop for ≥10 s; hypopnoea (recommended rule) = ≥30% flow reduction for ≥10 s with ≥3% desaturation or an arousal (an alternative rule uses ≥4% desaturation without arousal).

Phenotypes: positional OSA (supine-predominant), rapid eye movement (REM)-related OSA, Stage-independent (non-stage-specific), craniofacial/anatomical OSA, obesity-related OSA, overlap with chronic obstructive pulmonary disease (COPD), OSA with hypoventilation (e.g., obesity hypoventilation syndrome [OHS]).

CAUSES, RISK FACTORS & TRIGGERS

Pharyngeal narrowing and collapse during sleep is a multifactorial process. Sleep-related reductions in ventilatory drive, decreases in upper airway neuromuscular tone, and predisposing anatomical features combine to make the airway more collapsible, leading to intermittent obstruction.

Domain	Key factors / examples	Mechanism (why airway collapses)	Clues / notes
Sleep-related physiology	Decreased Ventilatory drive, decreased upper-airway neuromuscular tone during sleep	Less dilator-muscle activation → pharyngeal lumen narrows → intermittent obstruction	Universal in sleep; severity depends on anatomy/risk factors
Anatomical contributors	Micro/retrognathia, mandibular hypoplasia, facial elongation, large neck circumference, excess peripharyngeal soft tissue, tonsillar/adenoidal hypertrophy, low (inferior) hyoid	Reduced luminal space or increased surrounding tissue pressure → higher collapsibility	Craniofacial features, obesity, enlarged tonsils; visible on exam or imaging
Nonanatomical risk factors	Obesity (central fat), male sex, advancing age; alcohol, sedatives, smoking	Fat around airway raises closing pressure; age/sex affect muscle tone and fat patterning; substances depress tone/ reflexes	High BMI/waist; evening alcohol/sedative use; smoking history
Co-morbid contributors	Endocrine: diabetes, metabolic syndrome, acromegaly, hypothyroidism. Neurologic: stroke, SCI, myasthenia. Genetic: Down, Prader-Willi. Cardiac: CHF, atrial fibrillation. Overlap: obesity hypoventilation	Alter airway anatomy (e.g., acromegaly), reduce neuromuscular control, promote fluid shifts, or blunt arousal responses	Screen broadly; treat comorbidities to reduce OSA severity

Aggravating factors	Supine sleep; alcohol/sedatives; nasal congestion; fluid retention/ rostral fluid shift (e.g., CHF)	Supine: tongue/soft tissue fall back. Alcohol/ sedatives: tone and arousal declines leading to longer apneas. Nasal block: increases resistance & mouth breathing and collapse. Fluid shift: peripharyngeal edema narrows lumen	Advise side-sleeping/positional therapy; limit evening alcohol/ sedatives; treat rhinitis; optimize diuresis/CHF management

DIAGNOSIS

Domain	Key items/examples	Objective tools & thresholds	Notes
Core symptoms	Excessive daytime sleepiness (EDS), loud snoring, gasping/choking, witnessed apneas; some present with fatigue only	ESS >10 - EDS; FSS mean 4.0 - clinically significant fatigue	Differentiate sleepiness vs fatigue; track scores over time
Other complaints	Morning headache, nocturnal reflux, insomnia (onset/maintenance; often in women), nocturia, cognitive slowing, poor concentration, irritability, mood change, motor-vehicle accidents/near-misses	-	Reflect systemic impact of fragmented sleep and intermittent hypoxia
Examination clues	High BMI; neck circumference >40 cm (men) or >37 cm (women); Mallampati 3-4, retro/micrognathia, tonsillar hypertrophy, low palate, overjet, macroglossia	-	Lateral pharyngeal narrowing is an independent predictor after adjusting for weight and neck size
Associated conditions	Hypertension; features of hypothyroidism or acromegaly	Targeted labs/imaging as indicated	Evaluate comorbid endocrine disease if suspected

Screening tools

Validated screening tools can be used to identify patients at high risk of obstructive sleep apnea early, prioritize diagnostic testing, and guide pretest probability in settings with limited resources (at primary and secondary care).

Tool	Items assessed	Scoring & cut-offs	Use/settings	Strengths	Caveats / next step
STOP-BANG	Snoring, Tiredness, Observed apneas, high blood Pressure, BMI ≥35 kg/m², Age ≥50 y, Neck >40 cm, Gender (male)	0-8 points. ≥5 = high risk; 3-4 = intermediate; 0-2 = low.	Primary care, pre-op clinics, driver fitness, NCD/ cardiac clinics.	Very simple; high sensitivity for moderate-severe OSA.	Not diagnostic; many false positives. Positive → HSAT or PSG.

Berlin Questionnaire	Category 1: snoring; Category 2: daytime sleepiness; Category 3: hypertension/ obesity	High risk if ≥2 categories positive.	Community/ outpatient screening.	Stratifies by symptom clusters; easy to administer.	Variable specificity; confirm with objective testing.
NoSAS	Neck (≥40 cm), Obesity (BMI), Snoring, Age, Sex (male)	0-17 points; ≥8 = high risk.	Primary/ secondary care where brevity is key.	Very brief; reasonable performance in some cohorts.	May miss non-obese phenotypes; confirm with HSAT/PSG.
Epworth Sleepiness Scale (ESS)	Subjective daytime sleepiness in 8 situations	>10 = excessive daytime sleepiness. Track over time.	Any setting; baseline and follow-up symptom tracking.	Quantifies sleepiness burden and response to therapy.	Not a screen for OSA by itself; interpret with a risk tool.

Notes: Use these tools to prioritize testing and referrals in resource-limited primary/ secondary care. They do not diagnose OSA; positive or intermediate-risk screens should proceed to home sleep apnea testing (HSAT) for uncomplicated high-probability cases or polysomnography (PSG) when complexity or comorbidities are present.

CONFIRMATION OF DIAGNOSIS

Adult patients with unexplained daytime or sleep-related complaints such as excessive sleepiness, persistent fatigue, unrefreshing sleep, or witnessed apneas should be evaluated for sleep apnea.

Universal screening of asymptomatic individuals is not recommended except in high-risk occupations (drivers, divers, pilots) or in patients with key comorbidities such as refractory atrial fibrillation, resistant hypertension, or prior stroke, where OSA contributes substantially to disease burden and may be occult.

Domain	When/Who	Test & Key Criteria	Notes / Next steps
Gold standard	Any patient needing definitive diagnosis or with complex comorbidity	Attended in-lab Level 1 PSG: EEG, airflow (oronasal thermal + nasal pressure), respiratory effort, SpO_2, ECG, EMG; snoring recorded (not scored)	Performed in accredited lab with scoring by qualified staff
Event scoring (AASM)	All PSG interpretations	Apnea: ≥90% airflow drop ≥10 s. Hypopnea (recommended): ≥30% flow reduction ≥10 s with ≥3% desaturation or arousal. Acceptable alt.: ≥4% desaturation. Classify apneas as obstructive, central, mixed (by effort).	Use AHI (events/hour of sleep) from PSG

Home Sleep Apnea Testing (HSAT, Level 3)	Adults with high pretest probability of moderate-severe OSA and no major cardiopulmonary/ neurologic disease	Minimum channels: airflow, respiratory effort, SpO_2; structured oversight; data reviewed by qualified specialists per AASM	If negative/technically inadequate but suspicion remains → in-lab PSG. HSAT uses recording time, so index is REI (tends to underestimate AHI ~20%)
Alternative tech	When validated device available; adjunct to pathways	Peripheral Arterial Tonometry (PAT) derived apnea-hypopnea index (also called PAT-derived AHI; pAHI)	Correlates with PSG AHI but device bias exists; interpret in context
Follow-up after any portable test	All HSAT/PAT users	Mandatory clinical review of results and symptoms	If moderate-severe OSA still suspected after negative/ borderline study → confirm with PSG
Split-night study	Appropriate candidates per guideline	Single-night diagnosis + CPAP titration	Use when criteria met (sufficient early events); otherwise plan separate titration
Ancillary evaluation	To assess contributors/ comorbidity	Overnight oximetry (screen for nocturnal desaturation), TSH if hypothyroid features, HbA1c & lipids for metabolic risk, ECG/ echo if cardiac symptoms/ disease	Ancillary tests do not diagnose OSA; they guide risk assessment and comorbidity management

SEVERITY CLASSIFICATION AND EMERGING DIAGNOSTIC METRICS

AASM severity classification (adults, ≥18 y)

- **Normal:** apnea-hypopnea index (AHI)/RDI/REI < 5 events/hour
- **Mild OSA:** 5-14.9 events/hour
- **Moderate OSA:** 15-29.9 events/hour
- **Severe OSA:** ≥30 events/hour

Use AHI (polysomnography), RDI (includes RERAs), or REI (home test) with the same cut-offs.
Pediatric cut-offs differ (commonly: mild 1-4.9, moderate 5-9.9, severe ≥10 events/ hour).

Note: Do not rely solely on event frequency to determine clinical burden. Mild OSA should be evaluated in the context of symptoms (excessive daytime sleepiness, sleep maintenance insomnia, cognitive dysfunction) and comorbid risks.

Emerging and Complementary Metrics (for refined risk stratification) Consider adjunctive physiologic indices when available to better capture individual pathophysiology and prognostic risk:

i. Hypoxic burden (integrates depth, duration, and frequency of desaturations)

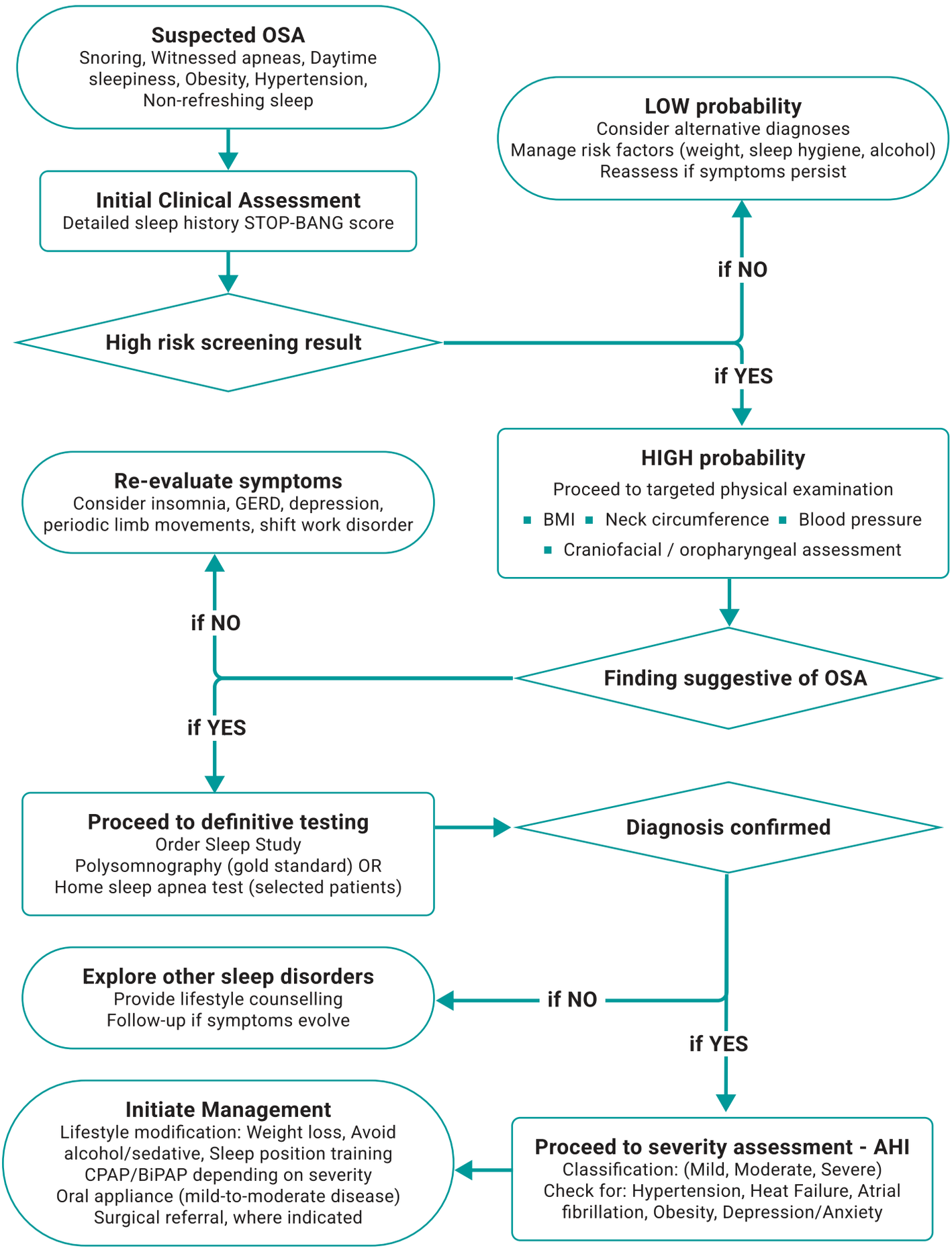
ii. Nocturnal heart rate variability/changes

iii. Total sleep time with SpO2 <90% (TST90)

v. Duration and clustering of obstructive events: Recognize that patients with similar AHI/REI/pAHI may have divergent cardiovascular or neurocognitive outcomes; use emerging metrics to inform personalized thresholds for escalation, follow-up intensity, or adjunctive therapy.

Use AHI/REI/pAHI to classify severity. Always correlate severity with symptoms and comorbidities before making treatment decisions, especially in mild OSA. If available, incorporate hypoxic burden, TST90, arousal burden, and related metrics to refine risk assessment in borderline or discordant cases. Reassess severity and risk periodically, particularly if symptoms change, weight fluctuates, or new comorbidities develop.

DIAGNOSTIC FLOWCHART



DIFFERENTIAL DIAGNOSIS

The differential diagnosis of suspected OSA includes several disorders that can mimic or coexist with it.

Condition	Key clinical clues	How to differentiate / tests	Can it coexist with OSA? / Notes
Central sleep apnea (CSA)	Apneas without effort; Cheyne-Stokes in heart failure/opioid use	PSG: absent thoracoabdominal effort during events	Yes; treat drivers (HF, opioids), consider ASV/CPAP per cause
Upper Airway Resistance Syndrome (UARS)	EDS, fragmented sleep; loud snoring; minimal desaturation; AHI often <5	PSG with elevated RERAs/ flow limitation; high arousal index	Often; CPAP/oral appliance may help even with low AHI
Chronic insomnia	Difficulty initiating/maintaining sleep; daytime fatigue > sleepiness	Sleep diary/actigraphy; normal PSG for breathing	Very common; manage insomnia (CBT-I) alongside OSA
Restless legs syndrome (RLS)	Urge to move legs at rest, worse in evening; relief with movement	History; ferritin; PSG may show periodic limb movements	Yes; treat RLS (iron, dopamine agents) to improve sleep quality
Nocturnal seizures	Stereotyped motor events, tongue bite, postictal confusion	Video-EEG/PSG; neurology eval	Possible; distinguish from apnea-related arousals
Hypoventilation syndromes (e.g., obesity hypoventilation)	Daytime hypercapnia, morning headaches, right-heart strain	ABG ($PaCO_2$ ≥45 mmHg), transcutaneous CO_2; PSG with hypoventilation	Frequently; requires PAP with backup rate and weight management
Narcolepsy	Sleep attacks, cataplexy, hypnagogic hallucinations, sleep paralysis	MSLT after adequate sleep; low CSF hypocretin in type 1	Yes; treat both -PAP for OSA, wake-promoting meds for narcolepsy
Behavioral/ circadian disorders (e.g., delayed sleep phase, poor sleep hygiene)	Late sleep schedule, variability, nonrestorative sleep without snoring/apneas	Sleep logs, actigraphy; response to circadian/ sleep-hygiene	Possible; distinguish from apnea-related arousals

Management Goals

- Improve sleep quality and daytime function; relieve sleepiness and cognitive deficits.
- Reduce respiratory events (lower AHI/REI) and accident risk from impaired alertness.
- Lower cardiovascular and metabolic risk; optimize control of hypertension, diabetes, and heart disease.Lower cardiovascular and metabolic risk; optimize control of hypertension, diabetes, and heart disease.

- **Treatment impact:** Clear benefits in moderate-severe OSA. In mild OSA, weigh symptoms and comorbid risk rather than event counts alone.

Management principles

- Tackle modifiable factors: weight loss, positional therapy (avoid supine), avoid evening alcohol/sedatives.
- Base decisions on objective testing (PSG or appropriate HSAT) and severity.
- Start first-line therapy by severity/symptoms - CPAP is default for moderate-severe or symptomatic disease.
- Monitor adherence and effectiveness with symptom scales and device data; adjust early.
- Use a stepwise plan: optimize current therapy → add/shift to oral appliance or advanced PAP/titration → refer to specialist/higher level if intolerance, inadequate response, or complexity.

NON-PHARMACOLOGICAL INTERVENTIONS

Management of OSA combines lifestyle modification, risk factor optimization, and targeted therapies based on severity and phenotype.

Intervention	Who/When	What to do	Follow-up / Notes
Weight reduction & lifestyle	All with overweight/obesity; adjunct in any severity	Structured diet + physical activity; aim 5-10% weight loss. Avoid alcohol/ sedatives before sleep.	Even modest loss improves AHI. Reinforce long-term habits.
Treat contributing conditions	Anyone with suggestive features	Manage nasal obstruction (e.g., rhinitis), hypothyroidism, acromegaly.	Treating drivers can ease PAP use and reduce events.
Positional therapy	Supine-predominant OSA	Side-sleeping via behavioral cues or devices.	Recheck response (symptoms/ HSAT). Often adjunct to PAP.
Alcohol/sedative avoidance	All patients	Stop evening alcohol; avoid sedatives/hypnotics where possible.	Lowers event duration/frequency; improves PAP tolerance.
PAP - CPAP (first-line)	Moderate-severe OSA or symptomatic mild	Fixed-pressure CPAP after titration. If no lab titration, start auto-CPAP, then adjust.	Early review (1-4 weeks): adherence, residual AHI, leaks, side effects. Education and mask fit are key.

PAP choice in overlap / hypoventilation	OSA with COPD ("overlap") or hypoventilation	Choose CPAP vs BiPAP based on COPD severity, hypoventilation, and pressure needs; add pulmonary rehab and optimize comorbidities.	Monitor gas exchange (SpO_2, ± CO_2). Consider backup rate if hypoventilation.
BiPAP (BPAP)	1) CPAP intolerance (exhalation discomfort) 2) Very high pressure needs (often >15-18 cm H_2O) 3) Obesity hypoventilation / significant hypoventilation	Lower EPAP with adequate IPAP to maintain airway and comfort; reduces aerophagia and improves adherence.	Track adherence and residual events; adjust IPAP/EPAP; consider ABG or transcutaneous CO_2 if hypoventilation.
Oral appliances (mandibular advancement)	Mild-moderate OSA, CPAP-intolerant, or isolated snoring	Custom, titratable device fitted by a qualified dentist in collaboration with a sleep physician.	Dental follow-up for bite/TMJ issues; repeat sleep testing to confirm efficacy. Often less effective than CPAP but better than no therapy.

CHILDREN (PEDIATRIC OSA)

Aspect	Management of Children (pediatric OSA)
First-line	Adenotonsillectomy if adenotonsillar hypertrophy; treats the anatomic driver in many children.
Diagnostics	Prefer polysomnography (PSG) in lab; home sleep apnea testing (HSAT) generally not recommended. Lower event thresholds for severity.
Severity cut-offs (apnoea-hypopnoea index, AHI)	Stricter: mild 1-4.9, moderate 5-9.9, severe ≥10 events/h.
Medical therapy (mild cases)	Intranasal corticosteroid ± montelukast can help mild OSA, especially with allergic rhinitis/adenotonsillar hypertrophy.
Positive airway pressure (PAP) role	CPAP/Bilevel (BiPAP) for persistent OSA after surgery, obesity-related OSA, craniofacial/neuromuscular disorders, or when surgery not possible. Mask fit and adherence coaching for growth/comfort.
Orthodontic/craniofacial	Rapid maxillary expansion (RME) or mandibular advancement in selected craniofacial phenotypes; early orthodontic input.
Weight management	Strong emphasis if overweight/obese; combine family-based lifestyle programs; monitor growth and puberty.
Surgery beyond adenotonsillectomy	Tailored by drug-induced sleep endoscopy (DISE) when residual disease; tongue-base or multilevel procedures at specialty centers. Tracheostomy for refractory syndromic/severe cases.
Special populations	Down syndrome, craniofacial syndromes, neuromuscular disease: high persistence after surgery → early PAP planning; enuresis, growth failure, behavior/learning issues are key outcomes.

Peri-operative risk	Higher post-op respiratory complications → inpatient monitoring after adenotonsillectomy for severe OSA or comorbidities.
Follow-up & adherence	Reassess 6-12 weeks after surgery; repeat PSG if symptoms persist or high risk. Frequent mask refits as the child grows; family training is central.
Outcome focus	Daytime behavior, attention, school performance, growth, blood pressure, enuresis.

MANAGEMENT OF OSA AND CO-EXISTING CONDITIONS

Overlap syndrome (OSA + COPD/other obstructive disease)

Start with CPAP. Improves sleep quality, lowers AHI, reduces nocturnal hypoxemia.

Mild-moderate COPD: CPAP usually sufficient; stabilizes airway and eases cardiovascular strain.

BiPAP (BPAP) preferred in case of

- Chronic hypercapnia, significant ventilatory impairment, or failed CPAP titration. Useful with respiratory muscle fatigue or severe COPD.
- BiPAP provides differential inspiratory/expiratory support → better CO_2 clearance and ventilation.

Monitoring: Regularly monitor arterial/venous blood gases, nocturnal oximetry, and device adherence/efficacy data to adjust settings.

Hypoventilation Syndromes (such as Obesity Hypoventilation Syndrome (OHS) or Chronic Hypercapnic Respiratory Failure)

OHS is defined as obesity (body mass index ≥30 kg/m²) with awake arterial $PaCO_2$ ≥45 mm Hg, not explained by other causes of hypoventilation (severe COPD, neuromuscular/chest-wall disorders, drug-induced hypoventilation) usually coexists with sleep-disordered breathing. Sleep study (PSG/HSAT) shows obstructive sleep apnoea or related SDB

AASM sleep hypoventilation rule (supporting):

- During sleep: $PaCO_2$ >55 mm Hg for ≥10 min, or
- Rise ≥10 mm Hg from awake to >50 mm Hg for ≥10 min (via ABG or validated transcutaneous CO_2).

1. **Confirm and stage**
   - History/exam; screen for OSA.
   - Baseline tests: ABG (daytime $PaCO_2$), nocturnal oximetry; transcutaneous CO_2 if available.
   - Identify contributors: sedatives/opioids, hypothyroidism, COPD/asthma control, neuromuscular disease.
2. **Stabilize contributors**
   - Stop/reduce respiratory depressants.
   - Treat nasal obstruction, hypothyroidism, COPD/asthma flares.
   - Start weight-management plan (diet, activity; consider bariatric referral in severe obesity).
3. **Initiate PAP (start with CPAP)**
   - Indication: OSA present and hypoventilation mild.
   - Fit mask, teach use/cleaning; start fixed or auto-CPAP if lab titration not available.
4. **Early reassessment (1-2 weeks)**
   - Check symptoms, adherence, leaks, residual events.
   - Repeat nocturnal oximetry; consider ABG (morning $PaCO_2$) or $TcCO_2$ trend.
5. **Escalate to BiPAP (BPAP) when**
   - Persistent daytime hypercapnia or nocturnal hypoventilation despite optimized CPAP.
   - CPAP intolerance (exhalation discomfort, aerophagia) or need for very high pressures.
   - Significant ventilatory impairment/respiratory muscle fatigue.
6. **Set up BiPAP**
   - Mode: **S** (spontaneous) if adequate drive; S/T (backup rate) if hypoventilation or central events.
   - Titrate **EPAP** to abolish obstructive events; titrate IPAP to improve ventilation/CO_2 clearance and comfort.

7. **Titration and optimization**
   - Prefer attended PSG titration (or structured titration with $TcCO_2$/oximetry).
   - Adjust settings based on symptoms, device data (residual AHI/REI, leak), oximetry/carbon dioxide CO_2.

8. **Adjuncts**
   - If SpO_2 remains low despite optimized PAP, add supplemental oxygen cautiously.
   - Pulmonary rehab, vaccinations, sleep-position coaching; manage cardiovascular/metabolic comorbidities.

Follow-up schedule: 1-2 weeks after any change then 1-3 months and then every 6-12 months. Track adherence, ESS/sleepiness, weight, ABG/$TcCO_2$ (as indicated), device downloads.

OHS specifics: Prioritize BiPAP when hypercapnia persists on CPAP. Expect improvements in survival, hospitalizations, and daily function; continue weight-loss program alongside PAP.

Red flags / higher-level care: Acute decompensated hypercapnic respiratory failure, progressive CO_2 rise, or refractory hypoxemia → urgent hospital-based NIV and specialist review.

SURGICAL OPTIONS (REFER TO TERTIARY CARE)

Consider surgery in case of

- Moderate-severe OSA with failed or intolerant CPAP (or oral appliance) and identifiable anatomic obstruction.
- Recurrent PAP nonadherence despite optimization, or anatomy unlikely to respond to PAP alone (e.g., severe retrognathia).
- Goal is symptom relief, AHI reduction, accident-risk reduction, and cardiometabolic risk mitigation.

Procedure-specific indications and steps

- Nasal surgery (septoplasty, turbinate reduction) in case of clinically significant nasal obstruction limiting sleep or PAP tolerance. It improves airflow and PAP adherence; rarely curative for OSA alone.

- Uvulopalatopharyngoplasty (UPPP) (often with tonsillectomy) in case of primary oropharyngeal/palatal collapse (large tonsils, redundant soft palate), BMI preferably <30-32, CPAP failure. It has modest durability; <50% maintain significant AHI improvement beyond 1 year.
- Maxillomandibular advancement (MMA) in retrognathia/mandibular deficiency, multilevel collapse, severe OSA, failed CPAP and soft-tissue surgery. Predictors of better response include younger age, smaller neck circumference, clear craniofacial restriction.
- Hypoglossal nerve stimulation (HNS) in case of adults with moderate-severe OSA, CPAP-intolerant, minimal central/mixed apneas, no complete concentric palatal collapse on drug-induced sleep endoscopy (DISE), BMI within program threshold (often ≤32-35). Results in substantial AHI reduction (median ~68% at 12 months) and improved sleepiness.
- Tracheostomy (definitive bypass) in case of refractory, life-threatening OSA not amenable to other options, severe craniofacial anomalies, or urgent airway control.
- Contraindications / poor candidates (procedure-dependent)
  - HNS: BMI above threshold, neuromuscular disease, hypoglossal palsy, significant cardiopulmonary instability, recent major cardiac events/arrhythmias, uncontrolled hypertension, active psychiatric illness, other non-respiratory sleep disorders, complete concentric palatal collapse on DISE.
  - UPPP/MMA/general: uncontrolled medical risk, inability to tolerate anesthesia, poor oral health (for MMA), unrealistic expectations.
- **Discuss Adverse effects with patient**
  - UPPP: velopharyngeal insufficiency, dysphagia, voice change; variable long-term efficacy.
  - MMA: numbness (V3), malocclusion, surgical risks.
  - HNS: tongue soreness/abrasion, device discomfort/malfunction (uncommon).
  - Nasal surgery: bleeding, crusting; may need medical therapy afterward.

Documentation (for every surgical pathway)

CPAP/oral appliance trials and reasons for failure, DISE findings, chosen procedure and rationale, informed consent on expected benefit vs need for adjunct PAP, and plan for postoperative PSG and follow-up.

PHARMACOLOGICAL THERAPY

There is no primary drug that treats the obstruction. Wake-promoting agents are not substitutes for treating the underlying airway obstruction. Pharmacotherapy is adjunctive and used in select scenarios:

1. **Nasal corticosteroids:** if nasal obstruction contributes (e.g., allergic rhinitis), use per standard allergic rhinitis guideline.
2. **Weight-loss medications** may be considered in obesity management integrated with overall OSA care, following obesity guidelines.
3. **Modafinil / Armodafinil: 3. Modafinil / Armodafinil:** for residual excessive daytime sleepiness after optimal OSA therapy (only after confirming adherence to CPAP and ruling out other causes). Dose per package insert, modafinil 200 mg once daily in the morning. **Caution**: psychiatric history, cardiovascular disease.

ASSESSMENT OF RESPONSE

Assessment of response to therapy combines symptoms, objective data, comorbidity metrics, and adverse effects.

Domain	What to assess	Metrics / thresholds	Action if not on target
Symptoms	Daytime sleepiness, fatigue, cognition, driving risk	ESS (Epworth) >10 = excessive sleepiness; track change from baseline. (STOP-BANG is for risk screening, not response tracking.)	Check adherence/device data; optimize PAP; address comorbid insomnia/PLMs; consider repeat sleep study.
Device data (CPAP/BiPAP)	Usage, leaks, residual events	Adherence: ≥4 h/night on ≥70% nights. Leak: within manufacturer's acceptable range. Residual AHI/REI: aim <5/h (or ↓ ≥50% from baseline if severe).	Low use - coaching, mask refit, desensitization. High leak - refit, alternate mask, chin strap, humidification. High residual AHI - pressure adjustment,
Comorbidity metrics	BP, weight, HbA1c/lipids (if relevant), AF burden/ arrhythmias	BP toward target; weight trend stable/declining; glycemic and lipid control improving	Intensify risk-factor management; coordinate with primary/cardiology/ endocrinology; ensure PAP is effective.
Adverse effects	Mask discomfort, nasal dryness/congestion, aerophagia, skin issues	Patient-reported	Mask change, heated humidification, nasal steroids/saline, pressure relief features, adjust EPAP/ IPAP, treat rhinitis; consider dental/oral appliance if persistent intolerance.

Early follow-up	Efficacy and fit after start	Timing: 1-3 months from initiation (earlier if issues)	Review data, adjust settings/interface, reinforce use and cleaning; address side effects.
Ongoing follow-up	Stability and adherence	Timing: every 6-12 months when stable	Update symptoms, download data, check BP/weight; refresh education; plan annual supplies.
Triggers for earlier review	Symptom return, large weight change, new meds/ conditions, adherence drop	Weight change: ≥10%; new cardiopulmonary/ neuro issues; device use <4 h/night or residual AHI	Reassess with device data; consider HSAT/PSG; retitrate pressures; evaluate for alternate therapy (OAT, surgery, HNS) if PAP failure.
When to repeat sleep testing	Persistent symptoms despite “adequate” use, discordant device data	Good adherence + symptoms or high residual events, or clinical change	Repeat HSAT/PSG (± titration). Rule out central events, positional changes, PLMs, or new comorbidity.
Before escalate/ de-escalate	Objective use, residual symptoms, residual AHI, weight, new meds/ conditions	Confirm ≥4 h/70%, symptom trend, device AHI, weight trend	If escalating: optimize PAP - BiPAP/ advanced titration or add OAT/ adjuncts, then refer. If de-escalating: trial step-down only after sustained control, with close monitoring.

REFERRAL STRATEGY

Care level	Core tasks	Diagnostics / treatments available	When to escalate
Primary care	Screen; initial risk stratification; lifestyle counseling (weight, alcohol/sedatives, sleep hygiene); start positional therapy; coordinate next steps	Use validated tools (STOP-BANG/NoSAS/ ESS); refer for HSAT/PSG where available	If diagnosis uncertain/complicated; suspected central apnea or hypoventilation; severe cardiopulmonary disease; CPAP intolerance despite basic optimization; potential surgical candidacy
Secondary care	Arrange HSAT or refer for PSG; initiate CPAP with basic support after specialist input; manage common comorbidities; trial oral appliance with dental collaboration	HSAT setup/interpretation; CPAP education/mask fitting; basic titration adjustments; comorbidity optimization	Same triggers as primary plus: persistent symptoms or residual AHI despite good adherence; need for advanced titration; evaluation for HNS or multilevel surgery
Tertiary care	Manage complex/refractory OSA; overlap syndromes; severe cardiopulmonary comorbidity; pediatric transition; evaluate for surgery/HNS	In-lab PSG with advanced titration; DISE; surgical assessment (UPPP/MMA/ nasal); HNS work-up/ implant programs; NIV (BiPAP/S/T) for hypoventilation	-

COMPLICATIONS

System / complication	Mechanism (why OSA causes it)	Clinical consequences	Monitoring / mitigation
Hypertension	Hypertension	Sustained or resistant HTN	BP at each visit; optimize PAP use; add standard antihypertensives
Coronary artery disease & MI	Oxidative stress, inflammation, platelet activation, metabolic dysregulation	Angina, myocardial infarction, higher cardiac events	Risk-factor control (lipids, diabetes), PAP adherence, avoid nocturnal hypoxemia
Atrial fibrillation & arrhythmias	Autonomic instability, atrial stretch from pressure swings/ hypoxia	AF recurrence, other tachy/bradyarrhythmias	ECG/ambulatory monitoring in symptomatic pts; PAP lowers AF recurrence risk
Heart failure (HFrEF/HFpEF)	Afterload ↑, nocturnal hypoxemia, negative intrathoracic pressure swings	Decompensation, reduced exercise tolerance	Guideline-directed HF therapy; PAP; manage fluid/sodium
Stroke & cerebrovascular disease	Endothelial dysfunction, hypercoagulability, BP variability	Ischemic stroke risk increases; worse recovery	Control BP, lipids; PAP; screen for AF
Metabolic syndrome & insulin resistance	Sleep fragmentation + hypoxia → cortisol and sympathetic drive	Central obesity, dyslipidemia, type 2 diabetes	HbA1c, lipids, waist/BMI; weight loss, PAP, diet/exercise
Neuropsychiatric effects	Sleep fragmentation, hypoxia → neural dysfunction	Depression, cognitive impairment, slowed processing	Screen mood/cognition; treat OSA, consider CBT, optimize sleep hygiene
Excessive daytime sleepiness	Fragmented sleep, unresolved apneas	Motor vehicle crashes, workplace errors	ESS tracking; counsel on driving safety; ensure PAP efficacy
Pulmonary hypertension	Chronic hypoxic vasoconstriction, vascular remodeling	Dyspnea, right-heart strain	Echo if suspected; treat OSA, manage comorbid lung/heart disease

PROGNOSIS AND PROGRESSION

With treatment, short-term outcomes are good, but long-term prognosis depends on sustained therapy and control of comorbidities. Weight gain, aging, and poor adherence drive progression; nearly half stop CPAP in the first month, which is the biggest barrier to benefit. Untreated or non-adherent patients face higher risks of complications as above, crashes, and higher healthcare costs. Consistent CPAP use improves ventricular mechanics and lowers these risks, underscoring the need for adherence and regular follow-up.

PREVENTION AND PROMOTION

Prevention and promotion for OSA focus on awareness, early detection, and integration into broader health systems.

- Public campaigns should highlight the link between obesity, alcohol or sedative use, poor sleep hygiene, and sleep apnea risk.
- Early screening in high-risk groups (obesity, hypertension, diabetes, resistant hypertension)
- Integrate sleep health into routine noncommunicable disease programs.
- Sleep health metrics and risk assessment should be embedded into routine noncommunicable disease programs to normalize evaluation and leverage existing touchpoints.
- Workplace safety campaigns about sleepiness especially those involving driving or safety-sensitive tasks to address the dangers of sleepiness, promote fatigue risk management, and encourage employees to seek evaluation when symptoms arise.
- Evaluate for daytime sleepiness such as for in job such as drivers, pilots, etc.

PATIENT EDUCATION

- During sleep the airway repeatedly narrows or closes. This causes low oxygen, broken sleep, and stress on the heart and brain.
- Treatment improves daytime alertness, blood pressure, heart rhythm stability, and thinking. It lowers crash risk.

CPAP/BiPAP: hands-on training

- Fit: choose a mask style that seals without pain. Adjust straps while lying down with the device on.
- Start-up: put the mask on first, then start the device. Use the ramp feature if high pressure feels uncomfortable.
- Humidifier: fill with distilled water to the line. Increase humidity for dryness, decrease for rainout.
- Exhalation relief or pressure support: use as instructed to ease breathing out.
- Mouth leak: if you breathe through your mouth with a nasal mask, add a chin strap or try a full-face mask.

- Do not change pressure settings unless your clinician tells you to.

Troubleshooting quick guide

- Dry nose or mouth: raise humidity, add heated tubing, use saline spray at night, treat nasal congestion.
- Mask leaks: refit straps, try a different size or mask type, check cushion wear. Replace worn parts.
- Aerophagia or bloating: review pressures with your clinician, try a lower ramp start, consider BiPAP if persistent.
- Skin marks or rash: loosen straps slightly, use mask liners, rotate mask type, check for contact dermatitis.
- Claustrophobia or anxiety: practice wearing the mask while reading. Use short daily desensitization sessions.
- Persistent sleepiness despite use: confirm hours used and residual AHI with a download. You may need re-titration or a repeat sleep study.

Cleaning and maintenance

- Daily: empty humidifier, air-dry tank, wipe mask cushion.
- Weekly: wash mask, tubing, and tank with mild soap and warm water. Rinse and air-dry.
- Filters: check monthly and replace per manufacturer schedule.
- Travel: carry the device as hand luggage. Bring a universal plug if needed.

Expectations

- Snoring often improves in days. Daytime sleepiness may take 2 to 4 weeks to improve.
- Blood pressure and mood changes improve over weeks to months with regular use.

Red flags that need prompt review

- Ongoing excessive sleepiness despite regular use.
- New or worsening chest pain, palpitations, morning headaches, severe breathlessness.
- Rapid weight gain or loss, new snoring pattern, or repeated mask intolerance.

Instructions to patient or caregiver

- Use CPAP every night for at least 4 hours, preferably all night. Do not stop because of mild discomfort. Report problems early.
- Bring device data or app reports to visits. First check at 1 to 3 months, then every 6 to 12 months when stable.
- Keep the mask, tubing, humidifier tank, and filters clean.
- Sleep on your side if tests show a positional component.
- Keep good sleep habits: fixed bedtime and wake time, dark quiet room, no caffeine after mid-afternoon, no screens in bed. Keep a simple sleep diary and note daytime sleepiness.
- Avoid alcohol in the evening. Avoid benzodiazepines, opioids, and sedating antihistamines unless your clinician approves. Some antidepressants can worsen OSA; discuss changes with your prescriber.
- Never drive when sleepy. If you feel drowsy, do not start or pull over.
- Attend scheduled follow-ups and bring device data.
- Report new symptoms such as morning headaches, palpitations, heavy fatigue, or changing snoring.

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