

Sleep Medicine Basics: Implications for the General Practitioner



Miguel Meira e Cruz, DDS, PhD, FICD^{a,*}, Hrayr Attarian, MD^b,
David Gozal, MD, PhD^c, Meir Kryger, MD, FRCPC^d

KEYWORDS

- Sleep disorders • Primary care • Insomnia • Obstructive sleep apnea • COMISA
- Cognitive behavioral therapy • Circadian rhythm • Pediatric sleep medicine

KEY POINTS

- Sleep disorders are highly prevalent and underrecognized in primary care but can be effectively screened and managed using structured tools.
- A multidisciplinary approach is essential for accurate diagnosis and long-term management of sleep disturbances, especially in complex or comorbid cases.
- Behavioral interventions such as cognitive behavioral therapy-I and sleep hygiene education are first-line treatments of insomnia and circadian disturbances and should be integrated early.
- Primary care providers play a critical role in identifying, initiating management, and coordinating care for common sleep disorders.
- Pediatric sleep issues require age-specific considerations and collaboration with families to implement effective behavioral strategies.

INTRODUCTION

Sleep is a core pillar of human health, alongside nutrition and physical activity. Despite its role in physiologic, cognitive, emotional, and cardiometabolic regulation, it remains underrecognized in clinical practice.¹ While sleep disturbances are linked to chronic diseases, this knowledge is still insufficiently integrated into primary and preventive care.¹

General practitioners (GPs), who often first encounter and longitudinally follow patients, are well positioned to identify sleep disorders.² Yet, these conditions are

^a Sleep Unit, Centro Cardiovascular da Universidade de Lisboa (CCUL@Rise), Lisbon School of Medicine, Av. Prof. Egas Moniz MB, Lisbon 1649-028, Portugal; ^b Feinberg School of Medicine, Chicago, USA; ^c Joan C. Edwards School of Medicine, Marshall University, Huntington, USA; ^d Yale School of Medicine, New Haven, CT, USA

* Corresponding author.

E-mail address: macruz@medicina.ulisboa.pt

Med Clin N Am 110 (2026) 707–720

<https://doi.org/10.1016/j.mcna.2026.01.008>

[medical.theclinics.com](https://www.medical.theclinics.com)

0025-7125/26/© 2026 Elsevier Inc. All rights are reserved, including those for text and data mining, AI training, and similar technologies.

Abbreviations	
CBT-I	cognitive behavioral therapy for insomnia
COMISA	comorbid insomnia and sleep apnea
CPAP	continuous positive airway pressure
CRSWD	circadian rhythm sleep–wake disorders
ESS	Epworth Sleepiness Scale
GP	general practitioner
HSAT	home sleep apnea testing
ISI	Insomnia Severity Index
NREM	nonrapid eye movement
ODI	oxygen desaturation index
OSA	obstructive sleep apnea
PLMD	periodic limb movement disorder
PSG	polysomnography
REM	rapid eye movement
RLS	restless legs syndrome
RLS	restless legs syndrome
SCN	suprachiasmatic nucleus
SWS	slow-wave sleep

frequently overlooked, misattributed to lifestyle, or treated superficially, often due to time constraints, limited training in sleep medicine, and the nonspecific nature of sleep complaints.²

Epidemiologic data show high prevalence and clinical complexity. Insomnia symptoms affect up to 43% of adults, with chronic insomnia present in around 14%.³ Obstructive sleep apnea (OSA) affects an estimated 9% to 38% of adults, with nearly a billion affected globally.⁴ It is more common in males and increases with age, affecting up to 80% of those over 65.⁵ Restless legs syndrome (RLS) impacts 2.5% to 15% of adults, with daily symptoms in 2% to 3%, particularly in older women.⁶ Periodic limb movement disorder (PLMD) affects ~4% of adults,⁶ and circadian rhythm sleep–wake disorders (CRSWD) affect ~3% of adults, with up to 10% prevalence among those with sleep complaints.⁷

Sleep disorders often coexist. Insomnia and OSA, in particular, show high overlap: 30% to 50% of OSA patients have significant insomnia symptoms, and 30% to 40% of insomnia patients meet OSA criteria on sleep studies.⁸ These associations surpass chance, reflecting shared risks, overlapping pathophysiology, and bidirectional effects. In high-risk groups, greater than 50% of insomnia patients screen positive for OSA, and OSA patients show increased insomnia risk.^{8,9}

This overlapping condition, comorbid insomnia and sleep apnea (COMISA), represents a distinct clinical entity. It is linked to worse sleep quality, more daytime dysfunction, and greater health burden than either condition alone. Evidence suggests additive or synergistic effects on cardiometabolic risk.^{10,11}

This article offers a concise, practice-focused overview of sleep medicine for general medical professionals.

CORE PHYSIOLOGY OF SLEEP

Sleep is a dynamic and highly regulated biological process rather than a passive state of rest. It is essential for synaptic plasticity, immune function, metabolic regulation, emotional processing, and cardiovascular stability.¹² Two primary mechanisms govern sleep regulation as represented in Fig. 1: the homeostatic sleep drive (Process S) and the circadian timing system (Process C).

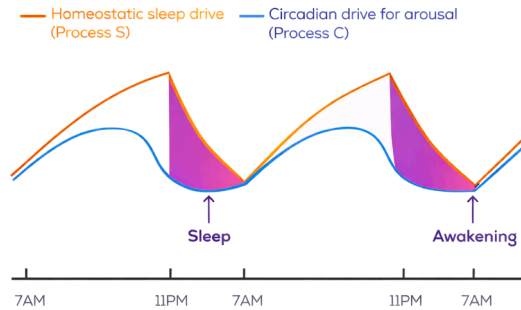


Fig. 1. The 2-process model of sleep regulation (Borbély Model). Sleep regulation is governed by 2 primary processes: Process S (homeostatic), which reflects sleep pressure that builds during wakefulness and dissipates during sleep, and Process C (circadian), which regulates the timing of sleep and wakefulness based on the body's internal biological clock. The interaction between these two processes determines optimal sleep–wake patterns. The homeostatic curve (Process S) decreases during deep sleep, while the circadian rhythm (Process C) modulates sleep propensity over the 24-hour cycle.

Process S reflects the accumulation of sleep pressure during wakefulness, primarily mediated by adenosine and other sleep promoting endogenous substances. This pressure dissipates during sleep, particularly during slow-wave sleep. Process C is controlled by the circadian clock, with the suprachiasmatic nucleus (SCN) acting as the master pacemaker while receiving inputs from multiple peripheral clocks. The SCN synchronizes physiologic rhythms to the external light–dark cycle via retinal input, coordinating hormone secretion, body temperature, appetite, alertness, and sleep propensity.¹³

Sleep architecture is organized into cycles of non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep, each cycle lasting approximately 90 to 110 minutes. NREM sleep is divided into 3 stages: N1 (light sleep), N2 (intermediate sleep), and N3 (also known as slow-wave sleep [SWS]) (Fig. 2.). SWS is particularly important for physical restoration, immune modulation, and memory consolidation. REM sleep is characterized by vivid dreaming, muscle atonia (except the diaphragm),

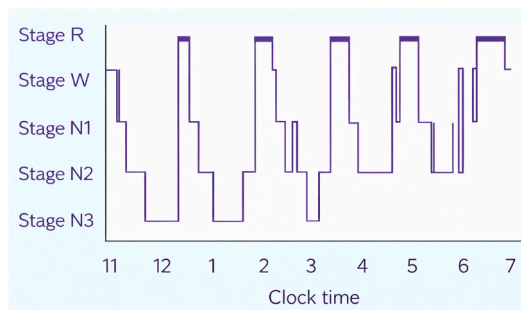


Fig. 2. Hypnographic representation of the cyclical structure of sleep throughout the night. Normal nighttime sleep is divided into non-REM (N1, N2, N3) and REM sleep. Each cycle lasts roughly 90 minutes, in adults, repeating approximately 4 to 6 times. Deep sleep (N3) predominates early in the night, while REM sleep increases toward morning. Understanding this architecture helps identify patterns relevant to major sleep disorders.

and heightened brain activity similar to wakefulness, and it plays a critical role in emotional regulation and learning.¹⁴

Across the lifespan, sleep patterns change substantially. Infants spend a greater proportion of sleep in active sleep, adolescents experience a physiologic delay in circadian phase, and older adults often have lighter, more fragmented sleep with reduced slow-wave sleep.¹⁵ While some of these changes are physiologic, they may also unmask or exacerbate pathologic sleep conditions.

Sleep also profoundly affects autonomic nervous system regulation. Parasympathetic activity predominates during NREM sleep, particularly SWS, resulting in lower heart rate and blood pressure. In contrast, REM sleep is associated with bursts of sympathetic activity. Disruption of these patterns, as seen in sleep-disordered breathing or chronic insomnia, contributes to cardiovascular instability and increased disease risk.¹⁶

CLINICAL RECOGNITION

Sleep disorders rarely present as isolated complaints. Instead, patients often report nonspecific symptoms such as fatigue, low energy, impaired concentration, mood disturbances, headaches, or poor work performance. These symptoms are frequently attributed to stress, aging, or psychiatric conditions without a systematic evaluation of sleep.¹⁷

A brief and focused sleep history can uncover clinically significant information. Key screening questions include whether the patient feels refreshed upon waking (sleep satisfaction),^{18,19} has trouble falling or staying asleep, snores loudly or has witnessed apneas (as per bed partner), or feels excessively sleepy during the day. Additional questions regarding sleep timing, variability across weekdays and weekends, nocturnal awakenings, and daytime consequences are invaluable.^{17,19}

Validated questionnaires support such initial clinical assessment. The Insomnia Severity Index (ISI) quantifies insomnia symptoms and related distress.²⁰ The Epworth Sleepiness Scale (ESS) assesses subjective daytime sleepiness.²¹ The STOP-BANG questionnaire provides a practical estimate of OSA risk²² and is particularly useful in primary care.

Certain features should prompt heightened clinical vigilance or referral. These include witnessed apneas with choking or gasping, excessive daytime sleepiness that compromises driving or occupational safety, parasomnias involving violent behavior, and symptoms suggestive of narcolepsy, such as cataplexy or sleep paralysis.

Integrating sleep questions into routine clinical encounters, particularly in patients with cardiovascular disease, metabolic disorders, chronic pain, or psychiatric diseases, can substantially improve diagnostic yield and patient outcomes.

MOST RELEVANT SLEEP DISORDERS IN PRIMARY CARE

Insomnia

Chronic Insomnia is defined as persistent difficulty initiating or maintaining sleep, or early morning awakenings, occurring at least 3 times per week for at least 3 months and associated with daytime functional impairments.³ It is the most prevalent sleep disorder encountered in primary care.

Chronic insomnia frequently coexists with anxiety, depression, chronic pain, and cardiometabolic disease. It may also coexist with other sleep disorders, particularly OSA. Chronic insomnia is increasingly recognized as an independent risk factor for hypertension, depression, and impaired quality of life.³

First-line treatment is cognitive behavioral therapy for insomnia (CBT-I), which includes stimulus control, sleep restriction, cognitive restructuring, relaxation techniques, and

sleep hygiene education.²³ Pharmacologic treatments should be reserved for short-term use or specific clinical scenarios and should be avoided at all cost as long-term solutions.³

Obstructive Sleep Apnea

OSA is characterized by recurrent upper airway collapse during sleep, resulting in intermittent hypoxia, sleep fragmentation, and sympathetic activation. Common symptoms include loud snoring, witnessed apneas, gasping or choking during sleep, nocturia, unrefreshing sleep, and excessive daytime sleepiness.⁵

OSA is strongly associated with obesity, male sex (menopausal females are as affected as males), craniofacial anatomy, and advancing age. It is a significant risk factor for systemic hypertension, atrial fibrillation ischemic heart disease, stroke, heart failure, dyslipidemia, diabetes, depression, cognitive impairments, and erectile dysfunction in males.^{5,24} Screening with STOP-BANG and ESS can guide referral for sleep testing either with PSG or home sleep apnea testing (HST).^{21,22} More recently, use of GLP-1 receptor agonists has emerged as an approved treatment option in selected cases.²⁴ Beyond their role in glycemic control, these agents promote clinically meaningful weight loss, which may translate into reductions in upper airway collapsibility, systemic inflammation, and cardiometabolic burden. In patients with obesity-related OSA, weight reduction induced by GLP-1–based therapies can lead to improvements in apnea–hypopnea index, daytime symptoms, and overall metabolic profile. Their use should be considered within a comprehensive management strategy that includes dietary counseling, physical activity, and behavioral interventions, and requires careful monitoring for tolerability and long-term adherence.

Management, in adults, includes continuous positive airway pressure (CPAP), mandibular advancement devices for selected patients, upper airway stimulation devices, and other surgical options in addition to positional therapy, weight reduction, and behavioral measures.²⁵

Comorbid Insomnia and Sleep Apnea

COMISA refers to the coexistence of insomnia and OSA, affecting up to 40% to 50% of patients with either condition. Patients with COMISA experience greater symptom burden, worse sleep quality, higher rates of anxiety and depression, and poorer adherence to CPAP therapy.^{8–11}

Recognition of COMISA is crucial, as treating only one component often leads to suboptimal outcomes. Integrated management combining CBT-I with OSA therapy improves adherence, sleep quality, and overall functioning.⁸

Restless Legs Syndrome and Periodic Limb Movement Disorder

Restless legs syndrome (RLS) is characterized by an imperious urge to move the legs, often accompanied by unpleasant sensations that worsen at rest and in the evening and are temporarily relieved by movement. It may be primary or secondary to iron deficiency, pregnancy, renal disease, or medication use.⁶

Evaluation should include assessment of ferritin levels, with supplementation recommended when ferritin is less than 75 ng/mL.⁵ Pharmacologic options include alpha-2-delta ligands and low-dose opiates.⁶

Circadian Rhythm Sleep-Wake Disorders

CRSWDs result from misalignment between endogenous circadian timing and external demands. Common forms include delayed sleep–wake phase disorder, shift work disorder, and travel jet lag disorder in addition to social jetlag lifestyle.

Management focuses on behavioral interventions, light therapy, and appropriately timed low-dose melatonin.²⁶

Narcolepsy and Central Hypersomnias

Narcolepsy is characterized by excessive daytime sleepiness, often accompanied by cataplexy, hallucinations, sleep paralysis, and disrupted nocturnal sleep. Diagnosis requires polysomnography preceded by 2 weeks of sleep diaries and followed by a multiple sleep latency test. Treatment includes wake-promoting agents and, when indicated, sodium oxybate.²⁷ Idiopathic hypersomnia differs from narcolepsy and is characterized by excessive sleep duration and sleep propensity with none of the other lacking REM-related features of narcolepsy.^{27,28}

Table 1 summarizes the diagnostic criteria and relevant screening notes on the most common sleep disorders encountered in primary care, including insomnia, OSA, RLS, narcolepsy, and circadian rhythm sleep–wake disorders.

Sleep and Comorbidities

Sleep disorders have far-reaching implications for physical and mental health. Short sleep duration, sleep fragmentation, and circadian misalignment are associated with increased risk of hypertension, coronary artery disease, arrhythmias, stroke, diabetes, obesity, depression, and anxiety.^{29,30–33} These often worsen the clinical scenario of OSA patients.^{34,35}

OSA contributes to sustained systemic low-grade sympathetic activation, oxidative stress, endothelial dysfunction, and inflammation. Insomnia, particularly when associated with objectively short sleep duration, independently increases cardiometabolic risk.³⁶ COMISA represents a particularly high-risk phenotype, combining the adverse effects of both disorders.⁸

Sleep also plays a critical role in pain modulation. Poor sleep increases pain sensitivity, while chronic pain disrupts sleep continuity, creating a self-perpetuating cycle.³⁷ In mental health, insomnia is both a symptom and a risk factor for mood disorders with REM sleep instability has been pointed out as a critical factor for emotional dysregulation.³⁸ Treating insomnia both prevents and improves psychiatric outcomes.³⁹

Table 1 Diagnostic criteria and screening notes for key sleep disorders		
Disorder	Diagnostic Criteria	Notes for General Practitioners
Chronic insomnia	Difficulty initiating/maintaining sleep ≥3 nights/week for ≥3 mo with daytime impairment	Use ISI ≥15 to indicate moderate-severe cases
OSA	AHI ≥5 events/hour with symptoms or AHI ≥15 regardless of symptoms	Screen using STOP-BANG ≥3
RLS	Urge to move legs worse at rest, in evening, relieved by movement	Check ferritin <75 µg/L
CRSWD	Mismatch between desired and actual sleep timing	Diagnose with sleep logs/actigraphy
Narcolepsy	EDS + cataplexy or MSLT with mean sleep latency ≤8 min and ≥2 SOREMPs	Refer for PSG + MSLT

Abbreviations: AHI, Apnea Hypopnea Index; EDS, Excessive Daytime Sleepiness; MSLT, Multiple Sleep Latency Test; SOREMP, Sleep Onset REM Period.

Emerging evidence links sleep disruption to neurodegenerative processes, highlighting the importance of sleep in cognitive aging and dementia prevention.⁴⁰

Diagnostic Approach in Primary Care

Primary care clinicians can effectively screen for sleep disorders using brief, validated questionnaires, sleep diaries, wearable devices, and targeted laboratory tests, many of which are simple, low-cost, and feasible within the general practice setting.

Screening Questionnaires and Rating Scales

Several standardized instruments support triage and initial assessment: ISI (moderate-severe insomnia ≥ 15)²⁰; ESS (>10 suggests excessive daytime sleepiness, raising suspicion for OSA, narcolepsy, or insufficient sleep)²¹; STOP-BANG (OSA risk ≥ 3)²²; PSQI (>5 indicates poor sleep quality and higher risk of comorbid psychiatric/metabolic burden)⁴¹; Berlin Questionnaire (OSA risk stratification based on snoring, sleepiness, and comorbidities)⁴²; IRLSSG criteria (RLS diagnostic checklist)⁴³; and chronotype tools such as MEQ⁴⁴ or MCTQ⁴⁵ to assess circadian misalignment and guide chronotherapeutic interventions.

Sleep Diaries

Sleep diaries provide a subjective yet valuable longitudinal view of sleep patterns, especially in patients with insomnia, circadian rhythm disorders, or behavioral sleep issues. They capture data on sleep latency, duration, nighttime awakenings, and variability, typically over 1 to 2 weeks.⁴⁶

Actigraphy and Wearable Technologies

Actigraphy, using wrist-worn devices, offers objective estimates of rest-activity cycles and sleep-wake patterns over extended periods (7–14 days). It is invaluable in diagnosing delayed sleep phase syndrome, fragmented sleep, or paradoxical insomnia, especially when polysomnography (PSG) is not readily available.⁴⁷

With technological advances, a growing number of consumer-grade and medical-grade wearables, including smartwatches, rings, and wristbands, have demonstrated moderate-to-high agreement with actigraphy and polysomnography for estimating sleep duration, timing, and, in some cases, oxygen saturation, and heart rate variability.⁴⁸

Some validated wearables are now capable of:

- Continuous oximetry (SpO₂ trends, desaturation events)
- Heart rate variability and electrocardiogram (ECG)
- Approximate sleep staging
- Detection of respiratory events (via surrogates like pulse oximetry or motion sensors)

These tools may support early screening for OSA, identify autonomic instability or confirm circadian misalignment, particularly when used in conjunction with sleep diaries or questionnaires. While they do not replace formal diagnostic testing, their convenience and accessibility make them valuable adjuncts in primary care and telemedicine contexts.^{49,50}

Overnight Pulse Oximetry

Nocturnal oximetry is a low-cost and widely available tool that can be used in patients with suspected moderate-to-severe OSA, especially when access to formal polysomnography is limited.^{50,51} Clinically significant oxygen desaturation (eg, $\geq 3\%$ drops, oxygen desaturation index (ODI) ≥ 5 events/hour recording) or patterns of intermittent

hypoxia and pulse variability can indicate a high likelihood of sleep-disordered breathing and support referral for definitive testing.^{49,51}

Interpretation of oximetry should consider:

- ODI
- Cumulative hypoxic burden
- Correlation with reported symptoms
- Comorbidities (eg, chronic obstructive pulmonary disease [COPD], heart failure) that may confound readings

In selected populations (eg, patients with chronic pain, hypertension, or insomnia with daytime fatigue), oximetry or wearable screening may prompt earlier recognition of hidden OSA or COMISA phenotypes, reducing delays in appropriate referral.

Indications for Referral and Advanced Testing

Referral for PSG or HSAT should be considered in patients with:

- High STOP-BANG or Berlin score
- Loud snoring witnessed apneas or choking episodes
- Abnormal oximetry or wearable data suggestive of OSA
- Suspected parasomnias or nocturnal seizures
- Symptoms suggestive of narcolepsy or idiopathic hypersomnia
- Treatment-resistant insomnia
- Children with atypical behaviors during sleep or significant daytime consequences

Laboratory Evaluation

Routine bloodwork may be appropriate in selected cases⁴⁰:

- Serum ferritin: Recommended in suspected RLS, especially if less than 75 µg/L⁵²
- Thyroid-stimulating hormone (TSH) and free thyroxine (T4): To rule out hypothyroidism in fatigue or hypersomnia
- Glicated hemoglobin (HbA1c), fasting glucose, and lipid panel: For metabolic risk assessment in suspected OSA, particularly if patient is overweight or obese
- Cortisol, iron panels, vitamin D: As needed based on comorbidities and systemic symptoms

First-Line Management

Many sleep disorders can be identified and managed in primary care using a stepped-care approach, beginning with sleep hygiene, behavioral strategies, and lifestyle changes, and escalating to pharmacologic or device-based interventions when needed.⁵³

Sleep Hygiene is a low-cost, foundational tool that supports CBT-I.⁵⁴ Core elements:

- Regular sleep–wake times
- Limiting caffeine, alcohol, nicotine after midafternoon
- Avoiding screens/heavy meals before bed
- Relaxing bedtime routine
- Using the bedroom only for sleep/intimacy
- Enhancing daytime light exposure and reducing evening artificial light

Chronobiologic guidance (eg, morning light, avoiding late-night light) helps adolescents and shift workers realign circadian rhythms.

CBT-I is first-line for chronic insomnia and adaptable to primary care.⁵⁵

Key techniques include

- Stimulus control (bed only when sleepy, leave bed if awake)
- Sleep restriction (limit time in bed to actual sleep)
- Cognitive restructuring (eg, challenge sleep myths)
- Relaxation (breathing, mindfulness, among others)

Brief formats (2–4 sessions or apps) are effective, especially in COMISA, where insomnia treatment enhances CPAP adherence.^{43,44} Digital CBT-I tools are valuable adjuncts to GP care.

CIRCADIAN RHYTHM DISORDERS: BEHAVIORAL AND LIGHT-BASED APPROACHES

Circadian Misalignment (eg, Delayed Sleep-Wake Phase Disorder [DSWPD])

Circadian misalignment can be managed with morning light exposure, evening melatonin (0.3–2 mg, 3–5 h before desired sleep), regular wake times, and reduced evening screen use or blue-light filters.⁵⁶ These are effective in adolescents, shift workers, and those with social jet lag.

Obstructive Sleep Apnea and Comorbid Insomnia and Sleep Apnea: Early Lifestyle Strategies

Before diagnosis or device use, OSA and COMISA management may begin with weight control and positional therapy.⁸ Patients with body mass index (BMI) ≥ 25 kg/m² should receive guidance on caloric restriction, Mediterranean-style diets, referrals to dietitians, and use of digital tracking tools. A 5% to 10% weight loss can significantly lower AHI or ODI and improve symptoms. This is critical in COMISA due to overlapping metabolic risks.

Physical activity improves both sleep and cardiometabolic health^{48,57} with morning walks favoring circadian alignment and mood regulation.

Restless Legs Syndrome and Periodic limb movement disorder

Initial RLS management includes⁵²:

- Iron supplementation if ferritin less than 75 µg/L
- Avoidance of medications that worsen symptoms (antihistamines, some antidepressants, dopamine antagonists)
- Encouraging regular moderate exercise, particularly in the morning or early afternoon
- Referral for pharmacologic treatment (eg, alpha-2-delta ligands) in moderate to severe cases⁵²

The Role of Wearables and Patient Feedback

Patients are increasingly using wearables to monitor sleep, physiologic variables (eg, SpO₂, heart rate) and physical activity. Clinicians can leverage these tools to

- Track bedtime consistency
- Monitor response to treatment
- Monitor changes after lifestyle interventions
- Increase patient engagement and self-efficacy

Validation and Expectation Management

Recognizing and validating a patient's sleep concerns build trust and enhances therapeutic alliance. Patients with insomnia or COMISA often feel dismissed or misunderstood, especially when diagnostic tests are unremarkable.

Clinicians should:

- Normalize some degree of sleep variability
- Emphasize function over perfection (eg, improved energy, mood, or alertness rather than total sleep duration)
- Set achievable goals collaboratively
- Reinforce progress using tools such as the ISI, PSQI, or patient-reported outcome diaries

Referral Pathways and Interdisciplinary Collaboration

Referral to sleep specialists is essential in complex or refractory cases, such as hypoventilation, opioid-related sleep-disordered breathing, suspected narcolepsy, injurious parasomnias, severe insomnia, or COMISA, to ensure accurate diagnosis and individualized treatment.⁵⁸

Interdisciplinary collaboration improves outcomes in both adults and children. Sleep physicians (neurologists, pulmonologists, and internists) lead diagnostics and manage complex cases. Clinical psychologists or behavioral sleep medicine specialists provide CBT-I and treat circadian and anxiety-related sleep issues.

Dentists trained in sleep medicine manage mild-to-moderate OSA with oral appliances and assess bruxism, orofacial pain, and TMD.^{59–63} In pediatrics, functional orthopedic and myofunctional strategies may be used.^{60,61} ENT specialists evaluate/surgically manage upper airway obstruction, especially in pediatric OSA.⁶⁴ Physiotherapists and exercise professionals support weight loss and physical conditioning.⁶⁵

Speech therapists assist in orofacial and upper airway rehabilitation in OSA, hypotonia, or dysphagia, and deliver myofunctional therapy when indicated.⁵³ Sleep technologists conduct and interpret sleep studies. Nurses and sleep educators reinforce therapy adherence. Pharmacists optimize pharmacologic regimens, ensuring safety and tapering when needed.

General practitioners initiate screening and treatment, manage comorbidities, refer as needed, and provide long-term care, ensuring continuity and integration across the patient's lifespan.

Special Considerations Across the Lifespan

Sleep disorders vary by age and are often underrecognized in children due to overlap with behavioral or developmental issues. Symptoms like irritability, inattention, or hyperactivity may reflect poor sleep rather than psychiatric conditions.⁶⁶

Sleep needs decrease with age: newborns (14–17h), toddlers (11–14h), school-aged children (9–11h), adolescents (8–10h). Yet many fail to meet these targets due to poor hygiene, screens, irregular schedules, academic demands, and circadian misalignment.⁶⁶

Pediatric sleep disorders include behavioral insomnia (sleep-onset association or limit-setting types), OSA, periodic limb movements, delayed sleep–wake phase disorder, and parasomnias like night terrors or sleepwalking. In adolescents, circadian delay and sleep deprivation are linked to mood issues and body image concerns.

Insomnia, RLS, and OSA are more frequent during pregnancy,⁶⁷ while older adults are vulnerable to sleep disruption and medication effects. Shift workers face chronic circadian misalignment with health risks and worsened sleep disorder outcomes.^{8,34,68–70}

Tailoring assessment and management to life stage is essential.

FUTURE DIRECTIONS AND TAKE-HOME MESSAGES

Sleep medicine is rapidly evolving. Advances in wearable and nonwearable technology, remote monitoring, artificial intelligence-derived applications, and digital therapeutics

are expanding access to diagnosis and behavioral interventions. Phenotyping tools may help tailor treatment in complex cases such as COMISA.

In summary, recognizing and addressing sleep disorders should not be viewed as an additional burden in general practice, but rather as an essential step toward holistic, preventive care. As our understanding of sleep deepens, so too must our commitment to translating that knowledge into practice, starting with the clinician who knows the patient best.

CLINICS CARE POINTS

- Sleep is a critical determinant of health yet remains underaddressed in general practice. General practitioners can play a transformative role by routinely assessing and managing sleep complaints.
- A brief sleep history, periodically supplemented with tools such as the Insomnia Severity Index, Epworth Sleepiness Scale, and STOP-BANG (eg, every 6–12 months), can effectively screen for common sleep disorders.
- Insomnia and obstructive sleep apnea are the most prevalent sleep problems, often coexisting (comorbid insomnia and sleep apnea), and require distinct yet integrated management strategies.
- Nonpharmacologic treatments such as cognitive behavioral therapy for insomnia, sleep hygiene guidance, and light therapy are effective and can be initiated in primary care.
- Referral to sleep specialists is essential in complex, high-risk, or treatment-resistant cases, and should be guided by clear clinical criteria.

DISCLOSURE

M. Kryger and M.M. e Cruz are scientific advisors of Bodimetrics, USA. H. Attarian and D. Gozal declare to have no conflict of interest.

REFERENCES

1. Grandner MA. Sleep, health, and society. *Sleep Med Clin* 2022;17(2):117–39.
2. Soenen S, Sweetman A, Balzer M, et al. Sleep health primary care clinical resource. *Aust J Gen Pract* 2024;53(6):349–55.
3. Paul AM, Salas RE. Insomnia. *Prim Care* 2024;51(2):299–310.
4. Benjafield AV, Ayas NT, Eastwood PR, et al. Estimation of the global prevalence and burden of obstructive sleep apnoea: a literature-based analysis. *Lancet Respir Med* 2019;7(8):687–98. Epub 2019 Jul 9. PMID: 31300334; PMCID: PMC7007763.
5. Anderson N, Tran P. Obstructive sleep apnea. *Prim Care* 2025;52(1):47–59.
6. Winkelman JW, Berkowski JA, DelRosso LM, et al. Treatment of restless legs syndrome and periodic limb movement disorder: an American Academy of Sleep Medicine clinical practice guideline. *J Clin Sleep Med* 2025;21(1):137–52. <https://doi.org/10.5664/jcsm.11390>.
7. Tomatsu S, Abbott SM, Attarian H. Clinical chronobiology: circadian rhythms in health and disease. *Semin Neurol* 2025;45(3):317–32.
8. Meira E, Cruz M, Sweetman A. Comorbid insomnia and sleep apnea: from research to clinical practice. *Semin Respir Crit Care Med* 2025;46(2):113–24.
9. Luciano YM, Porcacchia AS, Tufik S, et al. Prevalence and incidence of Co-Morbid insomnia and sleep apnea (comisa) in São Paulo, Brazil. *Chest* 2024;165(4):1004–8.

10. Meira E, Cruz M. Comorbid insomnia and sleep apnea: COMISA. *Dent Clin North Am* 2024;68(3):455–66.
11. Meira E, Cruz M, Kryger MH, et al. Comorbid insomnia and sleep apnea: mechanisms and implications of an underrecognized and misinterpreted sleep disorder. *Sleep Med* 2021;84:283–8.
12. Foster RG. Introduction and reflections on the comparative physiology of sleep and circadian rhythms. *J Comp Physiol B* 2024;194(3):225–31.
13. Pandi-Perumal SR, Paul S, Saravanan KM, et al. Clock-sleep communication. *Curr Mol Med* 2025;25(4):399–415.
14. Grace-Abraham N, Tran MN, Moore C, et al. Sleep in adults: normal sleep and its importance to health. *FP Essent* 2025;556:6–11.
15. Sun H, Ye E, Paixao L, et al. The sleep and wake electroencephalogram over the lifespan. *Neurobiol Aging* 2023;124:60–70.
16. Tobaldini E, Costantino G, Solbiati M, et al. Sleep, sleep deprivation, autonomic nervous system and cardiovascular diseases. *Neurosci Biobehav Rev* 2017;74(Pt B):321–9.
17. Robbins R, Quan SF. Sleep disorders. *NEJM Evid* 2024;3(10):EVIDra2400096.
18. Ohayon MM, Chen MC, Bixler E, et al. A provisional tool for the measurement of sleep satisfaction. *Sleep Health* 2018;4(1):6–12. Epub 2017 Dec 18. PMID: 29332682.
19. Ohayon M, Wickwire EM, Hirshkowitz M, et al. National sleep Foundation's sleep quality recommendations: first report. *Sleep Health* 2017;3(1):6–19. Epub 2016 Dec 23. PMID: 28346153.
20. Morin CM, Belleville G, Bélanger L, et al. The insomnia severity index: psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep* 2011;34(5):601–8.
21. Walker NA, Sunderram J, Zhang P, et al. Clinical utility of the Epworth sleepiness scale. *Sleep Breath* 2020;24(4):1759–65.
22. Cho T, Yan E, Chung F. The STOP-Bang questionnaire: a narrative review on its utilization in different populations and settings. *Sleep Med Rev* 2024;78:102007.
23. Walker J, Muench A, Perlis ML, et al. Cognitive behavioral therapy for insomnia (CBT-I): a primer. *Klin Spec Psihol* 2022;11(2):123–37.
24. Malhotra A, Grunstein RR, Fietze I, et al. Tirzepatide for the treatment of obstructive sleep apnea and obesity. *N Engl J Med* 2024;391(13):1193–205. Epub 2024 Jun 21. Erratum in: *N Engl J Med*. 2024 Oct 17;391(15):1464. doi: 10.1056/NEJMx240005. PMID: 38912654; PMCID: PMC11598664.
25. Ellender CM, Vakulin A, Stocks N, et al. Management of obstructive sleep apnoea in primary care. *Aust J Gen Pract* 2024;53(6):363–9.
26. Zee PC, Abbott SM. Circadian rhythm sleep-wake disorders. *Continuum* 2020;26(4):988–1002.
27. Blattner M, Maski K. Narcolepsy and idiopathic hypersomnia. *Sleep Med Clin* 2023;18(2):183–99.
28. Shahzadi M, Monderer R, Thorpy MJ. Understanding idiopathic hypersomnia: diagnosis, pathophysiology, and management. *Curr Opin Pulm Med* 2025;31(6):597–604.
29. Yiallourou S, Baril AA, Wiedner C, et al. Short sleep duration and hypertension: a double hit for the brain. *J Am Heart Assoc* 2024;13(21):e035132.
30. Ai S, Zhang J, Zhao G, et al. Causal associations of short and long sleep durations with 12 cardiovascular diseases: linear and nonlinear Mendelian randomization analyses in UK biobank. *Eur Heart J* 2021;42(34):3349–57.
31. Zhao J, Wang W, Wei S, et al. Fragmented sleep and the prevalence of hypertension in middle-aged and older individuals. *Nat Sci Sleep* 2021;13:2273–80.

32. Gottesman RF, Lutsey PL, Benveniste H, et al. Impact of sleep disorders and disturbed sleep on brain health: a scientific statement from the American heart association. *Stroke* 2024;55(3):e61–76.
33. Lal H, Verma SK, Wang Y, et al. Circadian rhythms in cardiovascular metabolism. *Circ Res* 2024;134(6):635–58.
34. Santos I, Rocha I, Gozal D, et al. Obstructive sleep apnea, shift work and cardiometabolic risk. *Sleep Med* 2020;74:132–40.
35. Ren R, Zhang Y, Yang L, et al. Association between arousals during sleep and hypertension among patients with obstructive sleep apnea. *J Am Heart Assoc* 2022;11(1):e022141.
36. Tasali E, Pamidi S, Covassin N, et al. Obstructive sleep apnea and cardiometabolic disease: obesity, hypertension, and diabetes. *Circ Res* 2025;137(5):764–87.
37. Haack M, Simpson N, Sethna N, et al. Sleep deficiency and chronic pain: potential underlying mechanisms and clinical implications. *Neuropsychopharmacology* 2020;45(1):205–16.
38. Riemann D, Dressle RJ, Benz F, et al. Chronic insomnia, REM sleep instability and emotional dysregulation: a pathway to anxiety and depression? *J Sleep Res* 2025;34(2):e14252.
39. Irwin MR. Insomnia and inflammation conspire to heighten depression risk: implications for treatment and prevention of mood disorders. *Biol Psychiatry* 2025;98(11):819–29.
40. Lyall LM, Wyse CA, Graham N, et al. Association of disrupted circadian rhythmicity with mood disorders, subjective wellbeing, and cognitive function: a cross-sectional study of 91 105 participants from the UK Biobank. *Lancet Psychiatry* 2018;5(6):507–14.
41. Buysse DJ, Reynolds CF 3rd, Monk TH, et al. The Pittsburgh sleep quality index: a new instrument for psychiatric practice and research. *Psychiatry Res* 1989; 28(2):193–213.
42. Senaratna CV, Perret JL, Matheson MC, et al. Validity of the Berlin questionnaire in detecting obstructive sleep apnea: a systematic review and meta-analysis. *Sleep Med Rev* 2017;36:116–24.
43. Sharma A, Rai NK, Singh R. Clinical profile of restless leg syndrome and its effect on sleep and quality of life. *J Family Med Prim Care* 2025;14(4):1359–67.
44. Horne JA, Ostberg O. A self-assessment questionnaire to determine morningness-eveningness in human circadian rhythms. *Int J Chronobiol* 1976;4(2):97–110.
45. Roenneberg T, Pilz LK, Zerbini G, et al. Chronotype and social Jetlag: a (Self-) critical review. *Biology* 2019;8(3):54. Published 2019 Jul 12.
46. Klier K, Wagner M. Agreement of sleep Measures-A comparison between a sleep diary and three consumer wearable devices. *Sensors (Basel)* 2022;22(16):6189.
47. Neikrug AB. Actigraphy in clinical sleep medicine. *Sleep Med Rev* 2023;68: 101767.
48. Asgari Mehrabadi M, Azimi I, Sarhaddi F, et al. Sleep tracking of a commercially available smart ring and smartwatch against medical-grade actigraphy in everyday settings: instrument validation study. *JMIR mHealth uHealth* 2020;8(10):e20465.
49. de Zambotti M, Cellini N, Goldstone A, et al. Wearable sleep technology in clinical and research settings. *Med Sci Sports Exerc* 2019;51(7):1538–57.
50. Meira E, Cruz M, Chen E, et al. A wearable ring oximeter for detection of sleep disordered breathing. *Respir Med* 2025;242:108092.
51. Blanchard M, Imler T, Hu WH, et al. Heart rate response and cardiovascular risk during obstructive sleep apnoea: an easy biomarker derived from pulse oximetry. *Eur Respir J* 2025;65(5):2401883.

52. Winkelman JW, Berkowski JA, DelRosso LM, et al. Treatment of restless legs syndrome and periodic limb movement disorder: an American Academy of Sleep Medicine clinical practice guideline. *J Clin Sleep Med* 2025;21(1):137–52.
53. King S, Damarell R, Schuwirth L, et al. Knowledge to action: a scoping review of approaches to educate primary care providers in the identification and management of routine sleep disorders. *J Clin Sleep Med* 2021;17(11):2307–24.
54. De Pasquale C, El Kazzi M, Sutherland K, et al. Sleep hygiene - what do we mean? A bibliographic review. *Sleep Med Rev* 2024;75:101930.
55. Buenaver LF, Townsend D, Ong JC. Delivering cognitive behavioral therapy for insomnia in the real world: considerations and controversies. *Sleep Med Clin* 2019;14(2):275–81.
56. Brown TM, Brainard GC, Cajochen C, et al. Recommendations for daytime, evening, and nighttime indoor light exposure to best support physiology, sleep, and wakefulness in healthy adults. *PLoS Biol* 2022;20(3):e3001571.
57. Vanderlinden J, Boen F, van Uffelen JGZ. Effects of physical activity programs on sleep outcomes in older adults: a systematic review. *Int J Behav Nutr Phys Activ* 2020;17(1):11. Published 2020 Feb 5.
58. Manetta IP, Ettlin D, Sanz PM, et al. Mandibular advancement devices in obstructive sleep apnea: an updated review. *Sleep Sci* 2022;15(Spec 2):398–405.
59. Manetta IP, Meira E, Cruz M. Phenotyping OSA: conceptualization and implications for treatment with mandibular advancement devices. *Dent Clin North Am* 2024;68(3):485–93.
60. Faber J, Mota A, Ho LI, et al. The role of orthodontists in the multidisciplinary management of obstructive sleep apnea. *Prog Orthod* 2024;25(1):40. Published 2024 Nov 4.
61. Meira E, Cruz M, Guilleminault C, et al. Rapid maxillary expansion in COMISA: preliminary observations from a 12 years follow-up. *Sleep* 2025;48(3):zsae306.
62. Simmons M, Sheats R, Meira E, et al. Emerging dental therapies for sleep disorders: evidence synthesis from the American Academy of Dental Sleep Medicine 2024 Consensus. *J Am Dent Assoc* 2026;157(1):10–9.
63. Quah B, Sng TJH, Yong CW, et al. Orthognathic surgery for obstructive sleep apnea. *Oral Maxillofac Surg Clin North Am* 2023;35(1):49–59.
64. Dipalma G, Inchingolo AM, Palumbo I, et al. Surgical management of pediatric obstructive sleep apnea: efficacy, outcomes, and Alternatives-A systematic review. *Life* 2024;14(12):1652. Published 2024 Dec 12.
65. Herrero BA, Lam JTAT, Exposto FG, et al. Interprofessional Collaboration in dentistry: role of physiotherapists to improve care and outcomes for chronic pain conditions and sleep disorders. *J Oral Pathol Med* 2020;49(6):529–37.
66. Meltzer LJ, Williamson AA, Mindell JA. Pediatric sleep health: it matters, and so does how we define it. *Sleep Med Rev* 2021;57:101425.
67. Liamsombut S, Tantrakul V. Sleep disturbance in pregnancy. *Sleep Med Clin* 2022;17(1):11–23.
68. Boivin DB, Boudreau P, Kosmadopoulos A. Disturbance of the circadian system in shift work and its health impact. *J Biol Rhythm* 2022;37(1):3–28.
69. Nobre B, Rocha I, Morin CM, et al. Insomnia and circadian misalignment: an underexplored interaction towards cardiometabolic risk. *Sleep Sci* 2021;14(1):55–63.
70. Meira e Cruz M, Pereira S, Rodrigues MJ, et al. Altered circadian patterns of diurnal blood pressure in clinical phenotypes of bruxism: a pilot study on autonomic imbalance in healthy young adults. *Dent and Med Prob*. In Press.