

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





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

Library of Congress Cataloging-in-Publication Data

Names: Lavigne, Gilles J., editor. | Cistulli, Peter A., editor. | Smith, Michael T. (Michael Timothy), 1967- editor.
Title: Sleep medicine for dentists : an evidence-based overview / edited by Gilles J. Lavigne, Peter A. Cistulli, Michael T. Smith.
Description: Edition 2. | Batavia, IL : Quintessence Publishing Co, Inc, [2020] | Includes bibliographical references and index. | Summary: "This book provides a rapid source of practical information to students, practicing dentists, and scientists about the evolving field of dental sleep medicine"-- Provided by publisher.
Identifiers: LCCN 2019056811 | ISBN 9780867158281 (hardcover) | ISBN 9781647240097 (epub)
Subjects: MESH: Sleep Wake Disorders | Dentistry--methods | Mouth Diseases--complications | Tooth Diseases--complications
Classification: LCC RC547 | NLM WL 108 | DDC 616.8/4980246176--dc23
LC record available at <https://lccn.loc.gov/2019056811>



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Quintessence Publishing Co, Inc
411 N Raddant Rd
Batavia, IL 60510
www.quintpub.com

5 4 3 2 1

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Editorial: Bryn Grisham & Samantha Smith
Design: Sue Zubek
Production: Sue Robinson

Printed in the United States

Second Edition

SLEEP MEDICINE FOR DENTISTS

AN EVIDENCE-
BASED OVERVIEW



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QUINTESSENCE PUBLISHING

Berlin | Chicago | Tokyo

Barcelona | London | Milan | Mexico City | Moscow | Paris | Prague | Seoul | Warsaw

Beijing | Istanbul | Sao Paulo | Zagreb



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Foreword



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



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



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



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

The Nature and Structure of Sleep

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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.

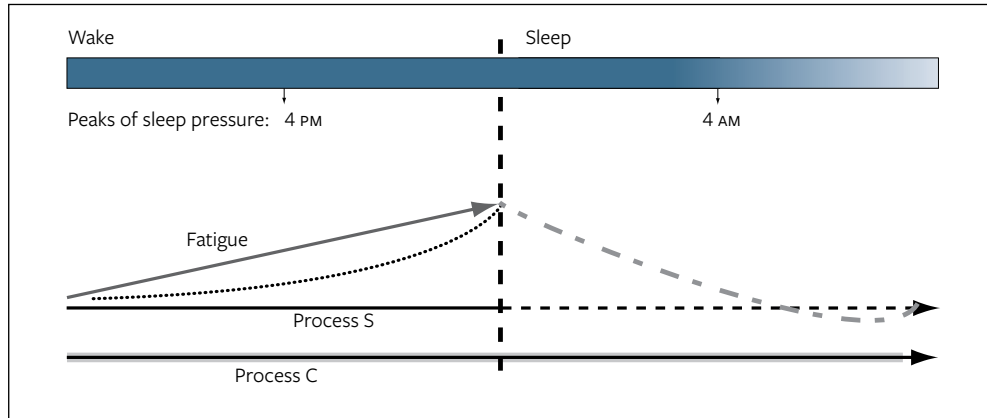


FIG 1-1 Normal cycle for circadian rhythm (process C) (solid black arrow) and process S (solid black line/dashed arrow) over about 24 hours. During wakefulness periods, the increase in sleep pressure (dotted line), parallels the increase in fatigue (gray arrow) and results in sleep (dashed and dotted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

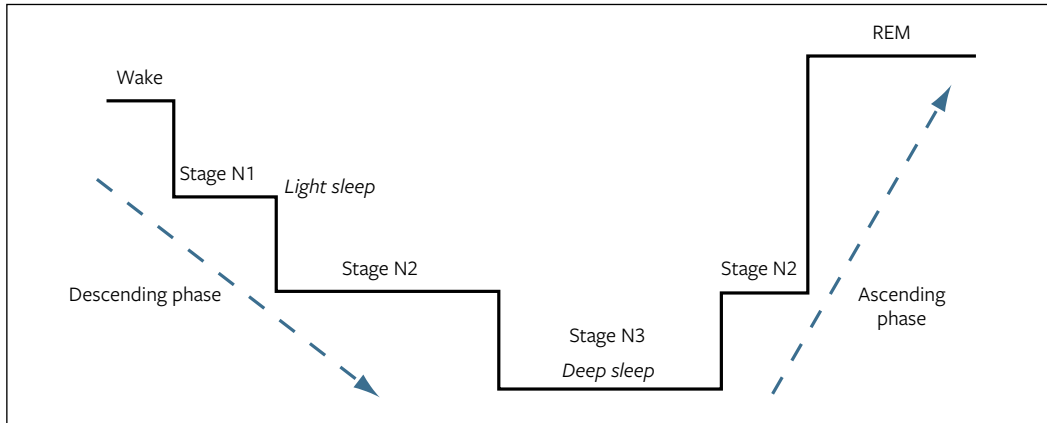


FIG 1-2 One NREM-to-REM cycle of consecutive sleep stages. This cycle is repeated every 70 to 110 minutes for a total of three to five NREM-to-REM cycles per sleep period.

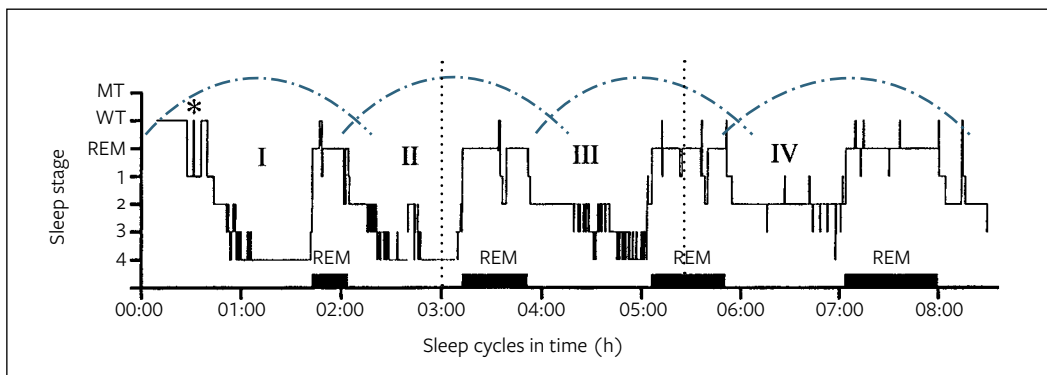


FIG 1-3 Consecutive waves of NREM-to-REM (solid horizontal boxes) sleep cycles (I to IV). During the first third of the night, slow-wave sleep (stage N3) is dominant. During the last third of the night, the REM stage is longer. MT, movement time; WT, wake time. (Adapted from Lavigne et al⁸ with permission.)

- 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.



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/behaviors¹⁵⁻²² (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

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 day²⁹ (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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