Sleep Medicine for Dentists:
An Evidence-Based Overview, Second Edition

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To our students, patients, and research associates who have contributed to the progress in dental sleep medicine.

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Second Edition

SLEEP MEDICINE FOR DENTISTS

AN EVIDENCE-BASED OVERVIEW

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Contents

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Foreword by David Gozal viii
Preface ix
In Memoriam x
Contributors xi
Abbreviations xvi

Section I Introduction to Dental Sleep Medicine

1 The Nature and Structure of Sleep *3*
Cibele Dal Fabbro, Monica L. Andersen, Gilles J. Lavigne

2 Sleep Neurobiology *9*
Florin Amzica, Gilles J. Lavigne, Barry J. Sessle, Florian Chouchou

3 A Dental Perspective on the Classification of Sleep Disorders *15*
Raphaël C. Heinzer, Peter A. Cistulli, Alberto Herrero Babiloni, Gilles J. Lavigne

4 Role of Dentists in Sleep Medicine *22*
Gilles J. Lavigne, Raphaël C. Heinzer, Cibele Dal Fabbro, Michael T. Smith,
Jean-François Masse, Fernanda R. Almeida, Takafumi Kato, Frank Lobbezoo, Peter A. Cistulli

Section II Sleep Breathing Disorders

5 Overview of Guidelines/Protocols for SDB *29*
Galit Almoznino, Rafael Benoliel, Frank Lobbezoo, Luc Gauthier

6 Sleep-Related Breathing Disorders *35*
Joseph M. Duncan, Andrew S.L. Chan, Richard W.W. Lee, Peter A. Cistulli

7 Pathophysiology of OSA *39*
Danny J. Eckert

8 Mouth Breathing, Dentofacial Morphology, and SDB *44*
Stacey D. Quo, Benjamin Pliska, Nelly Huynh

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9 Long-Term Consequences of OSA 50
Frédéric Gagnadoux

10 Periodontal Diseases and OSA 55
Maria Clotilde Carra, Joerg Eberhard, Peter A. Cistulli

11 Clinical Approaches to Diagnosis of Adult OSA 60
Anna M. Mohammadieh, Richard W.W. Lee, Andrew S.L. Chan

12 Imaging in OSA 66
Kate Sutherland, Richard J. Schwab, Lynne E. Bilston

13 An Overview of OSA Treatment in Adults 72
Jesse W. Mindel, Ryan Donald, Ulysses J. Magalang

14 Diagnosis and Management of Pediatric OSA 77
Dimple Goel, Dominic A. Fitzgerald

15 Orofacial Orthopedic Treatment 82
Stacey D. Quo, Benjamin Pliska, Nelly Huynh

16 Oral Appliance Therapy 87
Fernanda R. Almeida, Kate Sutherland, Peter A. Cistulli

17 Upper Airway Surgical Management of OSA 92
Leon Kitipornchai, Stuart G. MacKay

18 Emerging Therapies for OSA 96
Olivier M. Vanderveken

19 Risks of Anesthesia in Patients with OSA 100
David R. Hillman

20 Myofunctional Therapy for OSA 104
Wen-Yang Li, Jean-François Masse, Frédéric Sériès

21 Precision Medicine Approaches for OSA 107
Kate Sutherland, Peter A. Cistulli

22 Genetics of SDB 113
Sutapa Mukherjee, Lyle J. Palmer

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Section III Sleep Bruxism: From Oral Behavior to Disorder

23 Definitions, Epidemiology, and Etiology of SB *119*
Frank Lobbezoo, Jari Ahlberg, Daniel A. Paesani, Ghizlane Aarab

24 Clinical Approaches to Diagnosis of SB *124*
Kiyoshi Koyano, Yoshihiro Tsukiyama, Peter Wetselaar

25 SB as a Comorbid Condition of Other Sleep-related Disorders *129*
Ghizlane Aarab, Ramesh Balasubramaniam, Milton Maluly Filho, Gilles J. Lavigne

26 Physiologic Mechanisms Associated with SB Genesis *135*
Takafumi Kato, Kazuo Okura, Guido M. Macaluso, Gilles J. Lavigne

27 Psychosocial Factors in Sleep and Awake Bruxism and Other Oral Parafunctions *142*
Richard Ohrbach, Sylvia D. Kreibig, Ambra Michelotti

28 Genetic and Environmental Factors in SB *146*
Kazuyoshi Baba, Yuka Abe, Samar Khoury, Frank Lobbezoo

29 Consequences of SB on the Dentition, Dental Restorations, and Implants and How to Mitigate Them *152*
Sandro Palla, Iven Klineberg, Mauro Farella

30 Behavioral, Dental, Pharmacologic, and Alternative Management of SB *157*
Daniele Manfredini, Charles R. Carlson, Ephraim Winocur, Frank Lobbezoo

31 SB in Children and Adolescents *162*
Nelly Huynh, Naomi Kadoch, Christian Guilleminault

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Section IV Sleep and Orofacial Pain

32 Definition and Classification of Orofacial Pains *169*
Alberto Herrero Babiloni, Donald R. Nixdorf

33 Pathophysiologic Conceptualizations of the Transition from Acute to Chronic Pain *175*
Claudia M. Campbell, Robert R. Edwards, Janelle E. Letzen

34 Mechanisms Underlying the Interactions Between Sleep Deficiency and Pain *178*
Monika Haack, Navil Sethna

35 Behavioral and Pharmacologic Approaches to Manage Chronic Pain Comorbid with Sleep Disturbances *183*
Monika Haack, Navil Sethna

36 Association and Putative Causality of Orofacial Pain Conditions and Sleep Disturbances *187*
Peter Svensson, Lene Baad-Hansen, Taro Arima, Antoon De Laat

37 Sleep and Headache *194*
Scott Maddalo, Shuja Rayaz, Michael T. Smith, Nauman Tariq

38 Pharmacologic Management of Sleep-Pain Interactions *201*
Traci J. Speed

39 The Use and Risks of Opioids in the Management of Orofacial Pain *205*
Alberto Herrero Babiloni, Léa Proulx-Bégin, Gilles J. Lavigne, Marc O. Martel

40 Nonpharmacologic Management of Insomnia and Orofacial Pain *210*
Daniel Whibley, Nicole K.Y. Tang, Michael T. Smith

Index 217

Foreword

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It is quite unbelievable that more than 10 years have elapsed since the first edition of *Sleep Medicine for Dentists* appeared! At that time, it seemed almost daring to publish a book on sleep disorders aimed at the dental profession. Yet, there is little doubt now that dentists are one of the many important portals of entry through which patients can gain earlier detection and therefore benefit from improved management of sleep disorders. Furthermore, the array of uniquely valuable and efficacious tools that dentists bring to the field is further enhanced by the fact that increased knowledge of sleep among any health care professional and by the public at large can only lead to better outcomes.

We cannot forget, or for that matter let anyone else forget, that sleep is a vital function and constitutes the fourth pillar of health and wellness. As such, rather than continue the isolationist route of silo building across professions and disciplines, focusing only on our area of expertise, there has been a slow and steady progressive evolution toward multidisciplinary and interdisciplinary cooperation in sleep medicine. Are we there yet? No, not yet, but we are moving in the right direction, and to continue getting there, we need to make sure that all health care professionals receive adequate and informative training focused around sleep and its disorders.

Before I comment on how this new edition of the book elegantly achieves such lofty goals, I want to remind ourselves that we tend to forget large portions of the wisdom generated by our predecessors. I was recently pointed to a paper published in 1913 by *The Boston Medical and Surgical Journal* (now *The New England Journal of Medicine*). In this short manuscript, Dr Irving Sobotky was already challenging the effectiveness of adenotonsillectomy in children and remarked on the high frequency of patients who continued to be mouth breathers despite "successful" surgeries.[1] He further elaborated on the importance of nasal breathing. More than 100 years after this observation, we are still in pursuit of the elusive ideal of nasal breathing. Hopefully, this time, we can count on not only ENTs and sleep physicians but on the many other disciplines, and top among them, dentists, to help our patients breathe well through their noses.

As a sleep physician who has been deeply involved in sleep medicine over 30 years, I am thrilled to see the uniquely exquisite attention and effort paid in this new edition to facilitate learning and attract learners. The content is carefully divided and balanced between important areas of sleep that are pertinent to the dental profession, and the inordinately attractive and visually pleasing layout of text, tables, and graphics makes it nearly impossible to let go of the book once you get started. I would definitely hope that this textbook will become a mandatory part of the curriculum for all dental schools, and that it will stimulate many of its readers to not only put the knowledge gained to practice but also go and dig deeper and bring their ingenuity to the forefront, thereby advancing the field.

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1. Sobotky I. Persistent mouth breathing following adenoidectomies. Boston Med Surg J 1913;168:230–231.

Preface

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It has been 11 years since the publication of the first edition of this dental sleep medicine book with Quintessence. The key aim of *Sleep Medicine for Dentists* was to provide a rapid source of practical information to students, practicing dentists, and scientists about the evolving field of dental sleep medicine. We sought to put a stake in the ground to herald the emergence of a new interdisciplinary field. The first edition was an instant success, with such strong continued interest that in the last few years the book has only been available for resale by a previous owner. This is a strong indication that the field of dental sleep medicine is growing in both the clinical practice and academic spheres. The book became an academic and board exam reference—a testament to its stature as an authoritative but concise resource. We thank everyone who believed in our collective work.

The role of dentistry in sleep medicine has evolved considerably over the last decade and is now accepted as an important component of the multidisciplinary approach to diagnosis and management of patients with diverse sleep complaints across the lifespan. There is a critical role for dentistry from childhood upper airway and oral development to management of adult sleep apnea, diagnosis of oral conditions linked to sleep-disordered breathing, sleep bruxism, and orofacial pain syndromes. What was previously considered the exclusive domain of the medical profession has now expanded to other disciplines, including dentistry, psychology, and physical/speech therapy. Dentists, dental therapists, and hygienists are among a team of collaborators that are increasingly and, sometimes uniquely, well-positioned in health care systems to maintain quality of life and optimal health for patients suffering with sleep-related breathing disorders, sleep bruxism, orofacial pain, and other orofacial-related syndromes that disrupt sleep and exacerbate pain and fatigue. The role of concomitant conditions (ie, comorbidities) with the above three major sleep problems is also of critical concern.

We believe the timing of this second edition is a perfect way to highlight the incredible advancements that have occurred in the last decade to entrench the role of dentistry in sleep medicine. The 2020 edition has been expanded from 24 to 40 chapters. As before, the book has 4 sections: Introduction to Dental Sleep Medicine, Sleep Breathing Disorders, Sleep Bruxism: From Oral Behavior to Disorder, and Sleep and Orofacial Pain. All previous chapters were updated, and new ones have been added based on the suggestions of many of our readers. The objective of this new edition is to present evidence-based material in a practical manner to guide students in their training and clinicians in their practice.

Editing such a book would have been impossible without the collective, respectful, and professional effort of the three editors, and our colleague Frank Lobbezoo, who provided invaluable input on the sleep bruxism section. We owe our gratitude to all authors and coauthors for their generosity of time, commitment, and integrity. They have come together to share with you the best of their knowledge and their passion for dental sleep medicine. We also want to thank Bryn Grisham and Samantha Smith from Quintessence for their perseverance in working on the second edition of the book.

In Memoriam

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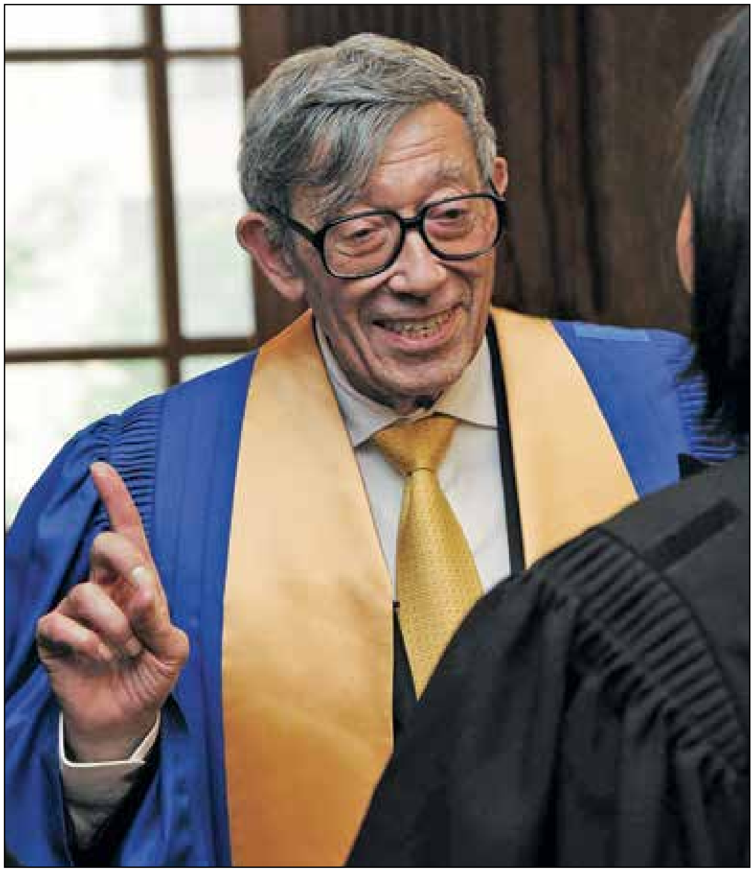

Christian Guilleminault (1938–2019)

This book is dedicated to Doctor Christian Guilleminault, who was a faithful advocate for the role and importance of dental sleep medicine.

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Abbreviations

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This reference list contains the most common abbreviations used throughout the book. Please note that these terms will not be spelled out in the book.

AHI	Apnea-Hypopnea Index
BMI	body mass index
CBCT	cone beam computed tomography
CPAP	continuous positive airway pressure
CSA	central sleep apnea
CT	computed tomography
DSM	dental sleep medicine
EDS	excessive daytime sleepiness
ENT	ear, nose, and throat specialist/surgeon
ESS	Epworth Sleepiness Scale
MAD	mandibular advancement device
MRI	magnetic resonance imaging
NREM	non-REM
OA	oral appliance
OAT	oral appliance therapy
OSA	obstructive sleep apnea
PAP	positive airway pressure
PCRIT	pharyngeal upper airway collapsibility
PLMD	periodic limb movement disorder
PSG	polysomnography/polysomnogram
RBD	REM behavior disorders
REM	rapid eye movement
SB	sleep bruxism
SDB	sleep-disordered breathing
SRBD	sleep-related breathing disorder
TMD	temporomandibular disorder

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CHAPTER 1

The Nature and Structure of Sleep

Cibele Dal Fabbro
Monica L. Andersen
Gilles J. Lavigne

In the animal kingdom, sleep is a universal and imperative biologic process to maintain and restore health. *Sleep* is defined as a physiologic and behavioral state characterized by partial isolation from the environment. A baby's cry, the vibration of an earthquake, or a sudden pain intrusion will all interrupt sleep continuity; a sleeping brain maintains a sentinel function to awaken the organism for protection purposes.

The duration of sleep usually is 6 to 9 hours in adults. Although most adults sleep an average of 7.5 hours, some are short sleepers and some are long sleepers (ie, less than 5.5 hours and more than 9.0 hours, respectively). Good sleep quality is usually associated with a sense of having slept continuously through the night and feeling refreshed and alert on awakening in the morning. The perception of sleep quality is subjective and varies widely among individuals. Some individuals perceive their sleep as satisfying most of the time, and some consistently report being poor sleepers (eg, having difficulties in initiating or maintaining sleep—insomnia, feeling unrefreshed when they awaken, and having nightmares). However, sleep recording systems indicate that, in general, poor sleepers tend to underestimate the length of time they sleep (as do some good sleepers). The neurobiology of sleep is described in chapter 2, and a classification of the various sleep disorders relevant to dentistry is found in chapter 3.

Sleep-Wake Cycle

An adult's 24-hour cycle is divided into approximately 16 hours of wakefulness and 8 hours of sleep. Synchronization and equilibrium between the sleep-wake cycle and feeding behaviors are essential for survival. Mismatches in the synchronization of the feeding cue and metabolic activity are associated with eating disorders.[1] Poor sleep can cause health problems and can increase the risk of transportation- and work-related accidents and even death.[2]

Homeostatic process

The propensity to sleep is directly dependent on the duration of the prior wakefulness episode. As the duration of wakefulness increases, sleep pressure accumulates and builds to a critical point, when sleep onset is reached. As this sleep pressure increases, an alerting circadian signal helps the person to remain awake throughout the day. The ongoing 24-hour circadian rhythm therefore runs parallel to the homeostasis process, also known as *process S* (Fig 1-1). The process S corresponds to the sleep pressure that individuals accumulate during the wakefulness period before being able to fall asleep. With increasing sleep pressure, sleep is proportionally longer and deeper in the following recovery period.

Changes in the frequency of slow-wave sleep waves can be estimated by a mathematic transformation of brain wave electrical signals or by quantitative spectral analysis of the electroencephalographic (EEG) activity. Rising or rebound of slow-wave EEG activity in the first hours of sleep is a marker of sleep debt.[3] In contrast, a reduction in slow-wave activity is observed in patients with chronic pain.[4,5] However, the cause-and-effect association of these biologic signals with reports of fatigue and poor sleep is unknown. During the day, the effects of energy expenditure are accumulated, which may be connected to the feeling of tiredness.

Two times in the 24-hour cycle are characterized by a strong sleep pressure, 4 PM and 4 AM, +/- 1 to 2 hours (see Fig 1-1). At a certain point, sleep pressure is so powerful that an individual will fall asleep regardless of the method or strategies used to remain awake.

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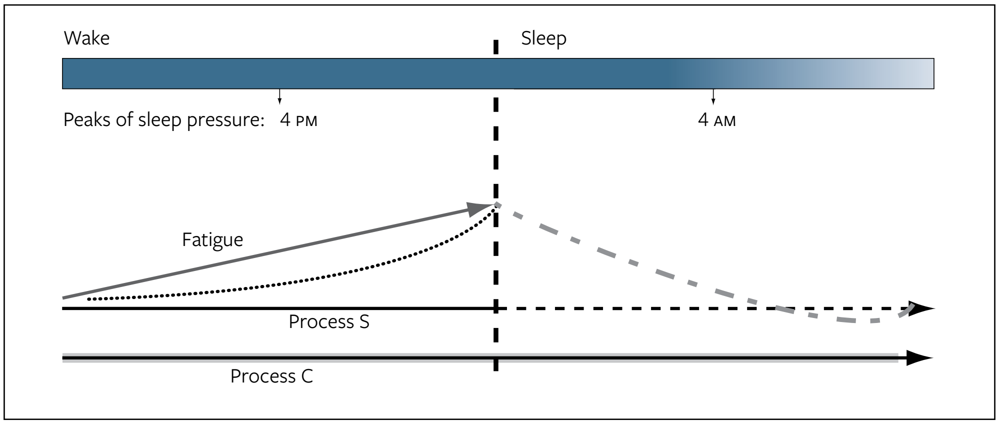

FIG 1-1 Normal cycle for circadian rhythm (process C) (*solid black arrow*) and process S (*solid black line/dashed arrow*) over about 24 hours. During wakefulness periods, the increase in sleep pressure (*dotted line*), parallels the increase in fatigue (*gray arrow*) and results in sleep (*dashed and dotted gray line*) at a given time over a 24-hour circadian cycle.

Circadian rhythm

Humans tend to alternate between a period of wakefulness lasting approximately 16 hours and a continuous block of 8 hours of sleep (see Fig 1-1). Most mammals sleep around a 24-hour cycle that is driven by clock genes that control the circadian rhythm (process C). Light helps humans synchronize their rhythm with the cycles of the sun and moon by sending a retinal signal (melanopsin) to the hypothalamic suprachiasmatic nucleus. The suprachiasmatic nucleus is a network of brain cells and genes that acts as a pacemaker to control the circadian timing function.[6]

The investigation of sleep-wake process C uses biologic markers to assess a given individual's rhythm. A slight drop (hundredths of a degree centigrade) in body temperature and a rise in salivary and blood melatonin and growth hormone release—peaking in the first hours of sleep, around midnight in the 24-hour cycle—are key indications of the acrophase (high peak) of the process C. Interestingly, corticotropins (adrenocorticotropic hormone and cortisol) reach a nadir (lowest level) during the first hour of sleep. They then reach an acrophase in the second half of the night.[1,7] The process C can also be studied using temperature recordings in relation to hormone release and polygraphy to measure brain, muscle, and heart activities.

Ultradian rhythm

Under the 24-hour process C of sleep and wakefulness, sleep onset and maintenance are governed by an ultradian cycle of three to five periods in which the brain, muscles, and autonomic cardiac and respiratory activities fluctuate (Figs 1-2 and 1-3).[8] These cycles consist of REM sleep (active stage) and NREM sleep (light and deep stages). The REM stage is known as *paradoxical sleep* in some countries.

In humans, a clear decline in electrical brain and muscle activities as well as heart rhythm is observed from wakefulness to sleep onset. This decline is associated with a synchronization of brain waves toward stage N1 of sleep. Stage N1 is a transitional period between wakefulness and sleep. Stage N2, which accounts for about 50% to 60% of total sleep duration, is characterized by two EEG signals—K-complexes (brief, high-amplitude brain waves) and spindles (rapid, spring-like EEG waves)—both described as sleep-promoting and sleep-preserving factors. Sleep N1 and N2 are categorized as *light sleep*.

Next, sleep enters a quiet period known as *deep sleep*, or stage N3, which is characterized by slow, high-amplitude brain wave activities, with dominance of delta sleep (0.5 to 4.5 Hz). This sleep period is associated with a so-called sleep recovery process.

Finally, sleep enters an ascension period and rapidly turns into either light sleep or REM sleep. REM sleep is associated with a reduction in the tone of postural muscles (which is poorly described as "atonia" in literature but is in fact *hypotonia* because muscle tone is never zero; see chapter 2, reference 13) and a rise in heart rate and brain activity to levels that frequently surpass the rates observed during wakefulness.

Humans can dream in all stages of sleep, but dreams during REM sleep may involve intensely vivid imagery with fantastic and creative content. During REM sleep, the body is typically in a paralyzed-like state (muscle hypotonia). Otherwise, dreams with intense emotional content and motor activity might cause body movements that could injure individuals and their sleep partners.

An understanding of the presence of ultradian sleep cycles is relevant because certain pathologic events occur during sleep, including the following sleep disorders:

- Periodic body movements (leg or arm) and jaw movements, such as SB, most of which are observed in stage N2 of sleep and with less frequency in REM sleep

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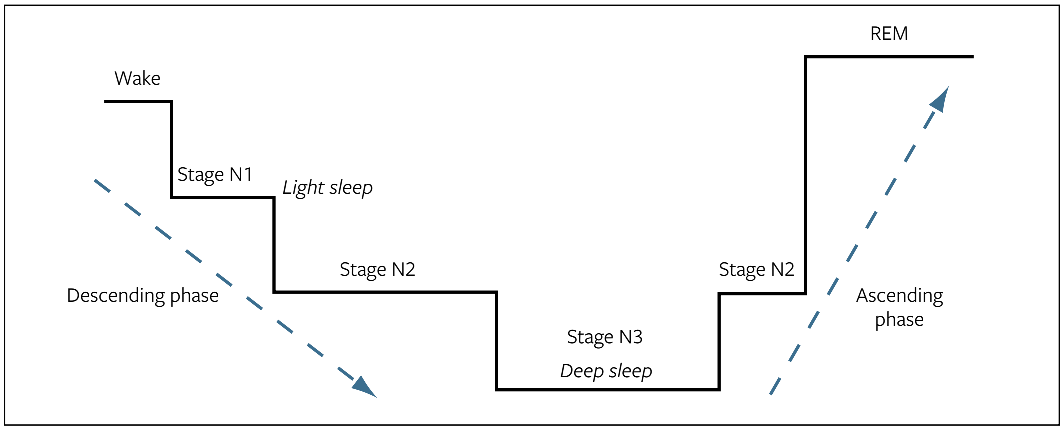

FIG 1-2 One NREM-to-REM cycle of consecutive sleep stages. This cycle is repeated every 70 to 110 minutes for a total of three to five NREM-to-REM cycles per sleep period.

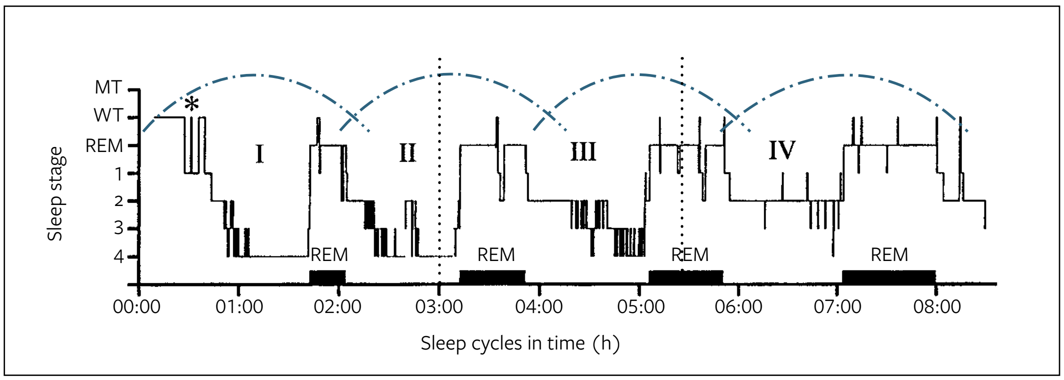

FIG 1-3 Consecutive waves of NREM-to-REM (*solid horizontal boxes*) sleep cycles (I to IV). During the first third of the night, slow-wave sleep (stage N3) is dominant. During the last third of the night, the REM stage is longer. MT, movement time; WT, wake time. (Adapted from Lavigne et al[8] with permission.)

- Sleep-related breathing events, such as apnea and hypopnea (cessation or reduction of breathing), observed in N2 and REM sleep
- Acted dreams with risk of body injury, diagnosed as RBD, which occur during REM sleep (see chapter 3)

Sleep Recordings and Sleep Arousal

When a PSG of a sleeping patient (collected either at home with an ambulatory system or in a sleep laboratory) is assessed, the scoring of sleep fragmentation is a key element in analyzing sleep quality. Poor sleep quality, as reported subjectively by the patient, is associated on PSGs with more bed time with wake after sleep onset (WASO), frequent arousals with or without body movements or with a high score of periodic limb movement (PLM), frequent stage shifts (from a deeper to a lighter sleep stage), respiratory disturbances (measured per hour by the respiratory disturbance index [RDI]), and higher muscle tone. All these signs of sleep fragmentation interrupt the continuity of sleep and alter the sleep architecture.

Sleep efficiency is another important variable to evaluate. A standard index of sleep impairment, *sleep efficiency* is defined as the amount of time asleep divided by the amount of time spent in bed, expressed as a percentage. Sleep efficiency greater than 90% is an indicator of good sleep.

The ultradian cycle of sleep, described previously, includes another repetitive activity: sleep-related arousals. During NREM sleep, arousals are recurrent (6 to 14 times per hour of sleep), involving brief (3 to 10 seconds) awakenings associated with increased brain, muscle, and heart activities (tachycardia or rapid heart rate) in the absence of the return of consciousness.[9–11] In the presence of sleep movements, breathing disorders, or chronic pain, these arousals are more frequent. Sleep arousals can be viewed as the body's attempt to prepare the sleeping individual (who is in a low-vigilance state) to react to a potential risk, ie, a fight-or-flight state.

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BOX 1-1 Functions of sleep

Fatigue reversal
- Sleep allows the individual to recover and reenergize.

Biochemical refreshment
- Sleep promotes synaptic efficiency, glymphatic lavage, protein synthesis, neurogenesis, metabolic (eg, glycogen) restoration, growth (secretion of growth hormone peaks during sleep), etc.

Immune function
- Reset or protection (complex interaction; causality under investigation).

Memory consolidation
- Daytime learning needs sleep for memory consolidation.
- Sleep seems to facilitate encoding of new information.
- May also facilitate learning of simple tasks, modify behavior.

Psychologic well-being
- Dreams occur in all sleep stages. REM dreams are more vivid.
- Lack of sleep presents a risk of mood alteration to depression.

Sleep arousals are concomitant with or precede most PLMs and SB (described also in chapter 26 on pathophysiology of SB, section III). In contrast, sleep apnea and hypopnea (described in section II) are respiratory distress–like events that trigger sleep arousals. An index of arousal per hour of sleep is estimated as well as arousal-related ones: frequency of shifts in sleep stage, PLMs, bruxism, snoring, and sleep-related apnea and hypopnea.

In addition to these methods, sleep fragmentation can be estimated by the presence of the cyclic alternating pattern (CAP) to evaluate the instability of sleep. CAP is an infraslow oscillation, with a periodicity of 20 to 40 seconds, between the sleep maintenance system and the arousal pressure involved in the dynamic organization of NREM sleep and the activation of motor events.[12]

CAP is the estimate of the dominance of active phasic arousal periods—that is, the rise in heart rate, muscle tone, and EEG activities (phase A)—over more stable and quiet sleep periods (phase B).[11,13] The active phase is subclassified as A1, a period that promotes sleep onset and maintenance; A2, a transition phase; and A3, the final phase, or the arousal window, involving a marked increase in muscle tone and cardiorespiratory rate. Note that most SB events are scored in phases A2 and A3 (see chapter 26).

People appear to have individual levels of tolerance for sleep fragmentation. These levels may be genetically determined. Nevertheless, recurrent sleep deprivation or fragmentation produces a cumulative sleep debt, which in turn is likely to increase complaints of fatigue, memory and mood dysfunction, and bodily pain. The cause-and-effect relationship remains to be supported by evidence.

Developmental Changes in Sleep-Wake Patterns

The human sleep-wake pattern changes with biologic maturation and aging. In the first 6 weeks of life, sleep of infants is dominated by REM sleep, which occupies about 50% of their sleep time. Around age 6 to 9 months, their wakefulness and nighttime sleep pattern tends to become more synchronized with their parents' feeding and sleeping schedule.[14] Preschool children sleep about 14 hours per 24-hour cycle, and most stop napping somewhere between the ages of 3 and 5 years. An important aspect related to development is the growth of the airway and involution of adenoids that seems to influence occurrence and resolution of snoring and apnea in children between 5 to 12 years of age (see chapter 14).

Pre-adolescent children are sleep-wake phase advanced. They fall asleep earlier and awake earlier than middle-aged adults. Teenagers tend to be phase delayed (get to bed later and wake later in morning) and tend to sleep about 9 hours per 24 hours (ranging from 6.5 to 9.5 hours), falling asleep and awakening later than their parents and younger siblings.

Most adults sleep about 6 to 7 hours on workdays and more on the weekends. By about the age of 40 to 45 years, adults' sleep starts to become more fragile, and individuals are more aware of being awake for a few seconds to a few minutes a night. In the elderly, the sleep-wake pattern returns to a multiphase pattern typical of young children. Elderly people go to sleep earlier than middle-aged adults and awake earlier in the morning, taking occasional naps (catnapping) during the day. Some may present advanced phase shift, ie, get to sleep earlier and wake earlier in morning.

The human biologic clock can adapt to sleep deprivation and changes in the sleep-wake schedule within certain limits. For example, some people can adapt better than others to jetlag or sleep deprivation because of night work (eg, flight crew, hospital staff), but most individuals find such variations difficult.

Sleep and Health

The diagnosis, prevention, and management of sleep disorders are currently domains of high impact in public health (eg, prevention of breathing disorders from childhood, management of daytime sleepiness to decrease the risk of transportation accidents, and the relationship of hypertension and sleep apnea).

Sleep and circadian rhythm entail several functions, including physical recovery, biochemical refreshment (eg, synaptic neuronal function; glial cell role in glymphatic process), memory consolidation, emotional regulation, and to a small extent, possible learning of simple tasks/behaviors[15–22] (Box 1-1). A persistent reduction in sleep duration can cause physical and mental health problems because of the cumulative effect of lack of sleep on several physiologic functions (see chapters 9 and 33 to 35).

Lack of sleep is also known as *sleep deprivation*, that is, insufficient sleep resulting from short sleep duration or loss of a sleep segment because of environmental factors (eg, noise) or

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a contributing medical condition (eg, pain, diabetes, mood/depression).

An experiment on sleep deprivation (4 hours of sleep over 3 to 4 days), done in young individuals who usually sleep for 8 hours, showed that sleep deprivation triggers mood alteration, sociability dysfunction, and complaints of bodily pain.[23] This was recently reassessed over a 3-week protocol, and sleep disruption had more deleterious effects on pain perception and slow recovery in the most vulnerable subjects (see chapters 34 and 35).[24] Another protocol using force awakening reported that women have altered temporal pain summation and men have more secondary hyperalgesia after a night of sleep disturbance.[25] Many recent research data support the idea that sleep deprivation, anxiety, and low-grade inflammation are deleterious to learning and memory.[26] Pain patients with sleep problems frequently report inflammation, poor sleep, and anxiety.[26]

Obviously, direct and indirect causalities of so many variables need more powerful analytic approaches; the emergence of "machine learning" in sleep research will help us to better delineate specific phenotypes and to select the most efficient treatment modality.[27]

Moreover, both too-short and too-long sleep durations have been associated with higher risks of diseases and mortality. However, the complicated interactions among lifestyle, mortality risk, and sleep duration remain to be understood.[28] In fact, there is some evidence to support the relationship between sleep duration (too little or too much) and the risk of cardiovascular diseases (such as myocardial infarction and atherosclerosis), diabetes, obesity, depression, and even cancer.[23, 28–31]

Although these risk estimates are modest, they have been reproduced in too many studies to reject the putative effect of cumulative sleep debt on health maintenance. Higher risks of myocardial infarction have been found in women who are short sleepers as well as women who are long sleepers.[31] Elevated risks of cardiovascular problems and atherosclerosis also have been observed in people who sleep too much during the day[29] (see also chapter 9).

Cost of Inadequate Sleep

The direct and indirect costs of sleep disorders in Australia was estimated at US $7.5 billion for 2004, and the cost of inadequate sleep was estimated close to US $32 billion in 2016–2017.[2] Furthermore, a study from Denmark, covering the period of 1998 to 2006, revealed that annual direct and indirect costs for patients with snoring, sleep apnea, and obesity hypoventilation syndrome were €705 (about US $800), €3,860 (about US $4,400), and €11,320 (about US $13,000), respectively.[32] Furthermore, these individuals had lower employability and lower income—a condition present up to 8 years before the diagnosis of the conditions.

The American Academy of Sleep Medicine, in a report commissioned to the global research and consulting firm Frost & Sullivan, estimated the economic cost of untreated sleep apnea at US $150 billion, including loss in productivity as well as transportation and work accidents.[33]

Conclusion and Advice

Good-quality sleep is essential to physical recovery, biochemical refreshment, memory consolidation, and emotional regulation. The diagnosis, prevention, and management of disorders that interfere with the quality of sleep are domains of high impact in public health.

Dentists are in an excellent position to convey messages on the importance of good sleep habits and in collaboration with other health professionals to manage some sleep disorders such as SB, sleep apnea, and pain related to sleep (see chapters 4 and 5).

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Index

Page references followed by "f" denote figures, "t" denote tables, and "b" denote boxes.

A

A-beta fibers, 175
Acetylcholine, 10, 138
Acoustic reflection pharyngometry, for obstructive sleep apnea, 68t, 70
Acrophase, 4
Active phasic arousal periods, 6
Active sleep. *See* REM sleep.
Active theory, 11
Acute pain
  chronic pain transition of, 176
  description of, 175
  mechanisms of, 175–176, 176f
Adalimumab, 184
Adaptive servoventilation, 208
Addiction Behavior Checklist, 208
A-delta fibers, 175
Adenoids, 44
Adenosine, 11
Adenosine triphosphate, 11
Adenotonsillar hypertrophy
  in obstructive sleep apnea, 79
  sleep-disordered breathing associated with, 48
Adenotonsillectomy, 79, 80t, 165
Adolescents, sleep bruxism in, 162–165, 163t
Adrenocorticotropin hormone, 179
Adults
  dentofacial morphology in, 48–49
  mouth breathing in, 48–49
  sleep-wake patterns in, 6
Airway
  lower, 48
  upper. *See* Upper airway.
Alcohol consumption, 16
Allodynia, 176, 189
Alpha-delta sleep, 20
American Academy of Sleep Medicine
  International Classification of Sleep Disorders 3, 15, 16b, 24
  obstructive sleep apnea recommendations, 72
  sleep apnea costs, 7
  sleep bruxism diagnostic criteria, 125, 125b
  sleep duration recommendations, 183
Analgesics, 202, 202t
Anesthesia
  in obstructive sleep apnea patients, 100–103
  upper airway obstruction during, 101
Angiopoietin-2 gene, 114
Ankylosing spondylitis, 184
Antidepressants, 185, 202t, 202–203
Antiepileptics, 203
Antipsychotics, 203
Antispasmodics, 203
Anxiety, 143
Apnea
  central sleep. *See* Central sleep apnea.
  definition of, 15
  obstructive sleep. *See* Obstructive sleep apnea.
Apnea-hypopnea index
  definition of, 36
  grading of, 15–16, 64
  obstructive sleep apnea assessments, 64
  oral myofunctional therapy effects on, 104
β-arrestin 1 gene, 114
Arthralgia, 170, 171t
Athens Insomnia Scale, 211
Atrial fibrillation, 61
Attention, obstructive sleep apnea effects on, 50
Attention-deficit/hyperactivity disorder, 163
Atypical facial pain, 173
Atypical odontalgia, 173
Aura, migraine with/without, 171, 194, 195b

B

Baclofen, 203
Bariatric surgeons, 23
Bariatric surgery, 74
Barrett esophagus, 18
Benzodiazepines, 16–17, 204
Berlin questionnaire, 16, 62, 195, 211
Beta-adrenergic blocking agents, 185
Bi-level positive airway pressure, 73, 79, 80t
Biofeedback, 158, 158t
Blood-brain barrier, 13
Bone-anchored maxillary protraction, 83, 84f
Bone-borne implant expansion, 83
Botulinum toxin, for sleep bruxism, 158t, 159–160
Brainstem, 9–10, 10f
Breathing. *See* Mouth breathing; Sleep-disordered breathing.
Bremer, Frédéric, 9
Bruxism, 142. *See also* Sleep bruxism.
Burning mouth syndrome, 172t, 173, 175, 191
Buspirone, 138

C

Caffeine, 195
Calcitonin gene-related peptide inhibitors, 196, 198
Cancer, obstructive sleep apnea and, 54
Candidate-gene association studies, 113t, 114–115
Cannabinoids, 181, 185, 204
CAP. *See* Cyclic alternating pattern.
Car accidents. *See* Motor vehicle accidents.
Carbamazepine, 203
Carbon dioxide, 42
Cardiovascular diseases, obstructive sleep apnea and, 52f, 53
Catastrophizing, 207
Catathrenia, 18
Central obesity, 61
Central pattern generators, 136, 137f
Central sensitivity syndrome, 189
Central sensitization, 176–177, 189
Central sleep apnea. *See also* Sleep apnea.
  characteristics of, 35, 37
  description of, 15
  diagnosis of, 37
  opioids and, 206
  polysomnographic findings in, 36f
  risk factors for, 37
Central sleep apnea syndrome, 17
Cephalometry, for obstructive sleep apnea, 66, 67t, 70
C-fibers, 175
Cheyne-Stokes respiration, 37
Children
  dentofacial morphology in, 48
  mouth breathing in, 48, 82
  obesity in, 79
  sleep apnea in, 16
  sleep bruxism in, 162–165, 163t
  sleep-wake patterns in, 6
Choking, 20
Cholinergic-activating system, 9–10, 10f
Chronic insomnia, 210

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Chronic migraine, 194
Chronic pain
  acute pain transition to, 176
  cannabinoids for, 185
  description of, 23
  etiology of, 175
  hypothalamus-pituitary-adrenal axis involvement in, 179–180
  imaging studies of, 177
  immune dysregulation in, 184
  peripheral sensitization in, 176
  sleep deficiency and, interactions between
    analgesics for, 202, 202t
    antidepressants for, 202t, 202–203
    antiepileptics, 203
    antipsychotics for, 203
    antispasmodics for, 203
    behavioral approaches for, 183–184
    causality of, 187–192
    mechanisms of, 178–181
    pharmacologic management of, 201–204
  sleep-disturbing medications for, 185
Circadian rhythms
  control of, 115
  description of, 3–4
  functions of, 6
  regulation of, 11
  ultradian rhythm, 4–5
Circadian rhythm sleep disorders, 16b
Classification, 169
Clonazepam, 138, 158t, 159–160
Clonidine, 139, 158t, 159–160
Cluster headache, 171, 172t, 192, 195t, 197–198
Cognitive behavioral therapy
  for exploding head syndrome, 199
  for insomnia, 183–184, 196–197, 212–214, 213t
  for orofacial pain, 214
  for sleep, 214
Cognitive therapy, 213t
Comorbidities, 24
Computed tomography
  cone beam, 67t, 71
  obstructive sleep apnea evaluations, 66–68, 67t
Cone beam computed tomography, 67t, 71
Confusional arousal, 17
Congenital central hypoventilation syndrome, 115
Continuous positive airway pressure
  data monitoring capabilities of, 110
  mandibular advancement devices and, 88, 91
  obstructive sleep apnea treated with, 72–73, 80t, 96, 110
Cortical arousals, 42
Cortical neurons, 13
Corticosteroids, 185
Corticotropin-releasing hormone, 179
Cortisol, 138–139, 179, 185
Cranial nerve stimulation, 94
Craniofacial growth and development, 44–45, 82
Craniofacial phenotyping, 110f, 111
C-reactive protein, 183
Current Opioid Misuse Measure, 207
Cyclic alternating pattern, 6, 139
Cyclobenzaprine, 203
Cyclooxygenase, 180
Cytokines, 179

D

Deep sleep, 4
Delta waves, 13
Dental casts, 31
Dental history, 29
Dental hygienists, 25
Dental schools, 25
Dental sleep curricula, 24–25
Dental sleep education, 24–25
Dental sleep medicine
  definition of, 22, 119
  dentist's role in, 22–25
  interdisciplinary and intersectoral team effort in, 22–23
  neurologists in, 23
Dentists
  credentials/certification of, 33
  dental sleep education for, 24–25
  dental sleep medicine role of, 22–25
  expertise of, 23–24
  in opioid crisis, 207–208
  sleep-disordered breathing screening by, 29
Dentoalveolar pain, 170, 172t
Dentofacial morphology, 48–49
Depression, obstructive sleep apnea and, 50
Development
  sleep's role in, 14
  sleep-wake patterns affected by, 6
Diabetes mellitus, 52, 52f
Diaphragm, 48
Difficult airway, 101, 103
Dopamine, 150, 160
Down syndrome, 162
Dreams/dreaming
  recollection of, 13
  in REM sleep, 4, 9, 13
Dronabinol, 75
Drug-induced sleep endoscopy, 68, 70, 96
Dual-block mandibular advancement devices, 90
Duloxetine, 203

E

Ecologic momentary assessment and intervention, 158
Edentulism, 56
Elderly, sleep-wake patterns in, 6
Electroencephalogram
  non-REM sleep, 13
  REM sleep, 13
  sleep activity, 3
  sleep bruxism findings, 163
  sleep stages, 13
  wakefulness, 12
Electromyographic measurement systems, 126
Electro-oculogram, 63
Endocannabinoid system, 180–181
Epigenetics, 115t, 150
Epigenome-wide association studies, 115t, 115–116
Epilepsy, sleep bruxism and, 132
Epworth Sleepiness Scale, 50, 51f, 62–63
E-selectin, 56
Excessive daytime sleepiness
  Epworth Sleepiness Scale for, 50, 51f, 62–63
  obstructive sleep apnea as cause of, 50–51, 74
Exercise, for obstructive sleep apnea, 72
Exome sequencing, 115, 115t
Exploding head syndrome, 20, 199
Extraoral examination, 31, 32f

F

Facial growth and development, 44–45
Faciomandibular myoclonus, 17, 19
Fatal familial insomnia, 14
Fatigue
  obstructive sleep apnea as cause of, 50, 62b
  poor sleep and, 3
  sleepiness versus, 63
Fatigue Severity Scale, 62b, 63
Feeding behaviors, sleep-wake cycle and, 3
Fibromyalgia, 20, 24
Fight-or-flight state, 5
Flip-flop switch model, 11, 11f
Free radicals, 14
Functional pain, 189–190

G

Gabapentin, 184–185, 203
Gabapentinoids, 184–185
Gastroenterologists, 23
Gastroesophageal reflux disease, 17–18, 132–133
Genioglossus muscle, 39f, 46–47, 84, 96
Geniotubercle advancement, 94
Genome-wide association studies, 113t, 115
Genome-wide linkage studies, 113t, 114
Giant cell arteritis, 173
Gingival inflammation, 56
Glial cells, 13
Glossary of Prosthodontic Terms, 120, 120t
Groaning, 18

Copyright
Not for publication
Quintessence

Grunting, 18
Gurgling sounds, 18

H

Headaches
cluster, 171, 172t, 192, 195t, 197–198
exploding head syndrome, 20, 199
herpes zoster and, 199
hypnic, 20, 192, 195b, 198
medication overuse, 196
migraine. *See* Migraine headaches.
morning, 198
orofacial pain and, 192
sleep bruxism and, 133
sudden, novel, and intense, 198–199
tension-type, 20, 192, 195b, 197
in traumatic brain injury, 198
types of, 20, 194
Head-forward posture, 48
Health
oral, 58
sleep and, 6–7
Health-related quality of life, 50–51
Heartburn. *See* Gastroesophageal reflux disease.
Hemicrania, 171, 172t
Heritability studies, 113t
Herpes zoster, 170, 199
High-flow nasal cannulae, 79, 80t
High loop gain, 41f, 42
High-sensitivity C-reactive protein, 57
High throughput facial phenotyping, 110
Homeostatic process, 3
Home sleep apnea test, 72, 79
Horton headache, 171
HTR2A gene, 148–149
Hyperalgesia, 189
Hyperalgesic priming, 189
Hypercapnia, 35
Hypersalivation, 18
Hypersomnia, 16b
Hypertension, 52, 52f
Hypnic headache, 20, 192, 195b, 198
Hypocretin, 201
Hypoglossal nerve stimulation, for obstructive sleep apnea, 74
Hypopnea, 15, 130f. *See also* Sleep apnea-hypopnea.
Hypothalamus, 199
Hypothalamus-pituitary-adrenal axis, 178–180
Hypotonia, 4
Hypoventilation
hypercapnia associated with, 35
sleep-related, 37–38, 38f
Hypoxia, 52, 54, 56
Hypoxia inducible factor 1 alpha, 114

I

Idiopathic orofacial pain, 173
Imagery training, 213t
Imaging. *See also* specific modality.
incidental findings on, 71
obstructive sleep apnea evaluations, 66–71, 67t–68t, 69f
types of, 31
upper airway, 66–70, 67t–68t
Implants, 153f, 154
Inadequate sleep, 7
Infants, sleep-wake patterns in, 6
Inflammatory bowel diseases, 184
Inflammatory pain, 189
Infliximab, 184
Informed consent, 33
Insomnia
characteristics of, 20
chronic, 210
cognitive behavioral therapy for, 183–184, 196–197, 212–214, 213t
definition of, 16b, 131
management of, 159, 184
medical history findings, 23b
migraine headaches as cause of, 194
nonpharmacologic management of, 210–214
pharmacologic approaches for, 184
screening for, 211
sleep bruxism and, 131
sleep hygiene education for, 211, 212b, 213t
Insomnia Severity Index, 195, 211
Interleukin-1, 179
Interleukin-1ß, 56
Interleukin-6, 57, 179–180, 183
Interleukin-33, 57
Intermittent hypoxia, 52
International Association for the Study of Pain, 175
International Bruxism Consensus Group, 120
International Classification of Headache Disorder, 198
International Classification of Sleep Disorders, 119
International Classification of Sleep Disorders 3, 15, 16b, 24, 195b
International Sleep Genetic Epidemiology Consortium, 114
Intraoral examination, 31, 32f

J

Jaw bracing, 120
Jaw muscle tone, 138
Jaw thrust, 100
Jaw thrusting, 120
Jet lag, 6

K

K-complexes, 13

L

Laryngospasm, sleep-related, 18
Laterodorsal tegmental nuclei, 10f
Light sleep, 4
Limited-channel sleep studies, for obstructive sleep apnea, 64
Lower airway, mouth breathing effects on, 48
Lower airway resistance, 82
Lung volume, 48

M

Machine learning, 108, 109t, 111, 131
Magnetic resonance imaging, for obstructive sleep apnea, 67t, 68, 69f, 70
Mallampati classification, 61
Mandibular advancement devices. *See also* Oral appliance/oral appliance therapy.
adjustment of, 90
clinical outcomes of, 87–88
clinical protocol for, 88–90
combination therapy with, 90–91
continuous positive airway pressure and, 88, 91
dental assessment for, 89–90
dual-block, 90
factors associated with, 89, 90b
follow-up for, 90
hypertension reductions using, 52
limitations of, 88
mechanism of action, 87
monoblock, 90
morning headache managed with, 198
multidisciplinary approach to, 88
obstructive sleep apnea treated with, 58f, 74, 79–81, 80t, 87–91, 88b–89b, 97
periodontitis as contraindication for, 58
predictors of success for, 89, 90b
selection of, 90
side effects of, 88
uvulopalatopharyngoplasty and, 90
Mandibular growth and development, 44
Masseter muscle hypofunction, 47
Masticatory function, mouth breathing effects on, 47
Maxillofacial growth, 46
McGill oximetry scoring system, 79
Medical conditions, 16b
Medical history
elements of, 23b
screening uses of, 29
Medication overuse headache, 196
Melanopsin, 4
Melatonin, 180, 185, 196, 203
Meningitis, 198–199

Copyright Not for publication Quintessence

Metabolic disorders, 52–53
Metabolic syndrome, 52, 52f
Metabolomics, 115t
Microarousals, 138–140, 146
Migraine headaches
  characteristics of, 172t
  chronic, 194
  classification of, 194, 195b
  description of, 20, 171, 192
  insomnia associated with, 194–196
  management of, 196–197
  melatonin for, 196
  pathophysiology of, 196
  prevalence of, 171
  sleep assessments in patients with, 195–196
  sleep disturbances caused by, 194–196
  sleep quality affected by, 192
  treatment of, 196–197
  with aura, 171, 194, 195b
  without aura, 194, 195b
Miniature self-contained EMG detector and analyzer, 126
Mirtazapine, 203
Mixed episode, of rhythmic masticatory muscle activity, 135
Modafinil, 74
Monoamines, 10
Monoblock mandibular advancement devices, 90
Montelukast, 80t
Morning headache, 133, 198
Motor vehicle accidents, 51
Mouth breathing
  case presentation of, 84–85, 85f
  causes of, 82
  in children, 82
  lower airway effects of, 48
  masticatory function affected by, 47
  maxillofacial growth affected by, 46
  muscle rehabilitation for, 84
  to nasal breathing, 84
  nasal reflexes affected by, 47–48
  nasomaxillary lengthening for, 83, 84f
  nasomaxillary widening for, 82–83, 83f
  oral cavity effects of, 45, 46f
  perpetuation of dysfunction caused by, 47f, 48
  screening of, 82
  upper airway cyclic dysfunction of, 46, 47f
Movement disorders
  periodic limb, 19, 130–131
  sleep-related, 19b
Mucositis, 189
Müller maneuver, 68, 70
Muscle relaxants, 203
Myalgia, 171t
Myofascial pain, 171t

N

Nasal breathing, mouth breathing to, 84
Nasal obstruction, 94, 164
Nasal reflexes, 47–48
Nasal ventilation reflex, 47
Nasomaxillary lengthening, 83, 84f
Nasomaxillary widening, 82–83, 83f
Nasopharyngoscopy, for obstructive sleep apnea, 68, 68t, 70
Neuritis, 170
Neurologists, 23
Neuropathic orofacial pain, 170, 189
Neurovascular orofacial pain, 171, 172t
Nightmares, 18–19
N-methyl-D-aspartate receptors, 177, 189
Nociception, 175–176
Nociceptive pain, 188–189
Nociceptors, 175, 188–189
Nociplastic pain, 190
Nocturnal frontal lobe epilepsy, 132
Nonalcoholic fatty liver disease, 52
Nonbenzodiazepine receptor agonists, 204
Non-REM sleep
  blood flow in, 14
  definition of, 9
  description of, 4
  electroencephalogram findings, 12
  opioid effects on, 206
  sleep terrors in, 18
  stages of, 12f
Nonsteroidal anti-inflammatory drugs, 180, 184, 202
Norepinephrine, 138, 201
NoSAS score, 16, 17b
Nucleus tractus solitarius, 136

O

Obesity
  central, 61
  obstructive sleep apnea and, 36, 40, 61, 72, 79, 80t, 115
  sleep-disordered breathing associated with, 48
  upper airway affected by, 101
  weight loss for, 72, 80t
Obesity-hypoventilation syndrome, 16, 37, 73, 94
Obstructive sleep apnea. *See also* Sleep apnea.
  acoustic reflection pharyngometry of, 68t, 70
  airway features associated with, 61
  anatomical causes of, 16, 40
  anesthesia risks in patients with, 100–103
  apnea-hypopnea index for, 36–37, 64
  atrial fibrillation and, 61
  attention affected by, 50
  biomarkers of, 113
  cancer and, 54
  cardiovascular diseases associated with, 52f, 53
  causes of, 16, 40–42, 41f
  cephalometric analysis of, 66, 67t, 70
  characteristics of, 35
  in children
    diagnosis of, 77b, 77–79
    predisposing conditions, 77
    treatment of, 79–81, 80t
  clinical prediction models for, 78t
  comorbidities, 24, 30f, 61
  computed tomography of, 66–68, 67t
  consultation about, 33
  craniofacial features associated with, 61, 115
  definition of, 36
  dentist's role in management of, 30f
  depression associated with, 50
  description of, 15
  diabetes mellitus associated with, 52, 52f
  diagnosis of
    in adults, 36–37, 60–64, 72
    ambulatory overnight oximetry for, 211
    audio and video recording used in, 78
    in children, 77–79
    polysomnography for, 36f, 63f, 63–64, 78–79, 211
    preoperative, 101–102
    questionnaires for, 62t, 62–63, 78, 101, 211
  excessive daytime sleepiness caused by, 50–51, 74
  exercise for, 72
  fatigue associated with, 50, 62b
  gastroesophageal reflux disease and, 133
  genetics of, 113t, 113–116, 115t
  heterogeneity of, 107
  home sleep apnea test for, 72, 79
  hypertension caused by, 52, 52f
  imaging modalities for, 66–71, 67t–68t, 69f
  limited-channel sleep studies for, 64
  long-term consequences of, 50–54, 50f–53f
  magnetic resonance imaging of, 67t, 68, 69f, 70
  metabolic disorders caused by, 52–53
  metabolic syndrome associated with, 52, 52f
  morning headache associated with, 198
  motor vehicle accident risks, 51
  nasopharyngoscopy of, 68, 68t, 70
  nonanatomical causes of, 40–42, 41f
  obesity as risk factor for, 36, 40, 61, 72, 79, 80t, 115
  pathophysiology of, 39f, 39–42, 41f, 100
  patient education regarding, 33
  periodontal diseases and, 55–59, 56b, 57f–58f
  periodontitis and, 54, 58f
  perioperative management of, 102b
  perioperative risk, 101
  phenotypes/phenotyping of, 108f, 114–115

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physical examination for, 60–61
positional, 74, 97
postoperative nursing environment for, 103
precision medicine for, 107–111
preoperative diagnosis of, 101–102
prevalence of, 96
quality of life affected by, 50–51
questionnaires for, 62t, 62–63, 78, 101, 211
respiratory arousal threshold in, 42
risk factors for, 16, 36, 40, 61, 107, 115, 196
screening for, 16, 29–34, 30f–32f, 211
severity of, 64, 72, 78
signs and symptoms of
  in adults, 36, 60, 61b, 61t
  in children, 77b, 78t
sleep bruxism and, 129–130
sleep-related hypoventilation and, 37
sleep-related symptoms of, 60
snoring associated with, 61t, 77
temporomandibular disorder risks associated with, 170
tests for, 63f, 63–64
tooth loss effects on, 56
treatment/management of, 25
  adenotonsillectomy, 79, 80t
  American Academy of Sleep Medicine recommendations, 72
  anti-inflammatory medications, 79, 80t
  bariatric surgery, 74
  in children, 79–81, 80t
  continuous positive airway pressure, 72–73, 80t, 96, 110
  corticosteroids, 79, 80t
  cranial nerve stimulation, 94
  craniofacial morphology correction, 79, 80t
  decision-making, 72
  dentist's role in, 32
  drug-induced sleep endoscopy in, 68, 70, 96
  emerging concepts in, 74–75
  first-line, 72b, 72–73
  geniotubercle advancement, 94
  goals, 37
  guidelines for, 72, 73f
  hypoglossal nerve stimulation, 74
  mandibular advancement devices, 58f, 74, 87–91, 88b–89b, 97
  multidisciplinary approach to, 32–33
  oral appliance therapy, 74–75
  oral myofunctional therapy, 104–105
  orthodontics, 79, 80t
  orthopedic mandibular advancement, 79–81, 80t
  pharmacologic, 74–75, 96
  positional therapy, 74–75, 97f, 97–98
  positive airway pressure, 73, 75, 79, 80t, 102
  precision medicine, 107–111
  ribonucleic acid signatures used in, 111
  surgery, 74, 92–96, 93t
  tongue-retaining devices, 87
  tracheostomy, 94
  transcranial magnetic stimulation, 97–98
  upper airway imaging in, 70
  upper airway stimulation, 97–98, 98f
  upper airway surgery, 74, 92–96, 93t
  uvulopalatopharyngoplasty, 70, 74, 90, 94
  weight loss, 72
ultrasonography of, 67t, 68
upper airway in, 70
upper airway resistance syndrome, 36
ventilatory control in, 42, 115
in wakefulness, 60
Occipital headache, 171
Occlusion
  mouth breathing effects on, 45
  sleep bruxism-related trauma to, 153–154
Olanzapine, 203
Omics approaches, 108
Opioid(s)
  addiction to, 206
  adverse effects of, 205
  definition of, 205
  dentists' role in crisis involving, 207–208
  long-term use of, 205
  misuse of, 205–206
  orofacial pain treated with, 203–208
  problematic use of, 207
  recommendations for, 208
  screening of patients, 207–208
  sleep disruption caused by, 184–185, 206
  tension-type headache managed with, 197
Opioid Compliance Checklist, 207
Opioidergic system, 178–179
Opioid-induced hyperalgesia, 205, 207
Opioid receptors, 204
Opioid Risk Tool, 207
Opioid use disorder, 206, 206t
Oral appliance/oral appliance therapy. *See also* Mandibular advancement devices.
  contraindications for, 33
  description of, 24
  fabrication of, 33
  indications for, 33
  informed consent for, 33
  monitoring of patients with, 33–34
  obstructive sleep apnea treated with, 74–75, 89t
  side effects of, 34
  sleep bruxism treated with, 159
Oral cavity, mouth breathing effects on, 45, 46f
Oral health, 58
Oral myofunctional therapy, 84, 104–105
Orexin, 11, 201
Orofacial pain
  acute. *See* Acute pain.
  chronic. *See* Chronic pain.
  cognitive behavioral therapy for, 214
  definition of, 169
  description of, 32, 120
  electromyography activity, 191
  functional, 189–190
  headaches associated with, 192
  inflammatory, 189
  mechanisms of, 188–190
  modulatory systems for, 177
  nervous system plasticity involved in, 176–177
  nociceptive, 188–189
  opioids for, 203–208
  prevalence of, 210
  sleep deficiency and, interactions between
    analgesics for, 202, 202t
    antidepressants for, 202t, 202–203
    antiepileptics, 203
    antipsychotics for, 203
    antispasmodics for, 203
    behavioral approaches for, 183–184
    causality of, 187–192
    mechanisms of, 178–181
    pharmacologic management of, 201–204
  sleep disturbances and, 187–192
  sleep-disturbing medications for, 185
  in temporomandibular disorders, 170
  types of, 188f
Orofacial pain disorders
  characteristics of, 172t
  classification of, 169–173
  definition of, 169
  idiopathic, 173
  neuropathic, 170, 189
  neurovascular, 171, 172t
Orofacial Pain Guidelines, 119
Orthodontic treatments
  for obstructive sleep apnea, 79, 80t
  for sleep bruxism, 159
Orthopedic mandibular advancement, 79–81, 80t
OSA-18, 78

P

Pain
  chronic, 23
  definition of, 169
  endocannabinoids in modulation of, 181
  myofascial, 171t
  orofacial. *See* Orofacial pain.
  postoperative, 184–185
Pain modulatory systems, 177
Paradoxical sleep, 4, 9
Parafunction
  definition of, 119
  waking oral, 142–143, 144t, 153
Parasomnias
  definition of, 16b
  types of, 17
Paroxysmal hemicrania, 172t

Copyright Not for publication Quintessence

Passive theory, 11
Pedunculopontine tegmental nuclei, 10f
Periodic body movements, 4
Periodic limb movement disorder
description of, 19
sleep bruxism and, 130–131
Periodic limb movement index, 131
Periodic limb movements, 6
Periodontal diseases
definition of, 55
description of, 32
obstructive sleep apnea and, 55–59, 56b, 57f–58f
Periodontitis, obstructive sleep apnea and, 55, 58f
Peripheral nociceptors, 175
Peripheral sensitization, 176, 189
Persistent dentoalveolar pain, 172t, 173t
Persistent idiopathic facial pain, 172t, 173, 191
Phantom tooth pain, 173
Pharynx, 46
Phasic episode, of rhythmic masticatory muscle activity, 135
Pittsburgh Sleep Quality Index, 211
P4 medicine, 108, 111
Polysomnography
central sleep apnea findings, 36f
obstructive sleep apnea diagnosis using, 36f, 63f, 63–64, 78–79, 152, 211
overnight, 78
poor sleep quality findings, 5
rhythmic masticatory muscle activity, 135
sleep-related hypoventilation findings, 38, 38f
temporomandibular disorders evaluation, 210–211
Poor sleep
fatigue and, 3
headaches as cause of, 192, 194
migraine headaches as cause of, 194
polysomnographic findings, 5
Positional sleep apnea, 17, 74–75
Positional therapy
description of, 24
obstructive sleep apnea treated with, 74–75, 97f, 97–98
Positive airway pressure
bi-level, 73
cardiovascular disease benefits of, 53
continuous
data monitoring capabilities of, 110
mandibular advancement devices and, 88, 91
obstructive sleep apnea treated with, 72–73, 80t, 96, 110
hypertension reductions using, 52
metabolic dysfunction and, 53
obstructive sleep apnea treated with, 73, 75, 79, 102
sleep-disordered breathing treated with, 51–53
Post-herpetic neuralgia, 170, 172t
Postoperative pain, 184–185
Post-traumatic trigeminal neuropathic pain, 170
Postural muscles, 4
Pramipexole, 203
Precision medicine, 107–111
Pregabalin, 184–185, 203
Process C, 4, 11
Process S, 3, 4f, 11
Pro-inflammatory cytokines, 179
Prostaglandins, 179–180
Prosthodontics, 120
Proteomics, 115t
Psychiatrists, 23
Psychologists, 23

Q

Quality of life, obstructive sleep apnea effects on, 50–51
Questionnaires. *See also* specific questionnaire.
for obstructive sleep apnea, 62t, 62–63, 78, 101, 211
for sleep bruxism, 124–125, 125b, 148
Quetiapine, 203
Quiet sleep, 9

R

Ramelteon, 203
Rapid mandibular expansion, 80t
Rapid maxillary expansion, 79, 82–83, 83f
Rapid palatal expansion, 165
RDI. *See* Respiratory disturbances.
Relaxation training, 213t
Relay neurons, 10
REM behavior disorder
definition of, 17
description of, 19–20
sleep bruxism and, 132
tooth grinding associated with, 20
REM sleep
characteristics of, 10–11
definition of, 9
description of, 4
discovery of, 10
dreams/dreaming in, 4, 9
electroencephalogram findings, 12
monoamines in, 10
nightmares in, 18
opioid effects on, 206
postural muscles in, 4
stages of, 12f
wakefulness versus, 13
Respiratory arousal threshold, 42
Respiratory disturbance index, 36
Respiratory disturbances, 5
Respiratory effort-related arousals, 15, 191, 211
Respiratory event-related arousals, 37
Respiratory physicians, 22
Respiratory polygraphy, 79
Restless legs syndrome
description of, 19
sleep bruxism and, 130–131
Retropalatal airway, 94
Rheumatoid arthritis, 184, 189
Rhythmic masticatory muscle activity
definition of, 142
description of, 17, 127, 130–132
sleep bruxism. *See* Sleep bruxism-rhythmic masticatory muscle activity.
Ribonucleic acid signatures, 111
Risk indicators, 121, 122t

S

Screener and Opioid Assessment for Patients with Pain-Revised, 207
Screenings
obstructive sleep apnea, 29–34, 30f–32f, 211
sleep-disordered breathing, 29–34, 30f–32f
Second-order neurons, 189
Sedatives, 16, 102
Selective serotonin reuptake inhibitors, 160
Sensitization, 176–177, 189
Sensory reflexes, 47
Serotonergic system, 179
Serotonin, 150, 196, 201
Serotonin and norepinephrine reuptake inhibitors, 202–203
Serotonin receptor 2A, 148–149
Short-lasting unilateral neuralgiform headache attacks with conjunctival injection and tearing, 171, 172t
Short-lasting unilateral neuralgiform headache attacks with cranial autonomic symptoms, 171, 172t
Sleep
abnormal breathing during, 45
behavioral characteristics of, 9
cellular activities during, 13–14
definition of, 3, 9, 14, 138
duration of, 3, 6, 9
electrophysiologic correlates of, 12–13
endocannabinoids in modulation of, 181
functions of, 6, 6b, 14
genesis of, 9–11
health and, 6–7
historical studies of, 9
homeostasis of, 11
inadequate, 7
measurements of, 31
neurobiology of, 9–14, 201
non-REM. *See* Non-REM sleep.
paradoxical, 4, 9
regulation of, 11, 146
REM. *See* REM sleep.
structures involved in, 9–11, 10f–11f
suckling and smacking sounds during, 18
violent behavior during, 133

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Sleep apnea
  central. *See* Central sleep apnea.
  in children, 16
  description of, 6
  economic costs of, 7
  obstructive. *See* Obstructive sleep apnea.
  risks associated with, 16
  screening for, 211
  severity of, 15–16
Sleep apnea-hypopnea
  description of, 15–17
  gastroesophageal reflux disease associated with, 17
Sleep arousals, 5–6, 130
Sleep bruxism
  in adolescents, 162–165, 163t
  affective disturbances associated with, 143
  catastrophic structural failures caused by, 153, 153f
  characteristics of, 119, 144t
  in children, 162–165, 163t
  comorbidities, 24, 129–133
  definition of, 17, 119–120, 120t, 123–124, 142, 162
  dentition effects of, 152–154, 153f
  diagnosis of
    ambulatory monitoring, 164
    in children and adolescents, 164
    clinical examination, 125b, 125–126, 148
    electroencephalography, 163
    inaccurate, 152
    interview with patient, 164
    intraoral devices, 124b, 126
    methods used in, 125b
    polysomnography, 126–127, 127b, 152
    questionnaires for, 124–125, 125b, 148
    recording systems, 124b, 126, 127b
    sleep laboratory monitoring, 164
  differential diagnosis of, 17
  in Down syndrome, 162
  epidemiology of, 120–121, 162
  epigenetics of, 150
  epilepsy and, 132
  etiology of, 121–123, 122t, 143
  familial aggregation of, 148, 163
  gastroesophageal reflux disease and, 132–133
  genesis of, 136, 136f–137f, 140
  genetics of, 146t–147t, 146–150, 149t, 163
  heritability of, 146, 146t–147t
  implants affected by, 153f, 154
  insomnia and, 131
  load created by, 152–153
  management of
    approaches used in, 158t
    behavioral, 158t, 158–159
    biofeedback, 158, 158t
    botulinum toxin, 158t, 159–160
    in children and adolescents, 164–165
    clonazepam, 158t, 159–160
    dental interventions, 158t, 159
    electrical stimuli, 160
    "multiple P" strategy, 157, 157t
    occlusal/oral appliance for, 154–155, 158t
    orthodontics, 159
    pharmacologic, 158t, 159–160
  morning headache and, 133
  neurotransmitters involved in, 150
  obstructive sleep apnea and, 129–130
  occlusal trauma caused by, 153–154
  pathophysiology of, 162–163
  pediatric, 162–165, 163t
  periodic limb movement disorder and, 130–131
  prevalence of, 120, 121t, 142
  psychosocial factors related to, 142–143, 162
  REM behavior disorder and, 132
  restless leg syndrome and, 130–131
  rhythmic masticatory muscle activity. *See* Sleep bruxism-rhythmic masticatory muscle activity.
  risk factors for, 17, 157, 163t, 163–164
  risk indicators for, 121, 123
  severity of, 164
  sleep-disordered breathing and, 164
  stress and, 162
  teeth affected by, 152–154, 153f
  temporomandibular disorders and, 190–191, 198
  tooth cracks and fracture caused by, 154
  tooth wear caused by, 125, 154, 157
  twin studies of, 148
  waking oral parafunction and, 143–144
  wear facets caused by, 153f
Sleep bruxism-rhythmic masticatory muscle activity
  endocrine system, 138
  genesis of, 138–140
  genetic candidates, 139
  microarousals, 139–140
  neurochemical substances in, 138–139
  oral appliances for, 159
  oromotor activities, 138
  physiologic sequence of, 139
  sleep recordings, 135
  tooth grinding sounds, 135
  treatment of, 142–143
Sleep debt, 6
Sleep deficiency
  chronic pain and, interactions between
    analgesics for, 202, 202t
    antidepressants for, 202t, 202–203
    antiepileptics, 203
    antipsychotics for, 203
    antispasmodics for, 203
    behavioral approaches for, 183–184
    causality of, 187–192
    mechanisms of, 178–181
    pharmacologic management of, 201–204
  definition of, 183
  prevalence of, 183
Sleep deprivation
  adaptation to, 6
  definition of, 6
  health effects of, 7
  immune system affected by, 14
  neurogenesis affected by, 14
Sleep diaries, 195
Sleep-disordered breathing
  causes of, 45
  in children, 82
  continuum of, 96
  dentofacial morphology associated with, 48–49
  guidelines/protocols for, 29–34
  management of
    dentist's role in, 32
    multidisciplinary approach to, 32–33
    myofunctional therapy, 84
  markers of, 45
  medical history findings, 23b
  obesity and, 48
  obstructive sleep apnea. *See* Obstructive sleep apnea.
  positive airway pressure therapy for, 51–52
  screening for, 29–34, 30f–32f
  sleep bruxism and, 122, 164
  temporomandibular disorders and, 191
Sleep disorders
  costs of, 7
  medical conditions associated with, 29, 31
  in migraine headaches, 195–196
  orofacial pain and, 187–192
  periodontal diseases associated with, 55
  screening for, 210–211
Sleep efficiency, 5
Sleep fragmentation
  cyclic alternating pattern associated with, 6
  in obstructive sleep apnea, 52
  sleep debt caused by, 6
Sleep hygiene education, 211, 212b, 213t
Sleep hypopnea, 6
Sleepiness, 17, 50–51, 62–63
Sleep medicine. *See* Dental sleep medicine.
Sleep oscillations, 13–14
Sleep physicians, 22
Sleep pressure, 3
Sleep quality, 3
Sleep recordings
  description of, 5–6
  sleep bruxism-rhythmic masticatory muscle activity recognized from, 135
Sleep-related breathing disorders
  centra sleep apnea. *See* Central sleep apnea.
  classification of, 35b
  continuous positive airway pressure for, 88
  morbidity and mortality risks, 34
  obstructive sleep apnea. *See* Obstructive sleep apnea.
  sleep-related hypoventilation, 37–38
  types of, 16–17

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Sleep-related breathing events, 5, 16b
Sleep-related disorders, 133
Sleep-related hypoventilation, 37–38, 38f
Sleep-related laryngospasm, 18
Sleep-related movement disorders
  definition of, 16b
  medical history findings, 23b
  sleep complaints associated with, 24b
  types of, 19b
Sleep Research Society, 183
Sleep restriction, 213t
Sleep stages
  description of, 13
  dreaming in, 4
  electroencephalogram patterns, 13
  N1, 4, 5f, 13
  N2, 4, 5f, 13
  N3, 4, 5f, 13
  N4, 13
Sleep talking, 18, 133
Sleep terrors, 18
Sleep-wake cycle
  as homeostatic process, 3
  characteristics of, 201
  description of, 3
  developmental changes in, 6
  diagram of, 5f
  feeding behaviors and, 3
  24-hour, 3–4
Sleep walking, 133
Slow-wave sleep
  definition of, 9
  description of, 3
  monoamines in, 10
Snoring
  description of, 15
  management of
    dentist's role in, 30f
    first-line options, 33
  in obstructive sleep apnea, 61t, 77
Social history, 23b
Soluble intercellular adhesion molecule-1, 56
Somniloquy, 18
Spatial modulation of magnetization, 68
Sterol regulatory element binding protein, 114
Stimulus control therapy, 213t
STOP-BANG questionnaire, 16, 62, 78t, 101–102, 195, 211
Stress, 162
Stridor, 18
Supervised machine learning, 108, 109t
Suprachiasmatic nucleus, 4, 11
Surgery, upper airway
  complications of, 94–95
  obstructive sleep apnea treated with, 74, 92–95, 93t
Suvorexant, 203
Swallowing abnormalities, 18, 20

T

Tagged magnetic resonance imaging, 70
Taxonomy, 169
Teeth
  cracks in, 154
  fracture of, 154
  grinding of, 17, 20, 120, 135. *See also* Sleep bruxism.
  loss of, obstructive sleep apnea and, 56
  sleep bruxism effects on, 152–154, 153f
  tapping of, 17–19
  wear of, 125, 154, 157
Temporal arteritis, 173
Temporomandibular disorders
  characteristics of, 172t
  classification of, 170
  diagnostic criteria for, 171t
  incidence of, 170
  obstructive sleep apnea and, 24, 170
  pain associated with, 170
  sleep bruxism and, 190–191, 198
  sleep-disordered breathing and, 191
  studies of, 210–211
  waking oral parafunction and, 143
Tension-type headaches, 20, 192, 195b, 197
Tensor palatini muscle, 47
Tetrahydrocannabinol, 181
Thalamic nuclei, 10
Thalamocortical circuit, 13
Thalamocortical neurons, 10
Tocilizumab, 184
Tongue-retaining devices, 87
Tonic episode, of rhythmic masticatory muscle activity, 135
Tonsils, 44
Tooth. *See* Teeth.
Toothache, 191
Tracheostomy, 94
Transcranial magnetic stimulation, 97–98
Transcriptomics, 115t
Transoral robotic surgery, 94
Transpalatal advancement, 94
Traumatic brain injury, 198
Traumatic trigeminal neuropathic pain, 189
Trazodone, 203
Triazolam, 184
Tricyclic antidepressants, 202–203
Trigeminal autonomic cephalalgias, 171
Trigeminal neuralgia, 170, 172t, 192
Triptans, 197
Tryptophan, 138
Tuberomammillary nucleus, 10f
Tumor necrosis factor-α, 57, 114
24-hour sleep-wake cycle, 3–4

U

Ultradian rhythm, 4–5
Ultrasonography, for obstructive sleep apnea, 67t, 68
Unsupervised machine learning, 108, 109t, 111
Upper airway
  in adolescents, 44
  anatomy of, 39f, 39–40
  critical closing pressure of, 40
  development of, 44
  functions of, 39
  imaging of, 66–70, 67t–68t
  muscles of
    anatomy of, 40–42, 41f
    anesthesia-related relaxation of, 100
  obesity effects on, 101
  obstruction of, 37f
  in obstructive sleep apnea, 70
  soft tissues of, 70
  tooth loss effects on, 56
Upper airway resistance, 82
Upper airway resistance syndrome, 36
Upper airway stimulation, 97–98, 98f
Upper airway surgery, for obstructive sleep apnea, 74, 92–95, 93t
Uvulopalatopharyngoplasty, for obstructive sleep apnea, 70, 74, 90, 94

V

Vedolizumab, 184
Venlafaxine, 203
Ventilatory control, 42, 115
Ventrolateral preoptic nucleus, 11, 11f
Vertex waves, 13

W

Wake after sleep onset, 5
Wakefulness
  chewing during, 136
  cortical activation during, 10
  duration of, 4
  electroencephalogram recordings, 12
  movement disorders during, 19b
  neurobiology of, 201
  obstructive sleep apnea symptoms during, 60
  REM sleep versus, 13
Wake-sleep cycle. *See* Sleep-wake cycle.
Waking
  regulation of, 146
  stages of, 12f
Waking oral parafunction, 142–143, 144t, 153
Whole genome sequencing, 115, 115t
Widespread pain. *See* Fibromyalgia.

Z

Zopiclone, 184