SLEEP DISORDERS and SLEEP PROMOTION in NURSING PRACTICE

Editors
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Geoffry Phillips McEnany, PhD, PMHCNS, BC, is a professor at the University of Massachusetts at Lowell. In his academic work, he teaches at the Masters and PhD level and coordinates the international and interdisciplinary online Sleep and Sleep Disorders program. He is Associate Editor for *Issues in Mental Health Nursing*. He is on the advisory board for the National Educational Council of the American Psychiatric Nurses Association, advancing education for psychiatric nurses with a particular focus on sleep-related issues in the clinical care of persons diagnosed with psychiatric disorders. In this role, he has launched large-scale international educational initiatives directed toward meeting the sleep-related learning needs of psychiatric nurses in practice and academic work. He maintains a small practice in Boston where the focus of his work is in the care and treatment of adults with psychiatric illness and comorbid sleep disorders. Dr. Phillips McEnany completed his initial nursing education at the Massachusetts General Hospital School of Nursing in Boston. He continued his nursing studies in California, earning a Bachelor of Science in Nursing from the University of San Francisco. He completed his graduate education at the University of California San Francisco where he earned a Master of Science in Psychiatric Nursing and later completed his PhD studies. He was awarded a 2-year postdoctoral fellowship through the Agency for Healthcare Quality and Research. His initial research concentrated on sleep and biological rhythm dysregulation in women diagnosed with major depression. His current research is exploring sleep dysregulation and psychiatric/medical comorbidities in immigrant populations.

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To Jim Redeker, husband, partner, and best friend who accepts my long hours of work on this and other projects with good humor and love.

To Lois W. Schmieder, RN, who taught me what it means to be a nurse.

—Nancy Lou Schmieder Redeker

To Brian Phillips McEnany and Karen McEnany Farrington for their powerful presence in my life that is always loving and unconditionally supportive. They are treasured and celebrated examples of the richness of family.

—Geoffry Phillips McEnany

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Preface

The science of sleep and sleep disorders treatment has come of age, as demonstrated by inclusion of sleep-related goals in *Healthy People* 2020, released as our book goes to press. These goals build on incredible advances in basic, clinical, and community-based science addressing sleep and sleep disorders, including the notable contributions of nurse scientists (many of whom are contributors to this book), to this interdisciplinary field. There is no doubt that sleep contributes positively to health and well-being, and sleep disorders contribute to morbidity, mortality, and decrements in functional capacities, and quality of life; and there is powerful evidence of the efficacy of sleep promotion strategies. We wrote *Sleep Disorders and Sleep Promotion in Nursing Practice* to provide a reference for nurse educators, advanced practice nurses, and other nursing leaders on sleep and sleep disorders; to integrate the science of sleep into nursing practice, education, and research; and to facilitate the uptake and translation of evidence-based assessment and treatment of sleep into community-based and clinical settings where nurses practice.

Our focus is based on the need for nurses to have a strong grasp of the essence of sleep, sleep disorders, and practical strategies to integrate this information. We expect this book to be a critical resource for faculty teaching in prelicensure nursing programs, master's, and practice and research doctoral levels. We also expect that our book will serve as a seminal reference for advanced practice nurses across a variety of specialties.

As documented in a recent report commissioned by the Institute of Medicine (Colten & Altevogt, 2006), there is a tremendous gap between the strength of the evidence about sleep and sleep disorders and its dissemination, translation, and uptake into community and clinical settings—a problem partially explained by the lack of knowledge about sleep and sleep disorders among health care providers, including *nurses*. Although deficiencies in content and clinical experiences related to sleep and sleep disorders in formal nursing and medical educational programs and postgraduate continuing education have been well documented, there have been few systematic attempts to address this problem in *nursing education*. Even when nurses are knowledgeable about sleep, limited time and resources in clinical settings are major barriers to improving access to sleep promotion and sleep disorders treatment. Our book will address these important gaps.

Sleep Disorders and Sleep Promotion in Nursing Practice is written from a nursing perspective and incorporates interdisciplinary research, as befits this expansive field. It is based on our (NSR, GPM) extensive experiences in sleep research, practice, and nursing education. As veteran nurse educators, we understand the incredible demands for time in the nursing curriculum and the need for faculty expertise. We agree with Virginia Henderson (1955), who identified sleep and rest as one of the 14 basic "human needs," and Florence Nightingale who noted the importance of sleep to human health and healing (1860) that *sleep promotion is a fundamental element of nursing practice*. By virtue of our broad focus on health promotion, disease prevention, expertise in lifestyle, and behavioral change coupled with biobehavioral perspectives, nurses are well suited to promote sleep and assist individuals, families, groups, and

communities to prevent and treat sleep disorders. Although numerous texts are available on "Sleep Medicine," to our knowledge, there is no text on "Sleep Nursing." This text is one way of promoting the development of this field.

Our book emphasizes the importance of sleep across states of health, health care settings, and at all stages of human development. It uses an evidence-based approach to synthesize and integrate nursing and interdisciplinary research on sleep to serve as a foundation for curriculum, teaching, practice, and research activities. We recognize differences in the scope of nursing practice based on educational preparation, certification, and nursing specialty, and have included references to these distinctions, especially in Unit IV. However, as in other areas of nursing practice, the distinctions are not always clear-cut. The evolution of the science of sleep with its growing presence in the work of our clinically focused discipline is just beginning, and the chapters in this book reflect these exciting advancements.

The book is organized into four units that build on and complement one another. In Unit I, we address aspects of normal sleep, including normal sleep physiology and behavior and developmental and gender aspects. In Unit II, we provide an overview of the importance of sleep and its consequences from an epidemiological perspective, extant sleep-related nosologies, and a guide to sleep assessment. In subsequent chapters of Unit II, we discuss the epidemiology, consequences, assessment, and treatment of the most prevalent sleep disorders, discuss health disparities associated with sleep disorders, and provide an overview of complementary and alternative therapies for sleep. In each of these chapters, we discuss nursing implications and provide Web-based resources that can be used to obtain additional information. Where appropriate, we provide case studies that can be used as exemplars for learning purposes. There is some overlap in the chapters to facilitate a clear presentation, and the book is extensively cross-referenced. However, many of the chapters can serve as stand-alone references, especially if the reader has some knowledge about the basics of sleep.

Unit III focuses on integration of the clinical research on sleep into specific settings where many nurses work (occupational health, primary care, acute care, long-term care, psychiatric settings, pediatric primary care, pediatric acute care, and maternal-child health). These chapters represent our efforts to better define and "prescribe" the specific elements of sleep promotion and sleep disorders treatment that are most relevant to these settings in an attempt to provide a structured approach. While there is considerable literature about sleep in some settings (e.g., acute and critical care), there is little efficacy or effectiveness research conducted on sleep interventions delivered by nurses in any setting and few available guidelines for nursing practice. We expect that the readers of this book will use and test our suggestions that are based on currently available evidence, and that as a result, best practices will be refined in the future. In short, we recognize that best practices regarding sleep in the majority of settings are "works in progress." The chapters in Unit III are meant to stimulate ideas about these issues.

In Unit IV, we present suggestions about ways to integrate sleep and sleep disorders into pre-licensure and graduate nursing education, ideas about the future of nursing practice related to sleep, and proposed directions for future research. This work extends the white paper "Sleep and Chronobiology: Recommendations for Nursing Education," published in *Nursing Outlook* (Lee et al., 2004), the product of an ongoing collaboration among a group of "nurse somnologists" (i.e., sleep scientists) who recognized the importance of sleep in nursing curricula at a time when the national focus was on sleep in medical school education. *Sleep Disorders and Sleep Promotion in Nursing Practice* extends this work with ideas for updated learning objectives, placement of content within curricula, and resources, including Web sites. This information is supplemented by a Web site supported by our publisher, Springer.

We are incredibly excited about the collaboration represented in this book and gratified by the fact that not one potential author refused our invitation to contribute, despite

their busy schedules. Our contributors are part of a growing and impressive cadre of "nurse somnologists," and clinicians and educators who specialize in sleep. Some have been leaders in the field for many years. Others are now part of an emerging second generation of sleep experts. We invited each of the contributors because of their expertise in a specific area of this very complex field. Their sleep interests were nurtured by their experience in traditional areas of nursing (e.g., pediatric nursing, psychiatric nursing, maternal-child health, and acute care nursing). In turn, their sleep expertise enriches these fields. We the coeditors (NSR and GPM) are good examples of this diversity, and our experience is quite complementary. NSR began her career as a coronary care unit nurse and initially focused on sleep in the acute care setting and now focuses on sleep in adults with medical comorbidity, especially heart disease. GPM, an advanced practice nurse in psychiatric mental health nursing, focuses extensively on sleep and its relationship to psychiatric illnesses as well as the bidirectional comorbidities associated with both sleep and psychiatric disorders. Just as we arrived at our interests in sleep through very different routes, we expect that other nurses will have similarly diverse career trajectories. Some will become sleep specialists. The vast majority will effectively integrate sleep promotion and prevention and treatment of sleep disorders into all domains of nursing practice.

Nancy Lou Schmieder Redeker
Geoffry Phillips McEnany

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Foreword

Many kinds of evidence point to sleep being essential for health, including observed relationships between inadequate sleep and symptom, functional performance deterioration, physical and mental disease vulnerability, on through to premature mortality. In this book, contemporary sleep–wake knowledge is generatively profiled through the lenses of accomplished nurse scientists. As a prelude to delving into the details within this book, I briefly comment on sleep as addressed in this book, the importance of understanding sleep in synergy with wake, and the obvious alignment with nursing practice and research.

Sleep, a health-essential behavior, is regulated or influenced by three interactive components: (1) a sleep drive component that is a function of daily oscillations in the relative balance of brain neurochemicals that modulate alertness/sleepiness; (2) a circadian component that functions in synchrony with the 24-hour light–dark environmental cycle; and (3) a behavioral component by which individuals can facilitate or interfere with the regulation of sleep. Thus, sleep is a neurobehavioral phenomenon in synchrony with wake such that brain functions, either emotional or physical, can disrupt sleep.

Addressed in detail in this book, you will see profiled in Chapters 1–3 extant knowledge of sleep as a human health phenomenon and in Chapters 4–15 the plethora of conditions across the lifespan that encompass disrupted sleep manifestations, including insomnia, sleep-related breathing, movement, and circadian disorders and narcolepsy, along with sleep issues associated with chronic physical and mental conditions. These all point to the scope and importance of sleep as a health issue and summarize the enormous burden on individuals and society. Burden represents the impact on society or on individuals, measured by such indicators as financial cost, premature death, associated life restrictions (disabilities) or comorbidities, enduring symptoms, or other indicators that are often incorporated into the concept of health-related quality of life, a concept very much a part of nursing science. True to a long-standing nursing practice value for health promotion, Chapters 16–23 convey sleep promotion strategies in the context of a variety of practice environments and nursing perspectives and identify evidence-based strategies for sleep promotion in these settings.

SLEEP DISRUPTIONS: A MAJOR HEALTH PROBLEM

Indicative of the importance of addressing sleep as a major health problem are the prevalence, consequences, and scope of sleep disturbances, briefly mentioned here, but detailed in this book. The most commonly reported sleep problem is insomnia, a symptom and a condition, defined as perceiving inadequate sleep (quality or amount). Reported by upwards of one-third of the U.S. populations at any point in time, about 10% of people claim to have enduring insomnia and about 5%–15% report negative functional impact. Overall, women tend to report more sleep difficulties than men; the proportion of women reporting insomnia escalates in midlife. However, it remains unclear how much the fluctuations in reproductive hormones and accompanying vasomotor symptoms (hot flashes, night sweats) contribute. Insomnia is known to accompany many mental

and physical disease/illness, and high stress-producing (emotionally charged) situations convey risk. However, insomnia manifests in vulnerable individuals with no overt contributing factors (primary insomnia) and in its most chronic form manifests in individuals with a propensity for heightened emotional arousal and stress physiological activation. As will be seen in this book, the burden of insomnia to people and society is striking. For example, a financially conservative estimate of total annual U.S. costs, including direct costs of treatment and indirect costs were from $92.5 billion to $107.5 billion in 1994.

As detailed in this book, numerous other sleep-disrupting conditions occur and convey significant burden. These include a spectrum of sleep-disordered breathing patterns that span from upper airway resistance syndrome to sleep apnea-hypopnea syndrome—estimated to affect about 4% of midlife males and 2% of midlife females in the U.S. population. Other sleep-disrupting neurological conditions include movement abnormalities, most particularly periodic leg movements during sleep, that is, repeated rhythmic twitching movements of lower and occasionally upper limbs and restless legs syndrome, that is, unpleasant creepy or crawly sensation in the legs accompanied by an irresistible urge to keep the legs moving or walking around, which has been associated with low levels of serum iron, is more common in women (28%) than men (21%), and occurs in up to 15% of women during the last trimester of pregnancy. Narcolepsy, a condition of disturbed sleep–wake cycle regulation, is said to affect 1 in 2,000 people, although it is probably under-recognized and manifests in a spectrum of abnormal sleep patterns. A variety of disruptions to the circadian component of sleep occur, such that sleep is out of typical synchrony with the 24-hour light–dark cycle. These circadian abnormalities are driven either by physiological abnormalities or behavioral interference and are quite familiar example to nurses who work night shifts. Couple all of the aforementioned sleep problems with sleep disturbances that accompany many if not most physical and mental medical conditions (e.g., heart disease, cancer, arthritis, mood, and thought disorders), and the essence of sleep disturbances as a critical health problem is undeniable. Thus, there is a need for greater attention to promoting research and clinical nursing education. Forward thinking recommendations are highlighted throughout this book.

SLEEP AS A HEALTH ECOLOGICAL NURSING PRACTICE PHENOMENON

From evidence detailed in this book, understanding sleep has essential relevance for human health/wellness and, in my view, serves as an exemplar for transforming how nursing practice and science might be framed. For most in nursing, our "unit of interest" is predominantly the "person," generally along with their family or close support people; for some, the interest is communities, populations, and systems. Lifestyle behaviors are believed to outweigh even genetics in contributing to chronic disease/illness. Yet, we do not have a consensus framework for guiding nursing curricula and practice accordingly. It is my contention that the biomedical organ-system approach to defining health status continues to pervade the framing of our nursing curricula and influence nursing practice. Yet, we in nursing would be better served by articulating health dynamics differently, perhaps in terms of human health-related lifestyle functionality domains within a health ecology framework with an emphasis on person and environment. Accordingly, person "fit" with environment occurs through behavioral interactions with various components of environments and "goodness of fit" determines health/wellness status or dynamics. As we in nursing are aware, understanding environments is essential to understanding human health behaviors (or the behaviors of any systems, be it whole person or molecular). Accordingly, health/wellness status or outcomes could be framed across a set of age-span lifestyle functionality domains and understood as biobehavioral phenomena in the context of humans developing and aging. Consequently, we might view much of nursing practice as being aimed at helping people restore/heal, maintain/sustain,

or promote/enhance optimal lifestyle functionality in the context of aging, life transitions, and injury/chronic conditions. Sleep–wake, as profiled in this book, provides a robust example of a critical age-span lifestyle functionality domain. Without being comprehensive, further examples are as follows: breathing/oxygenating; moving/exercising; interacting/communicating; and thinking/processing/sensing/feeling/learning. Thus, the content in this book serves as an exemplar area for understanding human health as a way to appropriately transform nursing curricula and practice.

SLEEP RESEARCH FOR NURSING PRACTICE

Knowledge about sleep comes from a variety of research approaches. Experimentally, sleep is studied in basic biomedical science as the physiology of sleep regulation, mainly using animal models. Epidemiologically, patterns of sleep are studied in populations, often defined in terms of age (young, old), gender, or ethnicity. Sleep patterns also are descriptively studied during life transitions or in specific contexts, particularly those thought to be stressful: for example, pregnancy, menopause, or relocation. As might be surmised, a large amount of sleep or sleep-related physiology research is done to describe sleep-related disorders or how various physical or mental diseases/illnesses (e.g., heart disease, cancer, fibromyalgia, and depression) or symptoms (e.g., pain) affect sleep patterns or, alternatively, how sleep patterns affect the course of disease/illness or modulate symptom experiences. Less investigation has gone into testing of therapeutics. However, tests of therapies to promote sleep in a variety of contexts are evident: for example, sleep restriction to treat insomnia or environmental manipulation to promote sleep in the context of adult or neonatal critical care. Needed are more studies to test the therapeutic effects of sleep (the effects of improved or optimal sleep on health outcomes): for example, tissue healing and cognitive improvement.

Most studies done by nursing scientists have been mainly descriptive and have addressed sleep in illness and stress conditions and in vulnerable and "at risk" groups. Remaining challenges for greater understanding of sleep as a lifestyle functionality domain include deriving stronger, plausible theoretical/conceptual views on sleep quality, sleeplessness, and sleepiness; creating and testing novel therapies based on theoretical perspectives; and better understanding the complexity, burden, and cost (of either poor sleep patterns or interventions). From a human ecological perspective, benefits would be accrued from testing more interventions with relevant behavioral modification in concert with modulation of environments or contextual factors. More sleep promotion science is needed to address choice and adherence dynamics; timing and exposure (dose); titration, personalization, or tailoring—using combined physiological, experiential, and behavioral measures.

In summary, by reading this book, you will come to understand the vast importance of sleep to human health and the scope of disordered sleep that occurs in multiple lifestyle and life contexts. The content herein represents a robust example of a human lifestyle functionality phenomenon that deserves greater attention within the discipline of nursing and in healthcare. Its authorship—top nursing clinical scientists in the field—makes this book a seminal touchstone from which to contemporize and transform nursing practice, curricula, and science.

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Acknowledgments

Building bridges has its challenges and rewards. The process starts with seeing the span and imagining what could be built. A decision to move forward requires careful collaboration among architects and engineers, giving attention to form and function, assuring both durability and esthetics. Breaking ground and successfully creating the structure entails working in thriving partnerships, drawing on the talents and skills of the experts, and fostering the apprentices to assure that future bridges will be built in ways that are strong, functional, and attractive. In many ways, the path of nurses in the field of sleep has paralleled the labors of those that build bridges. But our landscape is science, and the bridges we build make connections that allow us to explore and nurture new clinical and scientific ground. The fruits of these labors benefit those we serve in our work as nurses.

We are very grateful to our colleagues who have contributed so generously to this book, a bridge of scholarship for others in our discipline. The chapters represent the works of nursing scientists, academicians, and clinicians. In their writing, each author has provided fine exemplars of creativity, rigor, and an impressive capacity for translation of scientific evidence for use in the clinical work of nurses across all specialties. We feel fortunate to have had the honor of working with each one in the creation of this book and believe that their efforts have contributed to exciting awakenings for our colleagues in nursing.

Physiological and Behavioral Aspects of Sleep

1

Carol A. Landis

Sleep is a multidimensional, biobehavioral process that is essential to human health and function. Knowledge of its biological and behavioral aspect has grown exponentially over the past several decades. However, the restorative functions of sleep are still not well understood. The purpose of this chapter is to explain the behavioral and physiological aspects of normal sleep and strategies used to measure them. This information is foundational to the understanding of sleep disorders and their pathological consequences and providing nursing care that focuses on sleep promotion and sleep disorders treatment.

DEFINITION OF SLEEP

Behaviorally, sleep is defined as a temporary perceptual disengagement from and unresponsiveness to environmental surroundings (Carskadon & Dement, 2005). Sleep and wake are alternating behavioral states over the course of a 24-hour day. During wakefulness, but not sleep, one is alert and conscious of one's surroundings. Typical sleep behaviors include closed eyes, little movement, recumbent posture, and reduced responsiveness to stimulation. The intensity and duration of sleep is increased after a period of sleep loss. Sleep is sometimes described as an unconscious state, but this is inaccurate. People who are comatose or under the influence of anesthesia or sedation display sleep-like behaviors, but these unconscious states are not naturally or easily reversed, and thus are not "usual" sleep. Rest is a behavior that also resembles sleep. However, one can lie awake for hours "resting" quietly in bed and still feel tired and perform poorly the next day due to the lack of adequate sleep.

SLEEP AND HEALTH

Sleep is a biological necessity, but despite decades of research, the functions of sleep remain poorly understood. Recently, somnologists (scientists who study sleep mechanisms and the consequences of sleep loss) (Institute of Medicine [IOM], 2006) have begun to ask once again, "what is sleep?" (Franken, Kopp, Landolt, & Luthi, 2009). They argue that any definition of sleep must include reference to its function or functions. Sleep has long been considered restorative, yet little is known about the brain and bodily functions that are restored during sleep.

Studies in animals, in this regard, show that sustained loss of total sleep or substantial loss of particular sleep stages leads to failure of body temperature regulation, increased metabolism, deterioration of hypothalamic neurons, progressive breakdown of host defenses, and death (Rechtschaffen & Bergmann, 2002; Siegel, 2005). These observations support theories that sleep functions to conserve energy and metabolism; keep physiological systems within proper homeostatic limits; maintain host defenses; and reverse or restore physiological processes that get progressively degraded during wakefulness (Hobson, 2009; Vassalli & Dijk, 2009). Scientists increasingly recognize that sleep may serve distinct functions at cellular/molecular, organ system, and whole organism levels of analysis.

Adequate sleep is important for optimum human health. Excessive daytime sleepiness is a major public health problem associated with poor job performance, reduced ability to handle daily

stressors, greater alcohol use, and a higher incidence of drowsy driving, especially among young adults (IOM, 2006). Habitual sleep of less than 6–7 hours each night is associated with increased blood pressure, reduced blood levels of anabolic hormones (e.g., growth hormone and prolactin) required for tissue repair, higher blood levels of inflammatory cytokines, and the stress hormone cortisol during evening hours when cortisol is usually low (Mullington, Haack, Toth, Serrador, & Meier-Hwert, 2009). Daytime sleepiness deficits in performing simple tasks; reduced motivation and negative mood are among the most consistent consequences of sleep loss and insufficient sleep (Pilcher & Odle-dusseau, 2005). Individuals whose sleep is restricted to 6 or fewer hours each night for 1 week under experimental conditions had cognitive impairments equal to that of a night or two of total sleep loss, but they often reported minimal sleepiness and overestimated their ability to carry out simple tasks (Van Dongen, Maislin, Mullington, & Dinges, 2003).

Research on the function of sleep and roles of specific sleep stages in memory processes and learning is interdisciplinary and controversial (Diekelmann & Born, 2010; Hobson, 2009; Vassalli & Dijk, 2009). However, adequate sleep before or after learning a task enhances performance and is necessary for memory stabilization and consolidation (Diekelmann & Born, 2010; Yoo, Hu, Gujar, Jolesz, & Walker, 2007). Sleep loss biases mental processes toward remembering negative events and emotions, and may explain the increased negative mood often reported after sleep loss. Regardless of the specific biological mechanism for the restorative function of sleep, it is clear that inadequate sleep poses serious personal and public health consequences.

SLEEP STAGES AND CYCLES

Sleep Stages

Sleep is divided into two distinct stages—non–rapid eye movement (NREM) and rapid eye movement (REM) sleep. NREM sleep is divided into three stages (Stages 1, 2, 3). These gradually deepen and are associated with larger and slower brain wave activity.

The staging of sleep is based on brain wave activity (electroencephalography), eye movements (electro-oculography), and muscle tone (electromyography).

At sleep onset, the electroencephalogram (EEG) pattern of low amplitude (peak-to-peak difference), high frequency (>15 hertz [Hz] or cycles per second) activity of wakefulness disappears and is replaced by Stage N1. Stage N1 is a transitional stage between drowsiness and sleep, indicated by a shift from EEG alpha waves (8–12 Hz). It is associated with relaxed wakefulness and low-voltage, theta (4–8 Hz) or vertex sharp waves, and absence of other sleep stage signs. An individual usually spends only a few minutes in Stage N1, followed by Stage N2 sleep. Stage N2 is characterized by specific types of EEG waveforms called sleep spindles (12 and 14 Hz) and K complexes (large slow waves of <1 Hz) (see Figure 1.1). Most of a night of sleep is spent in Stage N2. Stage N3 follows Stage N2 and is often called slow-wave sleep (SWS). In the original scoring system for NREM sleep stages (Rechtschaffen & Kales, 1968), SWS was differentiated into Stages 3 and 4. In the revised sleep scoring system, Stages 1 and 2 were retained with only slight modifications, but Stages 3 and 4 were combined into Stage N3 or SWS (American Academy of Sleep Medicine, 2007). In SWS, brain waves become highly synchronous and are of large amplitude and slow or delta frequency (<4 Hz) (see Figure 1.1).

REM sleep follows NREM sleep and is characterized by episodic bursts of rapid side-to-side (saccadic) eye movements, postural muscle atonia, and a low amplitude EEG pattern that resembles waking. REM sleep occurs in discrete periods throughout the night and is associated with vivid dreams.

Young adults spend 2%–5% of the night in N1, 45%–55% in N2, 13%–23% in N3, and 20%–25% in REM sleep (Carskadon & Dement, 2005; Carskadon & Rechtschaffen, 2005). With age, the percentage of time spent in N1 and N2 increases, whereas SWS decreases. By the 5th or 6th decade, SWS sleep is only a small percentage of a typical night of sleep or may be completely absent. The percentage of REM sleep remains fairly stable, but declines in old age (see Chapter 2).

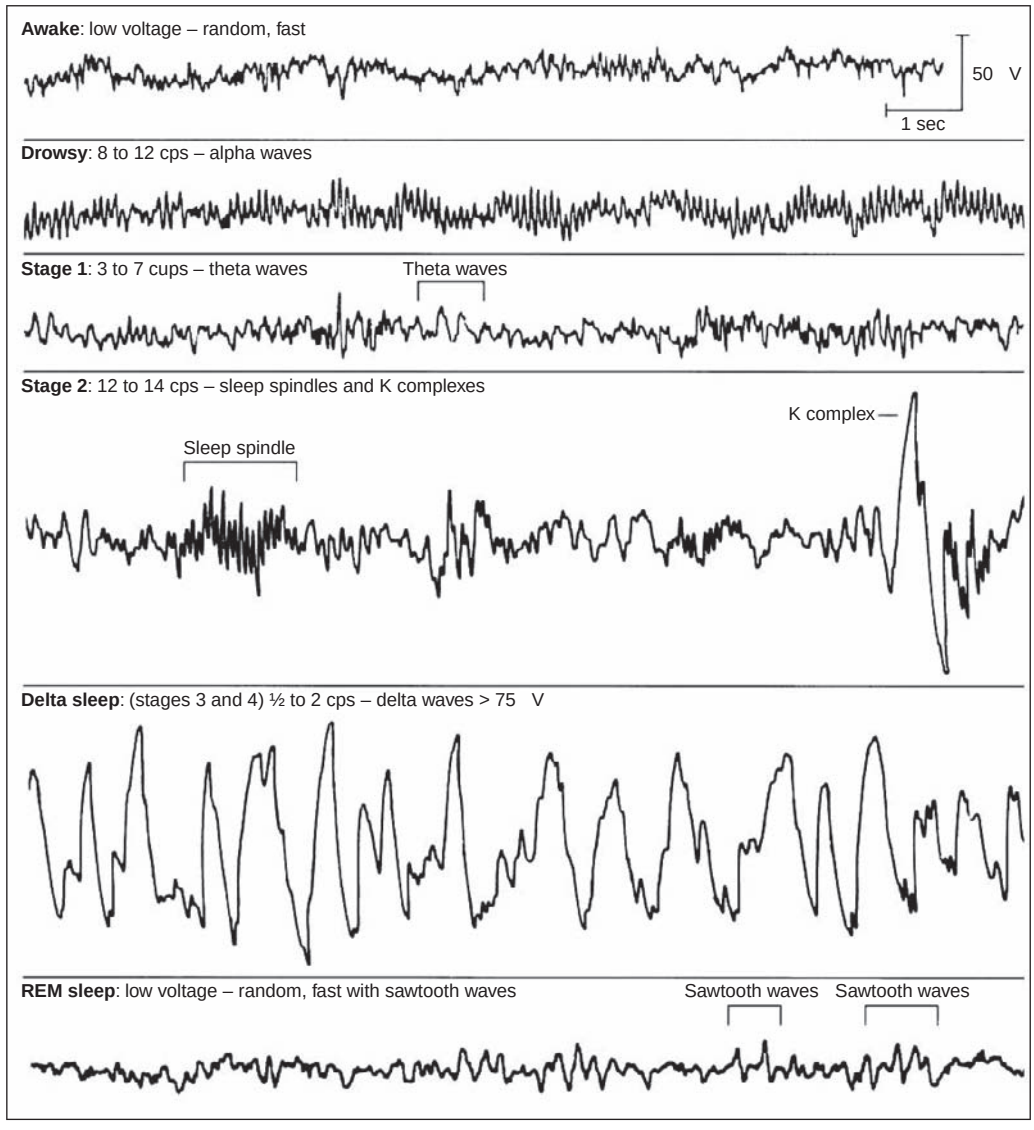

Figure 1.1 ■ Characteristic electroencephalogram wave forms by sleep stage. *Source: Atlas of Clinical Sleep Medicine* by M. H. Kryger, 2009, Philadelphia: Saunders-Elsevier. Reprinted with permission.

Sleep Cycles

Sleep is distributed in a cyclic pattern across the night. One complete sleep cycle includes an episode of NREM sleep followed by an episode of REM sleep. In adults, a typical night of sleep consists of 4–6 sleep cycles of 60–110 minutes. There is a great deal of variability from one person to another in the number and duration of sleep cycles, but the overall pattern is generally consistent in the same individual from night to night. The first third of the night is dominated by SWS, and the last third is comprised mostly of N2 and REM. The sleep histogram in Figure 1.2 shows the distribution of NREM and REM sleep stages and sleep cycles in a young adult. The distribution of SWS in the first portion of the night is related to the duration of the prior waking period (Longer time awake is associated with more SWS). The predominance of REM sleep in the latter third of the night is aligned with the trough of the body temperature cycle, which

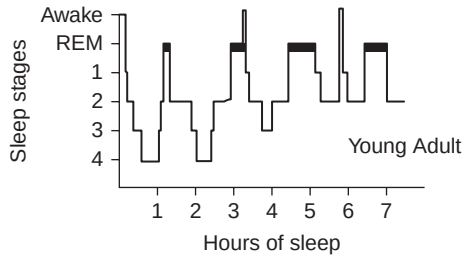

Figure 1.2 ■ *Sleep histogram.* Typical sequence of sleep stages in a young, healthy, adult man. Progression occurs through a sequence of 1–2–3–2 and into REM. *Source: Atlas of Clinical Sleep Medicine* by M. H. Kryger, 2009, Philadelphia: Saunders-Elsevier. Reprinted with permission.

is controlled by the circadian pacemaker. When sleep is restricted to less than 5 or 6 hours per night, a disproportionate amount of REM sleep is lost, creating a REM sleep deficit (Landis, 2005).

PHYSIOLOGICAL CHANGES DURING NREM AND REM SLEEP

Sleep is associated with changes in many physiological processes. Table 1.1 summarizes physiological parameters in NREM and REM sleep. Compared with waking, breathing is regular, but becomes deeper and is characterized by periodic oscillations in amplitude during NREM sleep. Blood pressure, heart rate, cardiac output, urine production, swallowing, esophageal motility, and muscle tension decline. Increase in parasympathetic nervous system activity relative to sympathetic activity leads to decrease in blood pressure, heart rate, and cardiac output. Peripheral blood vessels dilate, and body temperature declines.

During REM sleep, there is a surge of sympathetic activity relative to parasympathetic activity, especially during REMs. This results in increases and irregularity in heart rate and blood pressure, compared with NREM sleep. Regulation of body temperature is suspended during REM sleep.

Respiratory drive and upper airway muscle tone are reduced and lead to increased resistance to inspiratory airflow (Douglas, 2005; Orem & Kubin, 2005) during sleep. During NREM sleep, minute ventilation is reduced, and the arterial partial pressure of carbon dioxide rises. The partial pressure of oxygen falls, but remains within normal limits. Lung secretions are retained, and

Table 1.1 ■ *Physiological Changes During NREM and REM Sleep*

Physiological Variable	NREM	REM
Brain activity and blood flow	Decreases from wakefulness, especially in cerebral cortex.	Increases from NREM, especially in parts of the cerebral cortex, midbrain, and brainstem core.
Heart rate and blood pressure	Decreases from wakefulness.	Increases and highly variable compared to NREM.
Sympathetic nerve activity	Decreases from wakefulness.	Increases from both NREM and REM in some body areas.
Muscle tone	Slightly lower from wakefulness.	Absent.
Respiration	Decreases from wakefulness, displays rhythmic periodicity in breathing amplitude.	Increases and varies from NREM, may show brief stoppages; coughing suppressed.
Airway resistance	Increases from wakefulness.	Increases and varies from wakefulness and NREM.
Body temperature	Regulated at lower level from wakefulness.	Is not regulated, no shivering or sweating.
Swallowing and gastrointestinal motility	Suppressed from wakefulness.	Suppressed from wakefulness.

Source: Sleep Disorders and Sleep Deprivation: An Unmet Public Health Problem (p. 37), by H. R. Colten & B. M. Altevogt (Eds.), Institute of Medicine of the National Academies, 2006, Washington, DC: The National Academies Press.

there are decreased responses to bronchial irritation and decreased cough response.

During REM sleep, loss of muscle tone increases upper respiratory airway resistance. There is reduced responsiveness to changes in partial pressure of carbon dioxide and oxygen. Increased airway resistance is associated with snoring and may be associated with reduced airflow (hypopnea) or complete cessation of breathing (apnea). Hypopneas and apneas result in obstructive sleep apnea (OSA), a form of sleep-disordered breathing (SDB). Signs and symptoms of OSA often occur during REM sleep (see Chapter 8).

NEUROENDOCRINE INFLUENCES ON SLEEP

Secretion of several neuroendocrine hormones is tightly coupled with sleep (Leproult, Spiegel, & Van Cauter, 2009). Sleep is associated with a large surge in the secretion of growth hormone (especially SWS) and an increase in the secretion of prolactin during the latter half of a night of sleep. Secretion of growth hormone is sleep-dependent. Secretion of prolactin is partially sleep-dependent, but also follows a circadian rhythm. An increase in the plasma concentrations of these hormones is delayed until sleep onset, and secretion is reduced or absent with complete sleep loss. Growth hormone is also secreted during afternoon naps.

Sleep inhibits the secretion of corticotrophin stimulating hormone (ACTH) and cortisol, although the daily rhythm of these hormones is controlled primarily by the circadian pacemaker and is independent of sleep. Cortisol reaches its lowest level (trough) during the early part of the sleep period and reaches its highest level (peak) shortly before or after waking up.

The secretion of melatonin rises prior to sleep onset and remains elevated throughout the nocturnal sleep period. Melatonin secretion is controlled by ambient light levels, and the plasma concentration of melatonin rises with onset of darkness. It remains elevated during sleep, even during periods of wakefulness in the dark after sleep onset, but is suppressed by exposure to light (Scheer & Shea, 2009).

NEUROBIOLOGY OF WAKE AND SLEEP

Overview

Knowledge of the neurobiology (neurocircuitry, neurochemistry, cellular physiology) of wake and sleep has advanced considerably in the past few decades (Rosenwasser, 2009). Many decades ago, scientists thought that wake and sleep behaviors were passive processes dependent upon sensory inputs to the nervous system from environmental sources. In the middle of the 20th century, neuroscientists showed that direct nerve stimulation of the brainstem reticular formation, but not sensory pathways, caused an EEG pattern of slow waves in the cerebral cortex of an anesthetized or "sleeping" animal to change to a long-lasting "activated" waking pattern of low amplitude wave forms. Lesions of the brainstem reticular formation and midbrain resulted in a coma-like sleep state with high amplitude EEG slow waves and behavioral immobility. These studies demonstrated that wake and sleep, like any behavior, is regulated by complex and multiple overlapping neural systems.

A brief summary of key elements of the neurobiological systems of wake and sleep is presented. Throughout the discussion, selected examples of altered function in these systems are provided to illustrate potential linkages between the neurobiology of sleep and wake and clinical conditions. Pharmacological agents also affect various components of sleep and wake systems. Examples are provided in Table 1.2. Studies of some of these conditions have enabled better understanding of the physiology and pathophysiology associated with sleep. Research on the neurobiology of sleep and wake behaviors and the regulation of the sleep-wake cycle are active areas of neuroscience research, and new understanding is obtained as new technologies evolve.

Wake Behavior

Wake is associated with a low amplitude and fast frequency cortical EEG pattern (see Figure 1.1) and is maintained by a series of arousal systems. The reticular activating system (RAS) is associated with generalized EEG activation and behavioral

Table 1.2 ■ *Neuropharmacological Agents & Sleep*

Medication	Neurotransmitter/Peptide System Affected
Agents that enhance sleep	
Antihistamines: diphenhydramine, doxylamine	Block Histamine (HA_1) and Acetycholine (Ach) receptors
Antidepressants: Tricyclics Trazadone Mirtazapine	Block Norepinephrine (NE), HA, Ach Also block NE and Serotonin (5HT) reuptake Block 5HT2, NE, HA
Antipsychotics: Olanzapine, quetiapine	Alpha 2 antagonist, blocks serotonin receptor 2A Serotonin, dopamine antagonist
Orexin (Hypocretin) antagonists	Block Orexin 1 and 2 receptors
Benzodiazepines	Enhance GABA A* receptor nonselective complex inhibition
Non-benzodiazepines	Enhance GABA A receptor subunit$_{a1}$ inhibition
Melatonin and melatonin receptor agonists	Melatonin receptor agonist
Antiseizure drugs: valproate and others	Increases GABA by unknown action
Agents that promote wakefulness	
Amphetamines, methamphetamine	Enhance NE, dopamine (DA), 5-HT
Methylphenidate	Enhance NE, DA
Caffeine	Block adenosine 2a receptor

Sources: Stahl, S.M. (2006). *Essential Psychopharmacology: The Prescriber's Guide*. New York: Cambridge University Press. Krystal, A. D. (2009). Sleep Disorders: Neuropharmacology. In C.J. Amlaner and P.M. Fuller (Eds.), *Basics of Sleep Guide*, 2nd Ed., Chapter 9 (pp. 87-98). Westchester, IL: Sleep Research Society.

arousal. Two ascending parallel neural pathways from the brainstem reticular formation produce a waking EEG pattern in the cerebral cortex. One pathway is *nonspecific* and traverses the midbrain and thalamus. It eventually innervates the cortex; the other pathway involves populations of neurons in the lateral and posterior hypothalamus and basal forebrain that are critical for the maintenance of waking EEG pattern (Jones, 2005; Rosenwasser, 2009) (see Figures 1.3A and 1.3B). Lesions of the midbrain and of the posterior hypothalamus in humans lead to severe deficits in wake behavior and to coma.

Various neurotransmitters (glutamate, acetylcholine, noradrenalin, serotonin, dopamine, histamine) are involved in the establishment and maintenance of wake behavior. *Glutamate* is the main excitatory amino acid in the RAS. *Acetylcholine* is a major excitatory neurotransmitter involved in the nonspecific ascending RAS and the hypothalamic-basal forebrain system. When these two systems are active, acetylcholine is released throughout the cortex to generate fast EEG frequency activity of low amplitude. Alzheimer's disease is associated with loss of cholinergic neurons of the basal forebrain and EEG fast activity as well as sleep disturbances. The locus-coeruleus-*noradrenaline* (norepinephrine) system is an important neural pathway that leads to generalized arousal of many brain structures, including the cerebral cortex. There is evidence that this system also enhances activation of the acetylcholine-containing cell groups in the basal forebrain (Jones, 2005).

The raphe serotonergic system in the rostral brainstem has also been implicated in generalized arousal. This system effects both wake and sleep behavior (Imeri & Opp, 2009; Rosenwasser, 2009). Increased release of serotonin promotes waking, but is also necessary for NREM sleep. Serotonin induces the synthesis and release of sleep-promoting factors from the anterior hypothalamus. These sleep promoting factors inhibit brainstem arousal (Imeri & Opp, 2009). Many drugs used in the treatment of depression enhance noradrenaline and serotonin effects in the central nervous system. Sleep disturbance (e.g., insomnia) is a common, undesirable side effect.

Neurons in the substantia nigra and ventral tegmentum contain *dopamine* and are important in the control of motor activity, but also

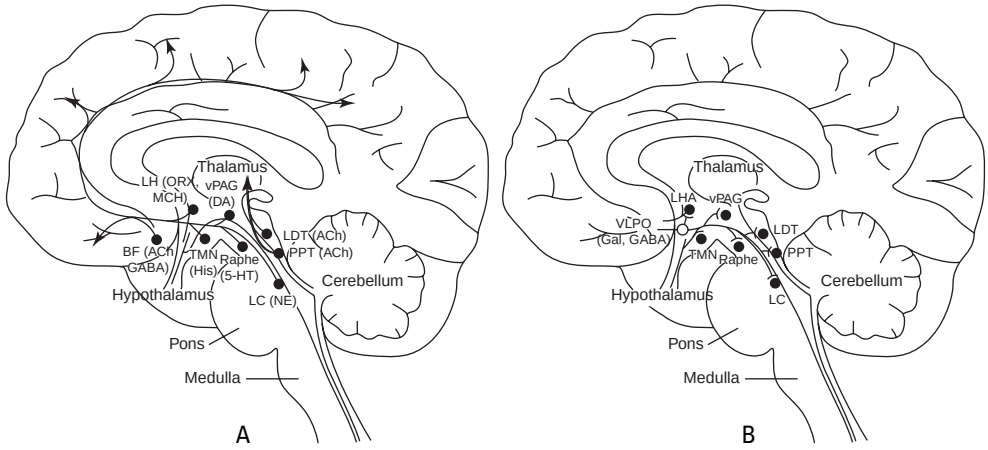

Figure 1.3 ■ *Major arousal and sleep-inducing areas in the brain.* **A.** Ascending arousal systems are more complex compared to the sleep systems. The arousal systems consist of noradrenergic neurons of the locus coerulus (LC), cholinergic neurons in the pedunculopontine and laterodorsal tegmental (PPT/LDT) nuclei in the pons, serotoninergic neurons in the dorsal raphe nucleus (DR), dopaminergic neurons of the ventral periaqueductal gray (vPAG), and histaminergic neurons of the tuberomammillary nucleus (TMN). These systems produce cortical arousal through 2 pathways: a dorsal route through the thalamus and a ventral route through the hypothalamus and basal forebrain. This latter pathway receives inputs from orexin (ORX) and melanin-concentrating hormone (MCH) neurons of the lateral hypothalamic (LH) area as well as from GABAergic and cholinergic neurons of the basal forebrain. **B.** The major sleep-inducing system arises from ventrolateral preoptic neurons (VLPO; open circle), which project to and inhibit the ascending arousal systems shown in A. VLPO neurons and those of the adjacent medial pre-optic area (area not depicted) contain inhibitory neurotransmitters GABA and galanin and are active during sleep. *Source:* "Sleep Architecture, Circadian Regulation and Regulatory Feedback" by P. M. Fuller, J. J. Gooley, and C. B. Saper, 2006, *Journal of Biological Rhythms, 21,* p. 484. Copyright 2006 by Sage Publications. Reprinted with permission.

modulate wake behavior. Dopamine is involved in behavioral hyperactivity and the action of amphetamine-like medications. One branch of the dopamine system enhances activation of the basal forebrain cholinergic system (Jones, 2005; Zeitzer, 2009). Parkinson's disease is associated with degeneration of dopamine neurons in the substantia nigra and with pathological excessive daytime sleepiness.

Histamine neurons in the posterior hypothalamus modulate cortical activation and wake behavior. Similar to noradrenaline and dopamine, histamine enhances activation of acetylcholine neurons. The sedating properties of many over-the-counter medications result from inhibiting acetylcholine and histamine. In contrast, many drugs that stimulate arousal and wake behavior enhance activity in the noradrenergic, dopaminergic or histaminergic systems. New drugs developed for the treatment of memory deficits in patients suspected of having Alzheimer's disease increase cholinergic activity and can lead to unwanted side effects, including insomnia and wandering.

In 1998, scientists discovered orexin (also called hypocretin), a neural peptide, in the lateral hypothalamus. Orexin stimulates EEG activation and waking (Sakurai, 2007). Initially investigators thought that orexin was only important in regulating food intake, but investigators discovered that decreased orexin, loss of orexin neurons, or mutations in orexin receptors were associated with an inability to remain awake and with narcolepsy. Orexin neurons also increase muscle tone and stimulate metabolism in parallel with neuroendocrine systems and the sympathetic nervous system. Collectively, these neurons stabilize the "sleep OFF switch" to inhibit sleep promoting neurons (see Figure 1.4) (Fuller, Gooley, & Saper, 2006). The orexin system may be critical to the maintenance of waking through its connections to cholinergic, histaminergic, and adrenergic neurotransmitter systems and may provide a link between vigilance and energy homeostasis.

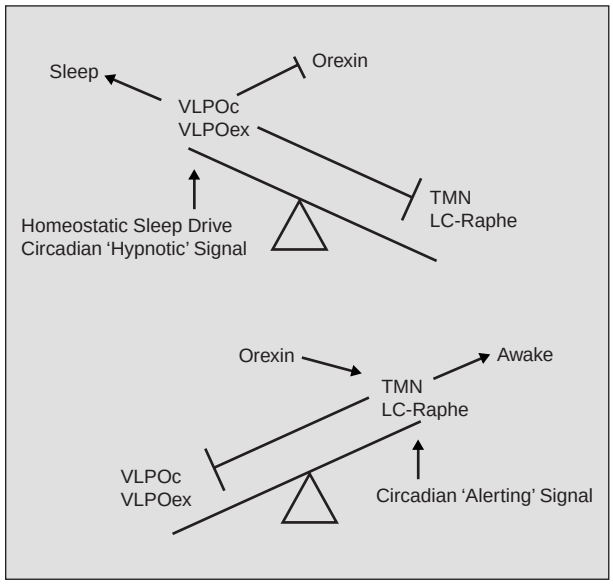

Figure 1.4 ■ *Schematic flip-flop switch model of homeostatic and circadian regulation of sleep and wake.* Neurons of the ventrolateral preoptic core and extended areas (VLPOc, VLPOex) are sleep active, and loss of these neurons produces profound insomnia and sleep fragmentation. These neurons have projections to arousal systems in the tuberomammillary nucleus (TMN), locus coerulus (LC), and the raphe nuclei. The interaction between the VLPO area and these arousal systems is mutually inhibitory, and functions as an electronic flip-flop switch. Homeostatic and circadian processes influence both sides of the switch to produce consolidated sleep and wake bouts. The lateral hypothalamus (orexin neurons) is likely a stabilizer for the switch. *Source:* "Neurobiology of the Sleep-Wake Cycle: Sleep Architecture, Circadian Regulation and Regulatory Feedback" by P. M. Fuller, J. J. Gooley, and C. B. Saper, 2006, *Journal of Biological Rhythms, 21*, p. 485. Copyright 2006 by Sage Publications. Reprinted with permission.

NREM Sleep

Sleep-promoting neurons exist at several levels of the brain: the medulla near the solitary tract nucleus the hypothalamus preoptic area, basal forebrain, thalamus, and cortex. At each level, sleep-promoting neurons are in close proximity to wake-promoting neurons (Fuller et al., 2006; Jones, 2005; Rosenwasser, 2009; Saper, Scammell, & Lu, 2005). The concept of a "sleep ON and OFF switch" is an example of a neural system model. This concept, based on experimental evidence, may explain the ways in which regions at distinct anatomical levels in the brain are involved in NREM and REM sleep onset and maintenance. Neurons in the ventrolateral preoptic region of the anterior hypothalamus contain gamma-aminobutyric-acid (GABA) and galanin and are not active during waking, but become much more active both during NREM and REM sleep. These sleep-promoting neurons are interconnected with regions associated with sleep onset and comprise the "sleep ON switch." Cholinergic neurons of the basal forebrain and brainstem, orexin neurons of the lateral hypothalamus, histaminergic neurons of the posterior hypothalamus, and noradrenergic neurons of the locus coeruleus are inhibited (Fuller et al., 2006; Saper et al., 2005) when the "sleep ON switch" is activated. Many hypnotic medications stimulate GABA receptors and inhibit arousal systems to produce a sleep-like state, but these medications are not thought to *directly* activate the "sleep ON switch."

Neurotransmitters and peptides including adenosine, somatostatin, growth-hormone-releasing hormone, delta-sleep-inducing peptide, prostaglandin D_2, and cytokines have sleep-promoting properties and seem to be responsible for the biochemical control of NREM sleep. Adenosine is a nucleoside released into synapses as a result of cellular metabolism throughout the brain. It is a sleep-inducing substance and potential biomarker of the homeostatic sleep drive because extracellular levels of adenosine rise with sleep loss (Rosenwasser, 2009). Caffeine, a widely used

substance to induce arousal and overcome sleepiness, inhibits adenosine A_2 receptors.

Experimental evidence supports a role IL-I and TNF in the physiological regulation of NREM sleep (Imeri & Opp, 2009). Of the many cytokines, the effects of interleukin-1 (IL-1) and tumor necrosis factor (TNF) have been the most studied. Although IL-1 may stimulate the release of sleep-inducing peptides, including adenosine, growth-hormone-releasing hormone, and prostaglandin D_2, the mechanisms explaining this process remain a mystery. However, cytokines are especially important in mediating sleepiness and lethargy associated with infectious illness. Sleep may be an important determinant of recovery from infection (Imeri & Opp, 2009). Finally, cholecystokinin and bombesin are peptides released by the gastrointestinal tract after food ingestion and may mediate the postprandial induction of sleepiness.

Cortical EEG spindle activity and slow waves are characteristic waveforms of NREM sleep. They require activation of complex neural networks with the release of GABA within the cortex and thalamus (Steriade, McCormick, & Sejnowski, 1993), brain areas rostral to sleep active neurons in the anterior hypothalamus and basal forebrain. Sleep spindles are an easily recognized feature of N2 sleep (see Figure 1.1) and regulate sensory inputs to the cortex during sleep. They also play a role in memory processing (Diekelmann & Born, 2010). Spindle activity and slow waves in the EEG are reduced in middle-aged and older adults at a time when memory functions begin to decline. The reduced number of sleep spindles in women with fibromyalgia is related to persistent pain (Landis, Lentz, Rothermel, Buchwald, & Shaver, 2004) and could also underlie cognitive impairment and "fibrofog" experienced in this disorder (Glass, 2009).

REM Sleep

In the 1950s, a sleep scientist first observed REMs, sign of REM sleep, while he was watching his child sleep. REM sleep is also called "paradoxical," because EEG brain waves resemble a wake-like pattern of low amplitude, fast activity, but the person is *sound asleep*. During REM sleep, powerful descending signals from brainstem neurons inhibit spinal cord neurons and reduce skeletal muscle tone. REM sleep is associated with reports of very vivid and emotional laden dream content. The loss of muscle tone (atonia) in REM may prohibit individuals from acting out dreams.

REM sleep has two distinct phases—tonic and phasic. Tonic REM includes muscle atonia and low amplitude EEG activity. Phasic REM sleep includes REMs, marked irregularity of heart rate and respiration, and brief muscle twitches. In a cat or dog, in addition to REMs, whiskers twitch, ears become flat, and paws move as if the animal is running. In humans, REM behavior disorder is a neurodegenerative condition that involves a loss of descending inhibition of muscle tone and results in bizarre behaviors such as pounding on the bed, pillow, or bed partner, and shouting, while the individual is otherwise asleep.

Neurons located in the brainstem control REM sleep (Siegel, 2006). "REM-on" cholinergic neurons and glutamatergic neurons are found in pontine nuclei. These neurons fire during waking and some fire selectively only during REM sleep but are quiet during NREM sleep. Periodically, these "REM-on" neurons become active while "REM-off" neurons are inhibited (Fort, Gassetti, & Luppi, 2009). Noradrenergic neurons of the locus coeruleus and serotonergic neurons of the raphe nuclei are silent during REM sleep and may play a role in mechanisms controlling REM sleep (Hobson, 2009). There is overlap between the systems that control EEG activation during wake and during REM sleep, but at least two systems associated with generalized arousal (e.g., noreadrenergic and serotonergic) are only active during waking.

REGULATION OF WAKE AND SLEEP

Two-Process Model

Two separate processes regulate sleep-wake (homeostatic and a circadian rhythm) (Borbely &

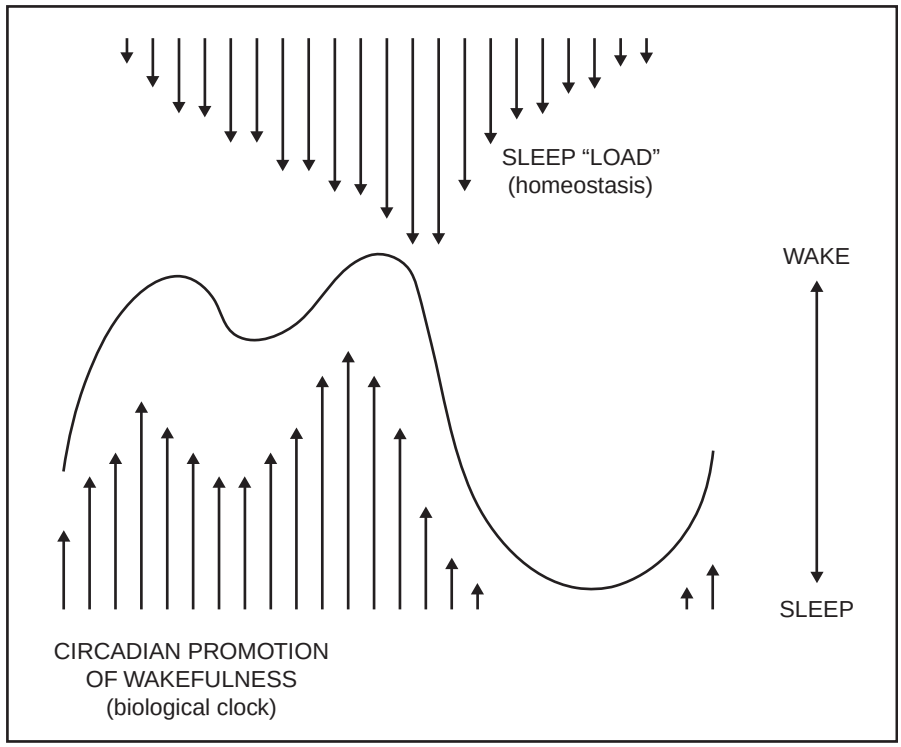

Figure 1.5 ■ *Two-process model of sleep-wake cycle regulation.* At least two variables interact to regulate the sleep-wake cycle and can explain the timing of sleep in humans. A homeostatic signal (down arrows) drives sleep onset. This signal increases the longer a person remains awake. A second signal from the superchiasmatic nucleus (SCN) promotes wakefulness by stimulating arousal systems. The alerting signal from the SCN (up arrows) increases throughout the day to oppose the sleep drive. The tendency to be sleepy during the day is related to the balance of the sleep drive and the alerting signal from the SCN. Just before bedtime the sleep drive and the alerting signal reach maximum levels and both the sleep drive and the alerting signal decrease during sleep. Melatonin, a hormone from the pineal gland, interacts with melatonin receptors in the SCN to reduce the alerting signal. Note that activity from the SCN is quiescent during the early morning hours (arrows missing). When activity in the SCN resumes, the alerting signal acts as an internal alarm clock to signal the end of the sleep period. *Source: Atlas of Clinical Sleep Medicine* (p. 26), by M. H. Kryger, 2009, Philadelphia: Saunders-Elsevier. Reprinted with permission.

Achermann, 2005). A two-process model explains how Process S (sleep history or need) and Process C (circadian timing) interact to account for a typical sleep-wake cycle. Process S reflects sleep need, drives sleep intensity, rises during waking, and declines during sleep. For example, sleep intensity increases when sleep is restricted and decreases in response to excess sleep. Adenosine may be the sleep-promoting substance associated with Process S (Rosenwasser, 2009). Process C reflects circadian timing of sleep and wake in the daily cycle. This timing system opposes the homeostatic sleep drive, enabling people to stay awake and alert throughout the day and asleep during the night, despite a decline in sleep need (see Figure 1.5) (Franken & Dijk, 2009).

Circadian Rhythms

Many biological rhythms of behavior, physiology, and biochemistry fluctuate in an approximate 24-hour time period. These circadian (about a day) rhythms persist when people are placed in isolated environments free of external time cues because the rhythms

are controlled by internal (endogenous) cellular "clock" mechanisms. The suprachiasmatic (SCN) nucleus in the hypothalamus is considered the "master clock" of the body. Specific neurons in the SCN contain a genetically driven clock mechanism that operates on nearly a 24-hour cycle (Scheer & Shea, 2009). This cycle is synchronized to the environmental light and dark periods through light detectors in the retina separate from those involved in vision. Pathways from the SCN innervate sleep-promoting cells in the ventrolateral preoptic area of the anterior hypothalamus and wake-promoting cells of the lateral hypothalamus (Chou et al., 2003) and brainstem. Some of the "clock" genes directly influence sleep intensity and duration independent of their role in regulating the circadian timing of sleep in the daily cycle (Franken & Dijk, 2009). The SCN integrates homeostatic and circadian components of the sleep-wake cycle along with other behaviors (e.g., activity, feeding) and physiological rhythms (hypothalamic-pituitary-adrenal axis) that are normally aligned with sleep and wake behaviors (Saper, Scammell, & Lu, 2005).

Rapid advances are being made in understanding the genetic basis of behavior. The "clock" genes involved in the cellular regulation of circadian rhythms have been well characterized, and this knowledge is being applied to understanding circadian rhythm sleep disorders (Takahashi, Hong, Ko, & McDearmon, 2008) (see Chapter 11). Process C "clock" genes (hPer 2, hPer 3, and Per 2 enzyme genes) are associated with circadian rhythm sleep disorders including advanced and delayed sleep phase syndromes (DSPS) (Zee & Manthena, 2007). Individuals with advanced sleep phase syndrome (ASPD) are called "larks," because they fall asleep early in the evening and wake up early in the morning. Individuals with DSPS are called "night owls" because they prefer to fall asleep late in the evening and wake up late in the morning. Phase refers to the timing of the circadian rhythm of sleep and wake relative to some reference point, such as a clock or light onset in the environment (see Chapter 11).

Genetic Influences on Sleep

The phenotypes representing sleep reflect distinct aspects of sleep behavior, regulation, or intensity (Andretic, Franken, & Tafti, 2008). For example, sleep quantity or habitual duration, latency to sleep onset, amount of slow–wave activity in NREM sleep, and changes in sleep quantity or quality after sleep loss are considered to be distinct behavioral traits potentially influenced by one's genotype. Studies in monozygotic and dizygotic twin pairs have revealed strong evidence to support a genetic basis for sleep. For example, EEG activity in the 8–16 Hz range has a heritability estimated at 96% (Andretic et al., 2008). Investigators were unable to ascertain any differences in EEG activity for any frequency band between monozygotic twin pairs discordant for chronic fatigue syndrome (Armitage et al., 2009). The presence of alpha activity (8–12 Hz) during NREM sleep in several twin pairs suggests a strong overriding genetic influence on the sleep EEG.

Many genes have the potential to affect sleep, and somnologists have yet to agree on a definition of what constitutes a "sleep gene" (Andretic et al., 2008). Nevertheless, studies on the genetic basis of sleep are an active area of basic and clinical research.

METHODS USED TO MEASURE SLEEP

A variety of physiological, behavioral, and self-report methods are available to measure normal sleep. Polysomnography (PSG) is used to simultaneously measure the multiple, simultaneous physiological parameters recorded during sleep. Behavioral measures include observation (video) and actigraphy (body movements). Self-report measures include sleep diaries or logs, questionnaires, and sleep history interview. PSG is the most objective sleep measure, but self-report and behavioral assessments provide important information about behavioral dimensions of sleep. Self-reports provide personal perceptions of sleep quality, sleep habits and history, as well as descriptions of dream experiences. However, they often do not correlate with either PSG or actigraphy (Buysse, Hall, Strollo, et al., 2008). Multiple methods may be simultaneously

used to capture multidimensional aspects of sleep in research and clinical practice. PSG is used together with self-report and behavioral indicators to evaluate sleep quality and architecture; sleep diaries are a necessary component of behavioral assessments with the use of actigraphy.

Polysomnography

PSG is the gold standard for objective assessment of sleep physiology (Carskadon & Rechtschaffen, 2005) and includes the EEG, electro-oculogram (EOG), and the electromyogram (EMG). It is the most accurate way to measure physiological changes in sleep, determine sleep stages, identify frequent brief arousals, and to diagnose particular sleep disorders. Standards define the anatomical placement of electrodes on the head, above and below each eye and on the chin for EEG, EOG, and EMG recordings, respectively (AASM, 2007).

Depending on the purpose of the research or clinical conditions for which people are being screened, PSG includes measurements of nasal and oral airflow, exhaled carbon dioxide, tracheal noise, chest and abdominal movements, bilateral leg movements, pulse oximetry, and electrocardiography. Body temperature may be recorded with a thermistor placed on the skin, in the esophagus or rectum. EEG, EOG, and EMG measurements are used to define sleep stages. Measurements of brief EEG arousals, physiologic cyclic-alternating EEG patterns, various respiratory parameters, and leg movements are used to measure events used in sleep assessment and diagnosis of sleep disorders.

Sleep experts standardized criteria for recording and visual scoring of PSG (EEG, EOG, and EMG) patterns of adults into sleep stages in the late 1960s (Rechtschaffen & Kales, 1968). These standards have also been used in children and adolescents. A separate standardized manual was developed and used for recording and scoring PSG patterns in newborn infants (Anders et al., 1971). The standards for PSG recordings and scoring of sleep stages in adults and children were recently revised (2007). Standard criteria for scoring events during sleep including respiratory events (pauses in breathing [apneas]), brief arousals, and leg movements are also used and periodically undergo revision (AASM, 2007).

In the past, PSG studies were visually scored on print outs from polygraph recordings. Today, most PSG studies utilize commercially available computerized systems for data acquisition, processing, and sleep scoring. Since none of these systems has emerged as a reliable and valid alternative to visual scoring of sleep stages (Carskadon & Rechtschaffen, 2005), highly trained and certified polysomnographic technologists apply standard sleep scoring criteria to the visual, computerized display. PSG is primarily used in clinical or research laboratory settings, but computerized ambulatory PSG systems are available for sleep monitoring in the home and inpatient hospital settings. PSG is still expensive and requires highly skilled technicians to place electrodes, care for the equipment, and score the sleep recordings. There are also many sources of electrical interference present in the home that can distort and interfere with signal quality.

PSG is not practical for the routine assessment of sleep in ill individuals and in hospital settings. Hypoxemia, fever, decreased cerebral perfusion, electrolyte imbalance and the effects of anesthesia, drugs used for sedation and analgesia, and other factors may distort normal sleep EEG signals and make it difficult to apply standard criteria for the scoring of sleep and wake stages (Redeker, 2008).

Behavioral Sleep Measures

Observation

Direct visual observation of sleep behavior is the oldest and most frequently used method to measure sleep. Sleep has characteristic features that are easily recognizable—closed eyes, recumbent posture, relaxed body and facial posture, regular and deep respirations, little movement, and a lack of awareness or interaction with environmental surroundings. Observation is a simple way to measure sleep, but is limited and very time consuming to use in research. Observation is limited to discriminating wakefulness

from sleep. Although it is possible to identify REMs indicative of active REM sleep, the observer must be in close physical proximity to make behavioral assessments, and these must be carried out continuously without disturbing the sleeper. Despite these limitations, behavioral observation is a practical way to measure sleep and has been used to measure sleep in infants (Whitney & Thoman, 1994).

Electronic equipment can be used to observe sleep behavior. Videotape recordings are often used in clinical and research laboratories to evaluate behaviors and neurological events during sleep. Recordings are less intrusive than direct observation and permit better documentation of sleep and wakefulness over time, but require special video recorders and time-consuming scoring of behaviors, and it is difficult to distinguish quiet wakefulness from sleep. Engineers have developed a unique sleep monitoring system that uses data derived from heart rate, video monitoring, and audio to derive assessments of sleep and wake using a laptop computer (Peng, Lin, Sun, & Landis, 2007). This system has undergone preliminary testing and is a model for low-cost, long-term monitoring of sleep in the home.

Body Movements

Pressure-sensitive mattresses are used to monitor body movements as an indicator of sleep states in adults and infants (Hyyppa & Kronholm, 1995; Thoman & Glazier, 1987; Whitney & Thoman, 1994). They can detect respiratory and body movements and generate analog signals that are recorded by battery-powered portable recording units for extended periods of time. Body movement signals are analyzed and scored with computerized procedures (Thoman & Glazier, 1987). However, this provides only an estimate of sleep based on patterns of decreased movement.

The actigraph is a widely used method of assessing sleep. Actigraphs are small wristwatch-like device that measure the frequency and intensity of body movements. These movements are translated into numeric representations. Applied to the nondominant wrist (or leg), actigraphs are used to measure sleep and wake patterns based on assumptions that periods of wakefulness are associated with increased arm movements and periods of sleep are associated with little or no arm movements. The most sensitive actigraphs use accelerometers and omni-directional sensors that are sensitive to motion in all directions. An actigraph can be programmed for continuous activity data collection 24 hours a day for a few days or for weeks at a time depending upon the sampling interval (e.g., 15 seconds, 30 seconds, or 1 minute epochs). Each actigraph has an event marker that is used to indicate removal or replacement of the device, bedtimes, and wake times (see Figure 1.6).

Actigraphs distinguish sleep from wake with very good agreement to PSG in healthy normal subjects (Webster, Kripke, Messin, Mullaney, & Wyborney, 1982) and in patients with sleep disorders (Kushida et al., 2001), but are less accurate in populations with very fragmented sleep or long intervals of nocturnal wakefulness (Paguet, Kawinska, & Carrier, 2007; Van De Water, Holmes, Hurley, 2010). Depending upon the type of actigraph, population, sample, and sleep-scoring algorithm, correlations

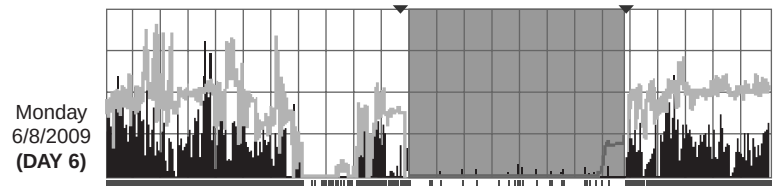

Figure 1.6 ■ *Actigram of normal sleep.* Variables used to derive estimates of sleep period (triangle) include activity counts (black), light levels (light grey), and time in bed (dark grey). Actiware, Philips/Respironics/Minimitter, Co. Figure is courtesy of Dr. Diana Taibi, University of Washington, School of Nursing, Sleep Laboratory, Seattle, WA.

between PSG and activity measurements of sleep in normal subjects range from 0.89 to 0.95 for total sleep time and 0.70 and 0.85 for time awake after sleep onset (Sadeh, Sharkey, & Carskadon, 1994; Webster et al., 1982). A recent systematic review found overall agreement rates between PSG and actigraphy from 72.1% to 96.5% (Van De Water et al., 2010).

Actigraphs tend to overestimate sleep time and less reliably identify wake after sleep onset if individuals lie quietly awake and have minimal arm movements. On the other hand, excessive tremors or abnormal arm movements during sleep could result in underestimation of sleep time. Summary measures of sleep and wake (e.g., total sleep time and wake after sleep onset) derived from actigraphs are reliable, but estimates of time to fall asleep (sleep latency) or final awakening are not (Edinger et al., 2004; Lichstein et al., 2006). Validity is increased when actigraphic data are supplemented with sleep diaries that can help control for artifacts in data collection, for example, removal of the device (Kushida et al., 2001). The reliability of actigraphic scores of sleep and wake increases with recordings of at least 7 days (Van Someren, 2007). This is especially true for patients with insomnia or for older adults with cognitive impairment. Increasing the sensitivity of the algorithm for scoring sleep improves concordance between PSG and actigraphy in elderly women with insomnia (unpublished observations from our laboratory) and in children with arthritis.

Actigraphy is widely used, but poses some methodological challenges. The American Academy of Sleep Medication recently issued practice parameters on the use of actigraphy, including guidelines for its use as an outcome measure in clinical trials of patients with sleep disorders (Morgenthaler et al., 2007). Additional guidelines are available to enhance decision making and reporting of research procedures for use of actigraphy (Berger et al., 2008).

Several different types of actigraphs with different physical properties and distinct scoring algorithms are commercially available. The accuracy of actigraphy compared to PSG is dependent upon the type of device used, sleep-wake algorithm, population studied, and the variables of interest (Van De Water, Holmes, & Hurley, 2010). Therefore, caution must be exercised in interchanging actigraphs from different manufacturers.

Self-Report Sleep Measures

An individual's estimate of sleep quality and duration adds an important perceptual dimension about habitual sleep practices. A wide variety of sleep charts, logs, and questionnaires are available for the assessment of usual or normal sleep patterns (see Chapter 5). These can be self-administered retrospectively to elicit usual habits and history) or prospectively (to describe daily patterns) and rely on an individual's ability to estimate time and report feelings. A sleep history interview is an important method for obtaining information about the nature of usual sleep patterns and sleep complaints (see Chapter 5).

Sleep Diaries

Sleep diaries or logs are used to measure sleep habits in research and clinical practice (Rogers, Caruso, & Aldrich, 1993). Sleep diaries include aspects of the previous night's sleep and events of the day that can influence the subsequent night's sleep (Monk et al., 1994). In the morning, individuals record bedtime, rise time, estimates of the amount of time it took them to fall asleep, the number of awakenings, estimated time awake, and the estimated duration of sleep obtained. In the evening, they record the number and duration of any naps taken during the day, feelings of sleepiness or irritability. Sleep diaries also elicit other information about habits and activities that have a negative impact on sleep, such as life stresses, medications, and the amount of alcohol, caffeine, chocolate, and other stimulants consumed during the day. Diaries can be in tabular or text format and may use Likert or numeric-rating scales and can be used to elicit information over days or weeks. They are used along with actigraphy and with new technologies for recording events directly into handheld computer devices or to Web sites for electronic data capture (see Figure 1.7 and Table 1.3).

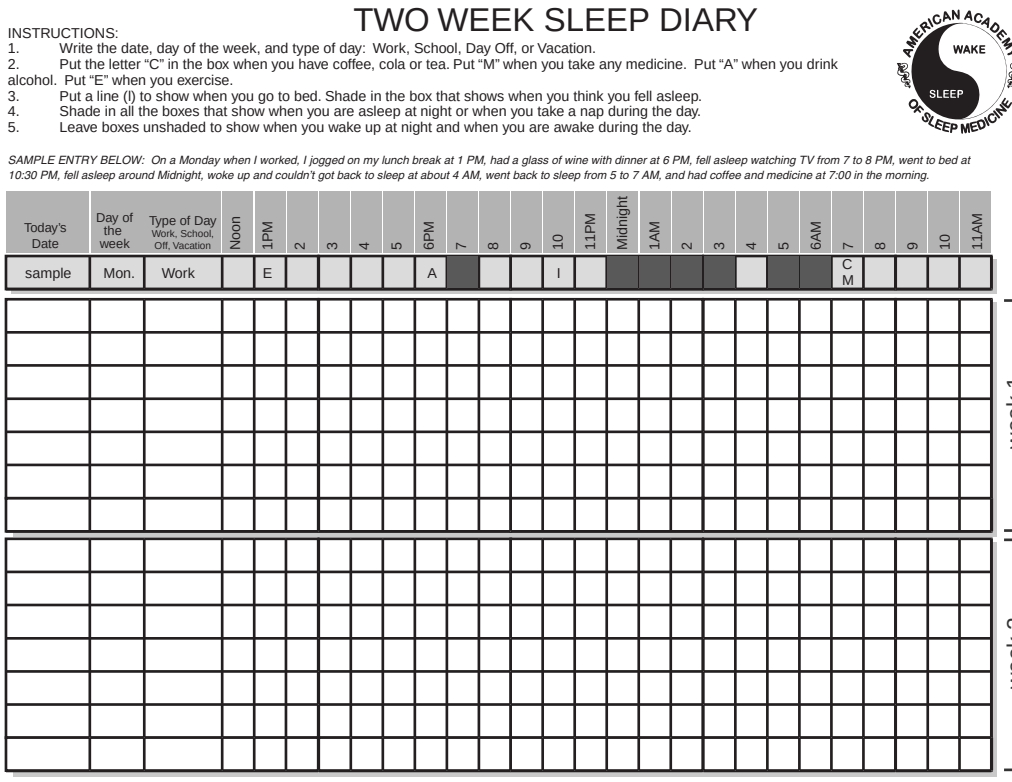

Figure 1.7 ■ Sleep Diary. *Source:* American Academy of Sleep Medicine. Reprinted with permission.

Table 1.3 ■ *Web-Based Resources*

Title and Focus	Web Site
Healthy Sleep Education site with videos, essays on topics of Why Sleep Matters. The science of sleep. Getting the sleep you need.	http://healthysleep.med.harvard.edu/portal/
National Sleep Foundation Nonprofit organization dedicated to improving personal and public sleep health.	http://www.sleepfoundation.org/
Sleep Research Society Professional organization of somnologists who do basic sleep research.	http://www.sleepresearchsociety.org/index.aspx
University of South Australia Centre for Sleep Research Source for applied, basic, and clinical sleep research.	http://www.unisa.edu.au/sleep/research/default.asp
Washington State University: Sleep and Performance Research Center Source for publications on effects of sleep loss on performance.	http://www.spokane.wsu.edu/researchoutreach/Sleep/research.html
American Academy of Sleep Medicine, Sleep Education Web site Source for information for the public and patients about sleep and sleep disorders.	http://www.sleepeducation.com/

Sleep Questionnaires

Various types of sleep questionnaires have been used in practice and research as post-sleep inventories after a night in a sleep laboratory setting or in retrospective surveys of usual sleep habits and quality. Sleep questionnaires have been used to define normative sleep patterns in retrospective studies (Halpern & Verran, 1987; Webb, Bonnet, & Blume, 1976). Actual questions in these instruments vary, but generally items focus on sleep duration and quality. Sleep habits questionnaires have been developed for use with adults (Monk et al., 2003), adolescents (Wolfson et al., 2003), and school-age children (Owens, Spirito, McGuinn, & Nobile, 2000). The Pittsburgh Sleep Quality Index is a questionnaire that is very commonly used in clinical research to measure overall sleep quality (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). In research or clinical practice, it is important to match the particular questionnaire to the specific attributes of sleep and the time frame that is of interest.

SUMMARY

This chapter has focused on an overview of sleep including a description of sleep stages and cycles, physiological changes associated with sleep stages, the neurobiology of wake and sleep onset and maintenance, a homeostatic and circadian rhythm model of sleep and wake regulation, and methods to measure sleep in humans across the lifespan. The emphasis in this chapter has been on the physiology and behavioral aspects of sleep as a foundation for material described in other chapters about sleep disturbances and sleep disorders.

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Developmental Aspects of Normal Sleep

2

Nancy S. Redeker

In many ways, sleep is a marker of human development. There are multiple changes that occur in sleep, a biobehavioral phenomenon, that correspond with chronological and maturational changes over the human lifespan from birth to old age. Physiological changes in circadian and homeostatic sleep systems are manifested in polysomnographic, behavioral, and perceptual characteristics of sleep, as well as rhythms of hormones and other biological substrates, such as melatonin. (See Chapter 1, Physiological and Behavioral Aspects of Sleep.) Cultural and behavioral factors have an important influence on observed sleep patterns. Understanding the changes in patterns of sleep across the lifespan is essential to preventing, diagnosing and treating sleep abnormalities, and providing effective sleep promotion to healthy individuals at all ages and developmental levels. The purpose of this chapter is to describe developmental aspects of sleep occurring from birth to old age. Implications for nursing practice and education are discussed.

Maturational and chronological differences in sleep represent physiological changes at the molecular, cellular, and tissue levels and in virtually all body systems and manifested in physiological, behavioral, and perceptual characteristics of sleep. However, despite the large number of studies designed to document "developmental" or normal changes in sleep, it is often difficult to differentiate "normal" from "abnormal" or "developmental" versus "pathological" changes. At present, incomplete understanding of the associations between sleep characteristics, pathophysiological processes, and psychiatric and medical conditions may result in inaccurate inferences about "normal" sleep due to the absence of rigorous control for these factors in descriptive studies. There have recently been attempts to define normative sleep by defining extreme values in sleep characteristics that are associated with pathophysiological consequences (Ohayon & Vecchierini, 2005). Sleep duration and other sleep characteristics have also changed in the population over time. These have accompanied societal changes, such as the increasingly 24/7 society. For example, children born in the 1980s have shorter sleep duration and more delayed bedtimes than children born in the 1970s (Iglowstein, Jenni, Molinari, & Largo, 2003). Therefore, definitions of "normal" developmental changes versus "abnormal" sleep are likely to change with emerging research findings and societal changes.

Observed variations in sleep between groups or studies may arise from differences in methodological approaches to sleep measurement and the attributes of sleep that are tapped (e.g., self-report, behavioral, or physiological) that make comparison of findings difficult. Even when polysomnographic data are available, methods of measurement may have an impact on measured sleep. For example, studies in the sleep laboratory may be constrained by predetermined "lights out" or "lights on" times or the well-known "first night effect" (sleep affected by the artificial conditions in the laboratory). Self-report measures of sleep or actigraphy often elicit information quite different from that obtained from polysomnographic studies

conducted in the sleep laboratory. These differences likely reflect the multidimensional, biobehavioral aspects of sleep, but each characteristic may contribute useful information. (See Chapter 5, Conducting a Sleep Assessment.)

Normative values, cultural practices, societal roles and expectations, education climate, work patterns, living arrangements, parenting practices, and other sociocultural factors are closely tied to observed sleep patterns, as they are to human development. For example, studies based on self- or family/parents' reports rely on perceptions about "normal" and "abnormal" that often vary among individuals. Cross-cultural studies demonstrate great variability in sleep patterns and differences in views of "normative" sleep (Jenni & O'Connor, 2005).

Most studies of normative sleep are collected from participants during everyday life. However, sleep patterns are subject to exogenous cues in the environment (e.g., lighting, meal times, social cues) (see Chapter 10, Circadian Rhythm Disorders), in contrast to studies of individuals who are living in experimental laboratory settings where such cues are minimized and permit evaluation of "endogenous" (within the individual) circadian rhythms. Thus, the sleep patterns that are detected may reflect these environmental influences and may not truly reflect the physiological or underlying circadian rhythmicity of sleep and wake.

Despite the limitations in available information, there has been exponential growth in understanding changes in sleep associated with human development, and the complex influences on sleep. This information is useful in guiding sleep-promotion strategies, but it must be recognized that sleep exists within a wide range of "normal" and within sociocultural contexts. We discuss dimensions of the biological, behavioral, and perceptual aspects of sleep as they occur across the human lifespan in the following narrative.

NEONATES AND INFANTS

Characteristics of Sleep Patterns

The neonatal period and infancy are associated with dramatic changes in behavioral and physiological sleep patterns. Sleep is an important marker of neurological organization during this time period. Although there is a great deal of variability in individual sleep patterns, normal full-term newborns spend approximately 16–18 hours per day asleep. As they develop over the first few months, more total daily sleep occurs at night. By the end of the first year, infants spend about 50%–55% of the 24-hour period asleep, with most sleep at night and one or two naps during the day (Crabtree & Williams, 2009).

There are two major forms of sleep during infancy: active sleep (associated with frequent movements, phasic eye movements, and irregular respiration); and quiet sleep (similar to NREM sleep with slow, regular breathing patterns and lack of eye or muscle movement). Neonates spend most of the time in active sleep. A third type, indeterminate sleep, cannot be defined by EEG as either quiet or active. Newborn sleep cycles begin with active sleep. About half of the 50–60 minute sleep cycle during the neonatal period is active sleep, with 14% of the time in quiet sleep (Burnham, Goodlin-Jones, Gaylor, & Anders, 2002; Louis, Cannard, Bastuji, & Challamel, 1997). This proportion changes over the first year of life, with increasing proportions of quiet sleep and transition of active sleep into REM sleep. By 12 months, infants spend most of their time in quiet sleep. Both active sleep and indeterminate sleep later become REM sleep during development (Anders, Sadeh, & Appareddy, 1995). By 3 months of age, infants exhibit REM and NREM sleep stages (Markov & Goldman, 2006).

The neonate's sleep consists of polyphasic sleep patterns comprised of 3–4 hour periods of sleep and wake distributed throughout the day and night in 50–60 minute cycles. Newborns begin to demonstrate a circadian rhythm of sleep and have more sleep during the night than the day (Sadeh, Dark, & Vohr, 1996). Although neonates are primarily responsive to cues associated with hunger and satiety (Crabtree & Williams, 2009), they develop a more pronounced circadian rhythm by the second or third month of age, at which time they are more responsive to light–dark cues. Sleep becomes more organized

and consolidated during nighttime hours, especially for 5 months of age (Touchette et al., 2005). Most infants sleep through the night by the age of 6–9 months (Anders et al., 1995), and there is a progressive increase in the duration of the nocturnal sleep period (Burnham et al., 2002; Iglowstein et al., 2003).

Factors Associated With Developmental Sleep Patterns in Neonates and Infants

Although there seems to be a normal physiologic progression of sleep patterns across the neonatal period and infancy, parental, cultural, and ecological factors interact in a bidirectional manner to influence sleep characteristics, such as sleep initiation, continuity, and duration (Anders, 1994). For example, breast or bottle-feeding, rocking or patting the child when they awaken at night or at bedtime, and later bedtimes are associated with shorter and more fragmented sleep (Mindell, Sadeh, Kohyama, & How, 2009; Touchette et al., 2005). This is particularly true in Asian families where infants and toddlers have shorter sleep durations, later bedtimes, and are more likely to share a bed or room with parents (Mindell, Sadeh, Wiegand, How, & Goh, 2010). These parents are more likely to report that their children have poor sleep. Shorter sleep time and more fragmented sleep are associated with parental involvement at bedtime. Only 4% of Asian infants and toddlers, compared with 57% of Caucasians, fall asleep independently (Mindell et al., 2010). Parent involvement at bedtime and during times awake at night is thought to lead to a decrease in the child's ability to self-soothe and fall asleep (or back to sleep) on their own.

Nursing Implications

Sleep is often of great concern to parents of neonates and infants who may report that their normal child "never" sleeps. Because the infant's sleep also has implications for parents' sleep, the sleep of infants is a family concern. Nurses assist parents to understand the meaning of sleep in relation to growth and development and normal variations associated with infant temperament and parental behaviors. Effective sleep promotion strategies include acknowledgement and support of parent perceptions, including cultural variations, regarding infant sleep, as well as assisting them to understand the meaning of changes over time.

TODDLERS AND PRESCHOOLERS

Characteristics of Sleep Patterns

Sleep patterns, including bedtimes, wake times, sleep architecture, and diurnal patterning continue to change from infancy through adolescence. Swedish researchers calculated reference values, including percentiles, from self-report measures of sleep from infancy through adolescence (Iglowstein et al., 2003). These data indicate increase in nighttime sleep until 1 year of age and a subsequent decline through age 16 (see Figure 2.1); overall decrease in total sleep duration; and decrease in daytime sleep duration from birth to age 4 (see Figure 2.2) (Iglowstein et al., 2003). These values are similar to actigraphic and polysomnographic data showing that total daily sleep time and nocturnal sleep duration decrease over the first 3 years of life (Crabtree & Williams, 2009; Iglowstein et al., 2003),with the most dramatic changes between the ages of 3 and 7 years (Montgomery-Downs & Gozal, 2006).

In the United States, bedtimes and sleep times are earliest for children at the age of 12 months and become later over the course of the preschool years. Bedtimes are typically around 8:00 P.M. and rise times at 7:00 A.M. for children at 1 year of age and are typically between 9:00 P.M. and 9:30 P.M. by age 5 (Acebo et al., 2005; Meltzer & Mindell, 2006). However, a large study of infants and toddlers up to 36 months of age (Mindell et al., 2010) found large cross-cultural variability in bedtimes, with an overall 3-hour difference between Asian and Caucasian populations, with later bedtimes in Asian children. Asian children, especially those in Japan, had 101 minutes shorter sleep duration than Western children. Cross-cultural variations in "bedtimes" and patterning of sleep may be associated with parental sleep patterns, climate, or other factors, and these variations are often consistent with cultural practices (Jenni & O'Connor, 2005).

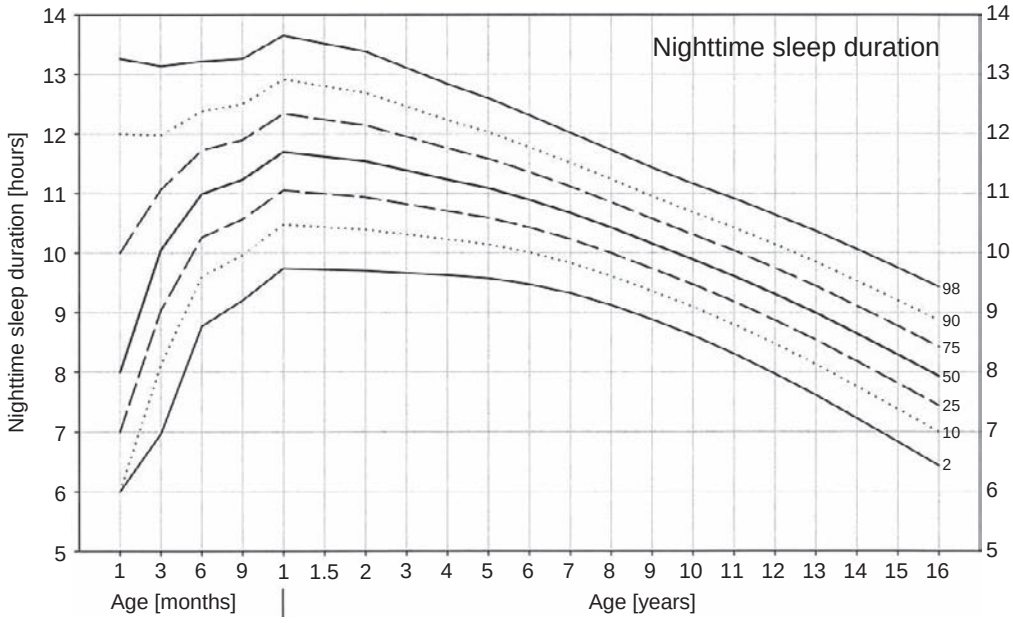

Figure 2.1 ■ Percentiles for nocturnal sleep duration from infancy to adolescence. *Source:* "Sleep Duration From Infancy to Adolescence: Reference Values and Generational Trends," by I. Iglowstein, O. G. Jenni, L. Molinari, and R. H. Largo, 2003, *Pediatrics, 111,* p. 303. Reprinted with permission.

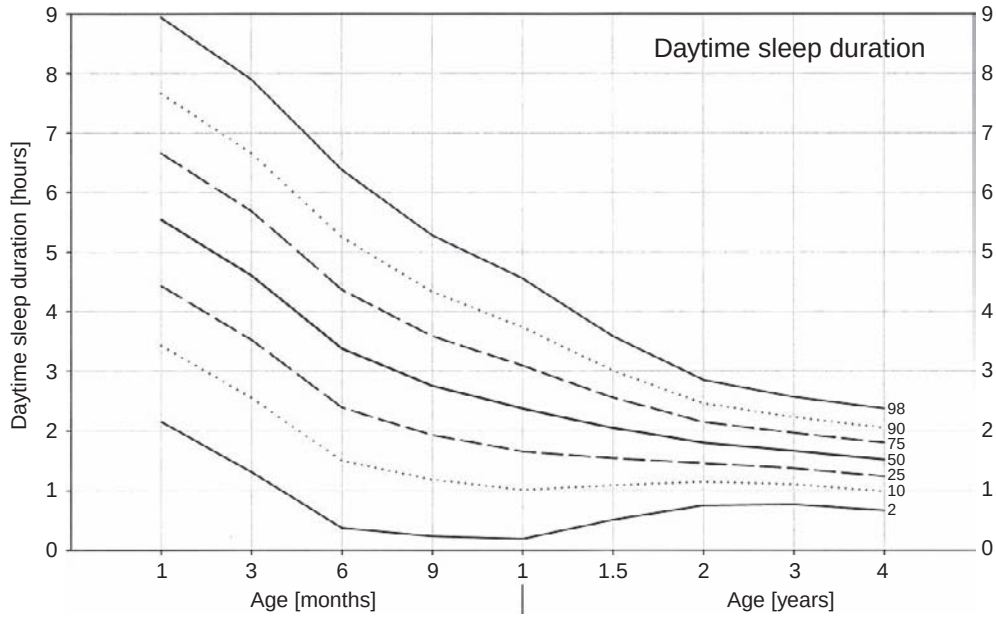

Figure 2.2 ■ Percentiles for daytime sleep duration per 24 hours. *Source:* "Sleep Duration From Infancy to Adolescence: Reference Values and Generational Trends," by I. Iglowstein, O. G. Jenni, L. Molinari, and R. H. Largo, 2003, *Pediatrics, 111,* p. 304. Reprinted with permission.

Earlier and regular bedtimes, as well as bedtime sleep rituals (e.g., bedtime stories, bathing), are associated with longer sleep duration (Mindell, Meltzer, Carskadon, & Chervin, 2009). Such rituals are more common in Western cultures, with the highest rates in the United Kingdom (Mindell, Meltzer et al., 2009). The impact of variations in bedtimes and sleep duration on long-term health is not known.

REM latency and the quantity of REM sleep increase (Montgomery-Downs & Gozal, 2006) until age 5. Compared with adults, young children have large amounts of slow wave and REM sleep.

Although sleep efficiency generally increases between 12 and 24 months (Acebo et al., 2005) and stabilizes at about age 5, toddlers and preschool children may continue to have frequent nighttime awakenings. Parents reported that approximately 10% of children a waken at least once per night and 50% awaken one night per week (Goodlin-Jones, Burnham, Gaylor, & Anders, 2001). More frequent awakenings may be associated with genetic predisposition, temperament or the ability to "self-soothe" and return more easily to sleep.

Napping is virtually universal until approximately 2 years of age, with the largest decrease in napping between 1.5 (94.6% of children) and 4 years of age (35.4% of children) (Iglowstein et al., 2003). Children between the ages of 3 and 5 years who attended full-time daycare slept an average of 76 minutes, and most stopped napping at age 4–5 (Ward, Gay, Anders, Alkon, & Lee, 2008). Ethnic and/or cultural differences may exist, as napping declined more gradually in African American than Caucasian children (Crosby, LeBourgeois, & Harsh, 2005). On the other hand, an international study of almost 30,000 infants and toddlers (Mindell et al., 2010) found few cross-cultural differences in number of naps or duration of daytime sleep, with a decrease from 3.5 to approximately 1 nap per day from birth to 36 months. Children between the ages of 24 and 36 months obtained about 2 hours of sleep during the day. Napping was associated with decreased nocturnal sleep time and increased awakenings in American children who were in daycare, but the causal direction of this relationship or its long-term consequences are not known (Ward et al., 2008). Nevertheless, the remarkable cross-cultural consistency in daytime sleep, despite large differences in nocturnal sleep and bedtimes, suggests that daytime sleep and napping during young childhood may be determined more by physiology than by cultural practices (Mindell et al., 2010).

Factors Associated With Sleep Patterns in Toddlers and Preschoolers

Like sleep during other developmental periods, many factors may influence sleep during the toddler/preschool years. For example, female gender was associated with higher levels of sleep efficiency and more Stage 3 sleep among children aged 3–7 years (Montgomery-Downs, O'Brien, Gulliver, & Gozal, 2006). Lower socioeconomic status is associated with more variable bedtimes and sleep periods, as well as later rising times, longer time in bed, and more time awake at night (Acebo et al., 2005). Increased maternal educational level, a surrogate marker for socioeconomic status, was associated with decreased awakenings, more Stage 4 sleep, and more prolonged REM latency. Family patterns, parental work, daycare, and school schedules also have an important influence on sleep during this developmental period.

Implications for Nursing

Early childhood is a time of rapid neurological and behavioral development, and the development of organized sleep patterns is an important marker of these changes. Nurses facilitate the development of healthy sleep behaviors during this time period. Since this is also a developmental period that may be associated with the development of sleep disorders, such as parasomnias and behavioral sleep issues (see Chapters 13, Pediatric Sleep Disorders, and Chapter 17, Sleep Promotion in Child Health Settings), it is important to support families to differentiate normal from abnormal sleep. Nurses may also work with daycare and preschool programs to assure that children have adequate opportunities for napping and with parents to assure that sleep needs are met.

SCHOOL-AGE CHILDREN

Among school-age children, the circadian pattern of sleep advances so that both bedtimes and wake times are earlier. Although normative values vary, parent reports of sleep duration during the school-age years suggest that U.S. children sleep approximately 9–10 hours (Russo, Bruni, Lucidi, Ferri, & Violani, 2007), with bedtimes just before 10 P.M. Actigraphic data suggest that total sleep times (~500 minutes) decrease in school-age children until about the 6th grade, due to later bedtimes. The increase in sleepiness during the day suggests that the sleep need of children at this age is greater than the sleep time that they obtain (Crabtree & Williams, 2009).

A meta-analytic study of the polysomnographic characteristics of school-age children's sleep showed that total sleep time, percentage of slow-wave sleep (SWS), and REM latency decrease, while Stage N2 sleep and REM sleep increase to a small degree until adolescence and then decline. Sleep latency and sleep efficiency did not change from childhood through adolescence (Guilleminault, Kirisoglu, & Ohayon, 2004) (see Figure 2.3). A study that employed home polysomnography (Quan et al., 2003) showed that total sleep time, sleep efficiency declined between the ages of 6 and 11, but REM sleep also declined.

Factors Associated With Sleep in School-Age Children

Gender, ethnicity, and environmental factors appear to be associated with sleep in school-age children. Female gender was associated with having more Stage N1 sleep in 6–7 year olds, (Montgomery-Downs et al., 2006) but no gender differences were found in sleep architecture in another study (Quan et al., 2003). Hispanic children had less SWS and more Stage N2 sleep than Caucasians. Time spent watching TV or using computers are also associated with shorter sleep duration (Adam, Snell, & Pendry, 2007).

Implications for Nursing

Nurses in pediatric and school settings provide support and education to guide children and their parents toward effective sleep promotion.

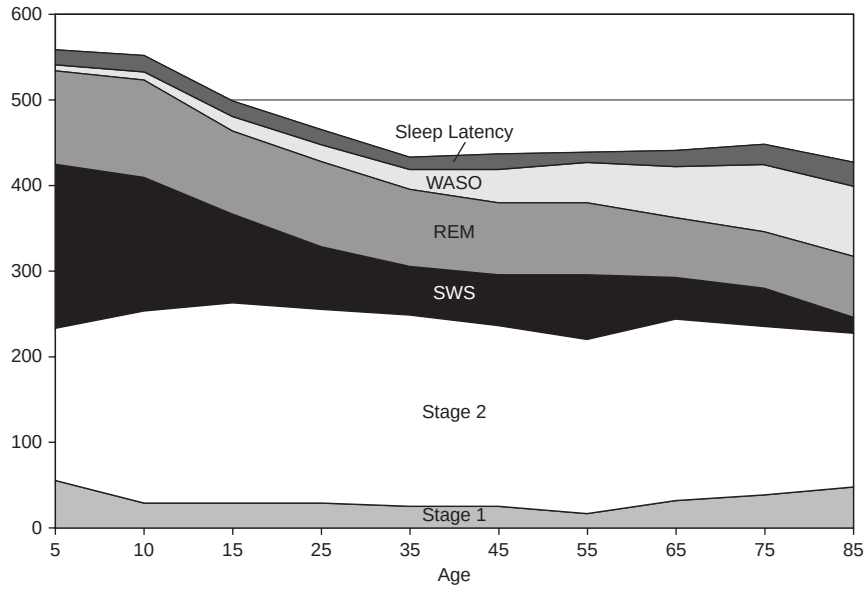

Figure 2.3 ■ Age-related trends for Stage 1 sleep, Stage 2 sleep, slow-wave sleep (SWS), rapid eye movement (REM) sleep, wake after sleep onset (WASO), and sleep latency (in minutes). *Source:* "Meta-Analysis of Quantitative Sleep Parameters From Childhood to Old Age in Healthy Individuals: Developing Normative Sleep Values Across the Human Lifespan," by M. Ohayon, M. A. Carskadon, C. Guilleminault, and M. Vitiello, 2004, *Sleep, 27,* p. 1270. Reprinted with permission.

This includes working with parents and children to manage the demands on children for performance at school and extracurricular activities and limiting the time for watching TV or using computers—all behaviors that reduce sleep time. Good "sleep hygiene" (e.g., bedtime before 9:00 P.M., an established bedtime routine and refraining from caffeine and sleep in bedrooms without television) also has an impact on sleep duration (Mindell, Meltzer et al., 2009). Children should be monitored, for daytime sleepiness, an important indicator of insuffic0ient sleep. There is also a need for educating teachers and children about the importance of sleep (National Sleep Foundation, 2006). (See Chapter 17, Sleep Promotion in Child Health Settings.)

ADOLESCENTS

As children move through their school-age years into their teens, total daily sleep time gradually declines from approximately 10 to 7–8 hours by the end of adolescence (Fredriksen, Rhodes, Reddy, & Way, 2004; Knutson & Lauderdale, 2009), especially on school days. The trend begun in young children for minimal change in weekday wake times, but later bedtimes (Guilleminault et al., 2004) continues during this time period.

Polysomnographic changes in sleep during adolescence include increases in REM sleep relative to earlier in childhood (Guilleminault et al., 2004), decreased latency to REM sleep (time from sleep onset to the onset of REM), and decreases in SWS relative to early childhood. There is little change in sleep latency and efficiency (Crabtree & Williams, 2009).

Circadian changes in sleep timing, or sleep phase delays (see Chapter 10, Circadian Rhythm Disorders), associated with later bedtimes and preferences for later rise times, may be normal in adolescence (Carskadon, Labyak, Acebo, & Seifer, 1999), despite the common perceptions of adults that staying up late into the night and arising late in the morning is volitional. It is common for adolescents to be difficult to arouse early in the morning and to suffer from excessive daytime sleepiness. These circadian changes have been found cross-culturally and in studies of developing animals.

Evidence that gonadal hormones influence maturation of the suprachiasmic nucleus or "circadian pacemaker" (Hagenauer, Perryman, Lee, & Carskadon, 2009) suggests that pubertal status, rather than chronological age is an important contributing factor (National Sleep Foundation, 2006). The patterning of sleep is also related to light stimuli, and excessive exposure to light in the evening, such as that associated with watching TV or using the computer, as well as social activities, may contribute to phase delay (Hagenauer et al., 2009; Randler, 2008).

Although findings about how many hours of sleep adolescents need are inconclusive, sleep experts have suggested that increased daytime sleepiness during adolescence is an indicator of a mismatch between sleep need and sleep obtained, with total sleep time not adequate to address sleep need (Dahl & Carskadon, 1995). The typical 7–8 hours of sleep obtained during weekdays falls short of the estimated sleep need of 9.2 hours (Wolfson, Spaulding, Dandrow, & Baroni, 2007). This is a cross-sectional phenomenon, with similar sleep patterns noted in West German (Randler, 2008) and Korean teenagers (Yang, Kim, Patel, & Lee, 2005) who report similar demands related to social and school-related activities to their U.S. counterparts. However, emerging data suggest that increases in sleepiness during adolescence may also be associated with overall decreases in the frequency of slow wave (delta sleep) between late childhood and early adolescence (Tarokh & Carskadon, 2010). Adolescents may be less responsive to the homeostatic sleep drive (i.e., less likely to fall asleep when sleep deprived) (Randler, 2008). Although this suggests a physiological basis for increased sleepiness, it is likely that a number of factors contribute to increased sleepiness during this developmental period.

Factors Associated With Sleep in Adolescents

Influences on adolescent sleep are multifactorial. Computer and social activities (Knutson & Lauderdale, 2009), paid employment, and homework activities contributed to short sleep among adolescents, but having regular meals and strict rules improved sleep (Adam et al., 2007). Early

school start times play a critical role (Blagrove et al., 1998; Fredriksen et al., 2004; Ohayon, Carskadon, Guilleminault, & Vitiello, 2004). Recent longitudinal data obtained on adolescents' sleep from the summer into the school year revealed that they lost approximately 120 minutes of sleep per weeknight night after the start of school compared with the summer months, and cognitive performance was better in the afternoons (Hansen, Janssen, Schiff, Zee, & Dubocovich, 2005). Delaying school start times from 8:00 to 8:30 A.M. led to a 45-minute increase in average sleep time and increases from 16% to 55% in the percentage of 9th–12th graders who had 8 or more hours of sleep. There were notable improvements in mood, motivation, sleepiness, and class attendance (Owens, Belon, & Moss, 2010).

Implications for Nursing

Nurses who work with adolescents play a critical role in assisting them to obtain adequate sleep and to manage the demands of school, work, family, and social relationships. Obtaining adequate sleep (~9 hours/day) is likely to have an important impact on performance at work and school, as well as injury prevention. Assessment of adolescents for adequate sleep and its negative consequences is critical in clinical as well as school-based settings. Teachers and other education professionals, as well as parents should also be aware of these issues and intervene with adolescents. Given growing evidence of the important role of early school start times, advocacy for "sleep-friendly" policies, such as later school start times is also an important role for nurses.

YOUNG AND MIDDLE-AGED ADULTS

Young adults typically report sleeping approximately 7.5 hours per night on weekdays and 8.5 hours or longer on weekends (Carskadon & Dement, 2005), but nocturnal sleep time decreases linearly by about 10 minutes per decade over the adult lifespan (Ohayon et al., 2004). However, these data contrast with data obtained from the National Sleep Foundation's Sleep in America Poll (2005) that indicates that adults sleep approximately 6.9 hours. Usually, there is one long sleep period, and little or no daytime napping. Both bedtimes and rise times are delayed on nonwork days, compared with work days. The amount of sleep is highly variable, however, and influenced by factors such as genetics, preferences, lifestyle, and environment.

Age-related differences in sleep are more pronounced for polysomnographic, compared to self-report findings (Floyd, Medler, Ager, & Janisse, 2000). REM sleep occupies 20%–25% of the sleep period, and NREM occupies 75%–80% in young adults. SWS (Stage N3) occurs mostly in the first third of the night, and REM occurs predominantly near the end of the night (Carskadon & Dement, 2005). Meta-analysis of polysomnographic recordings reveals large decrease in total sleep time and decrease in sleep efficiency, SWS, and increased wake after sleep onset with aging. There are also decreases in REM and REM latency. Although these changes are statistically significant over the adult lifespan, the changes are incremental with wake after sleep onset increasing approximately 10 minutes each decade from age 30 to 80. Sleep latency also increases, but the increase only differs by less than 10 minutes between young and older adults. Changes over time are not linear, with the largest differences between young and middle, and young and older adults (Ohayon et al., 2004). REM sleep decreased about 0.6% per decade, with no more decrease in the middle of the seventh decade (Floyd, Janisse, Jenuwine, & Ager, 2007).

Napping may be a response to or in anticipation of sleep loss, or used simply for enjoyment (Milner & Cote, 2009). Many people obtain intentional daytime sleep (naps), with rates ranging from 36% to 80% (Dinges, 1989), but more recent survey data suggest that 55% of U.S. adults take, on average, at least one nap during the week. One-third (35%) report that they take two or more naps (2005 Sleep in America Poll). Napping may be especially common and beneficial for night shift workers. Individuals living in "siesta cultures" may have biphasic sleep in which a nap is commonly taken at midday. This may occur in settings where normal work and other activities continue, but napping is considered normal or in settings where work

and business cease during the midday heat. Although globalization and the "24/7" mindset has led to changes in these practices throughout the world, napping persists in some Mediterranean countries, South America, Africa, and Asia (Jenni & O'Connor, 2005). Research is ongoing into the benefits associated with naps (Milner & Cote, 2009).

Factors Associated With Sleep in Young and Middle-Aged Adults

Like the sleep patterns of children, sleep of young and middle-aged adults is closely linked with the environment. The "24/7" nature of society with activities throughout the day and night and access to television, Internet, and other media provides plentiful opportunity to be awake. Additionally, young and middle-aged adults often have numerous work, family, and social relationships that may impact on sleep. For example, one-fourth of adults participating in the 2010 Sleep in American Poll (2005) indicated that their work schedules did not permit them adequate sleep. Those who work night or rotating shifts are particularly at risk for poor sleep. (See Chapter 22, Sleep Promotion in Occupational Health Settings.) Chronic sleep disorders, such as insomnia and sleep-disordered breathing (see Chapter 6, Insomnia and Chapter 8, Sleep-Related Movement Disorders and Parasomnias), as well as chronic psychiatric and medical conditions, such as type 2 diabetes and cardiovascular disease are also more common in middle-aged adults. (See Chapter 11, Sleep in Medical Disorders.) The impact of these conditions on sleep should not be confused with normal developmental changes.

Both gender (see Chapter 3, Gender and Sleep) and race seem to have an impact on developmental aspects of sleep. Over the adult lifespan, women had longer total sleep time, sleep latency, and less Stage N2 sleep and more SWS than age-matched men (Ohayon et al., 2004). However, researchers have often reported that women have more self-reported sleep complaints, and sleep disturbance may be associated with menopause, the perimenopause, and menstrual cycles. Data obtained in the 2010 National Sleep in America Poll showed considerable ethnic and racial differences in the amount of sleep obtained. For example, Black respondents were more likely to report less than 6 hours of sleep on week nights (27%), compared with 12%–17% of Asian, Hispanic, and White respondents. They were also more likely to report more time in bed awake (National Sleep Foundation, 2010). There were differences among various racial/ethnic groups in behaviors performed at bedtime, but the majority reported watching television at bedtime.

Implications for Nursing

As with children and adolescents, nurses play a supportive-educative role regarding promotion of normal sleep patterns in adults. It is common for many adults to minimize the importance of obtaining adequate sleep, despite the impact of having inadequate sleep. They may not perceive the effects of sleep deprivation. Middle adulthood is also the time of peak onset and development of sleep-disordered breathing, and the onset of sleep-related conditions such as hypertension and diabetes. Therefore, distinguishing normal from abnormal sleep is important during this developmental phase. (See Chapter 5, Conducting a Sleep Assessment.) Sleep promotion strategies should be incorporated in occupational health, as well as primary health care settings. (See Chapter 22, Sleep Promotion in Occupational Health Settings and Chapter 18, Sleep and Primary Care of Adults and Older Adults.)

OLDER ADULTS

A common misperception is that poor sleep quality is a normal part of aging. In fact, recent studies have suggested that good sleep is a marker of healthy aging (Driscoll et al., 2008). Polysomnographic studies suggest that total sleep time declines across the adult life-span (Bliwise, 1993; Bliwise, Yesavage, Sink, Widrow, & Dement, 1986; Floyd et al., 2000), but a recent study of healthy older adults living in the community (Ohayon & Vecchierini, 2005) and a large meta-analysis (Ohayon et al., 2004)

suggest little change in the median sleep duration over age groups from 60 to over age 75. Likewise, sleep latency (duration of time taken to fall asleep) increased somewhat in older adults (Floyd et al., 2000), but showed little change in a study of young–old and old–adult adults (Hoch et al., 1994). Sleep continuity generally decreases with aging (Bliwise, 1993; Floyd et al., 2000; Ohayon et al., 2004), but this decrease may be more pronounced in adults over the age of 85 years (Hoch et al., 1994). Data obtained in a recent telephone survey of 1,000 French adults who were 60 years of age and older revealed a median bedtime of 11:00 P.M., with a median wake time of 7:30 A.M. (Ohayon & Vecchierini, 2005).

The sleep of older adults is characterized by a flattened-circadian rhythm, circadian phase advance (earlier bedtime and early morning awakenings), decreased SWS, and more frequent arousals. These changes may be associated with age-related deterioration in the suprachiasmic nucleus (Cajochen, Munch, Knoblauch, Blatter, & Wirz-Justice, 2006). Advanced sleep phase syndrome (involuntary falling asleep early in the evening and awakening very early in the morning with difficulty falling back asleep) (see Chapter 10, Circadian Rhythm Disorders) is also common (Adam et al., 2007). Although sleep experts believe this finding to be a normal developmental pattern, advanced sleep phase syndrome becomes problematic when it interferes with preferred work and leisure-time activities. Light is an important synchronizer of circadian rhythms. While healthy older adults obtain exposure to about 1 hour of daylight daily (Neikrug & Ancoli-Israel, 2010), this stimulus is deficient in institutional settings, such as nursing homes and may contribute to the poor sleep found in these settings. (See Chapter 20, Sleep in Adult Acute and Critical Care Settings.)

There are consistent decreases in REM and SWS associated with aging. However, when only older adults are considered in meta-analytic studies across the lifespan, there seems to be a plateau effect (Floyd et al., 2000), such that there are no additional decreases after the age of about 60. There are also age-related increases in brief electrophysiological arousals across the adult lifespan, with statistically significant differences between healthy sleepers under the age of 20, compared with those of individuals who are 50 years of age and older.

Daytime napping is common among older adults and increases with age. However, the incidence and frequency varies depending on evaluation methods and populations studied. Rates of napping were 10% in adults between the ages of 55 and 64, and 25% among those between the ages of 75 and 84 (Foley et al., 2007). Within a 2-week period of sleep diary collection, 69% of healthy elders over the age of 75 reported napping (Driscoll et al., 2008).

There is considerable controversy about the health-related consequences of napping among older adults. Napping may lead to decreased nocturnal sleep for some individuals, but it may also be an attempt to compensate for nocturnal sleep loss. While several studies have shown that napping is associated with negative health implications, such as depressive symptoms, nocturia (Foley et al., 2007), cognitive impairment, daytime sleepiness, and obesity (Ohayon & Vecchierini, 2005), it is often not possible to determine cause and effect. For example, napping may be a result, rather than a cause of medical or psychiatric morbidity. On the other hand, napping may have beneficial effects (Milner & Cote, 2009). Further research is needed into the cause and effect nature of the relationship between napping and health or sleep quality (Ancoli-Israel & Martin, 2006) and the association of napping with other health-related consequences.

Many people believe that it is normal for older adults to report more sleep disturbances and sleepiness than young and middle adults. However, observed higher rates of sleep disturbance among older adults are associated with treatable sleep disorders, medical, or psychiatric illnesses (Asplund, 2004; Foley, Ancoli-Israel, Britz, & Walsh, 2004; Vitiello et al., 1990; Vitiello, Moe, & Prinz, 2002). The belief that sleep disturbance is a normal part of aging is particularly common among older adults and may prevent them from seeking help with genuine sleep problems.

Factors Associated With Developmental Sleep Patterns in Older Adults

Healthy older women who were 75 years of age and older had more total sleep time, better sleep efficiency, and more SWS than healthy older men (Driscoll et al., 2008). (See Chapter 3, Gender and Sleep.) Retirement, as well as lifestyle changes, and social interactions are often associated with aging and may have an important impact on sleep during this time period.

Implications for Nursing

A critical role for nurses who work with older adults is to dispel the misconception that poor sleep is a normal consequence of healthy aging, to evaluate sleep and assist older adults to seek treatment for symptoms of poor sleep. Nurses can also support older adults to manage changes in daily patterns in sleep that may result from endogenous circadian changes (e.g., phase advance) (see Chapter 10, Circadian Rhythm Disorders) and changes in sleep schedule that may be associated with changes in retirement-related changes in work and sleep schedules.

SUMMARY

Developmental aspects of sleep are manifested in physiological, perceptual, and behavioral aspects of sleep across the human lifespan from birth to old-age. These changes result from physiological changes but are also profoundly influenced by sociocultural factors, such as values, preferences, parenting, and work and family roles. They are also influenced by cultural views about "normal" and "abnormal." Healthy sleep is also a marker of neurological development, health, and well-being. Although there has been exponential growth in identification of "normal" developmental aspects of sleep over time, understanding of the full implications of sleep patterns over the course of human development is in its early stages.

Nurses can play a supportive–educative role with individual clients and their families in helping them to understand their sleep and in promoting normal sleep patterns in the community and across health care settings. Advocacy for policies that support healthy developmental aspects of sleep is needed (see Table 2.1). Nurses must have a thorough understanding of normal developmental variations in human sleep in order to provide sleep-promotion strategies and to assist patients and families to differentiate normal from abnormal sleep and to determine when sleep disorders may be present. Sleep promotion has an important place in health care settings, as well as schools and the workplace (see chapters in Unit III). A key nursing role is clarification of misconceptions about sleep and supporting families and individuals to engage in behaviors that facilitate healthy sleep behaviors. As knowledge grows about normal developmental changes in sleep and the gaps between sleep need and sleep actually obtained, there is a need for advocacy for policy changes at the institutional and governmental levels that support normal sleep. For example, this may include policy changes that support school start times with adolescents' sleep needs or workplace policies that permit napping during breaks for shift workers.

Although many of the observed developmental changes in sleep are physiological in origin, sociocultural factors play an important role

Table 2.1 ■ *Nursing Implications Related to Sleep Promotion*

■ Educate and support individuals and families to recognize normal chronological and maturational variants in sleep
■ Dispel misconceptions about normal and abnormal aspects of sleep
■ Recognize the discrepancy between observed sleep and potential sleep needs
■ Differentiate normal developmental changes in sleep from changes associated with pathology
■ Include understanding of cultural perceptions about sleep into sleep promotion efforts
■ Incorporate sleep promotion strategies in the community, schools, and the workplace, and across health care settings
■ Advocate for institutional and governmental policies that promote healthy developmental aspects of sleep
■ Engage teachers and other professionals who work with children in efforts to promote healthy sleep

and should be incorporated into nursing care. Therefore, incorporating knowledge of cultural variations is an important component of promoting normal sleep.

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3 Gender and Sleep

Pamela A. Minarik

Sex and gender differences occur during normal sleep and at sleep–wake transitions and may influence the prevalence and presentation of sleep disorders across the lifespan. These differences are influenced by neuroanatomy, hormones, and chronobiological and psychosocial factors. (See Chapter 1, Physiological and Behavioral Aspects of Sleep and Chapter 10, Circadian Rhythm Disorders.) Fluctuations in reproductive hormones influence women's sleep during puberty, across menstrual cycles, and throughout pregnancy and menopause (Paul, Turek, & Kryger, 2008). Levels of testosterone change more slowly over the male lifespan, but they also have an influence on sleep. Although understudied, stress, social roles, and other psychosocial issues also influence sleep and may partially explain some of the observed gender differences; and gender differences in sleep or underlying sleep mechanisms may contribute to disorders such as depression that are more prevalent in women. Understanding gender-related differences in sleep and factors that contribute to these differences and their consequences is necessary to guide gender-specific care related to sleep promotion and prevention and management of sleep disorders. The purpose of this chapter is to describe sex and gender differences in normal sleep throughout the lifespan and hormonal changes that affect sleep in women and men that may underlie risk for the development of sleep disorders. In this chapter, gender refers to gender identity within the context of relationships to the society and culture that may evolve over time. The term "sex" is defined by genetic sex, gonadal sex, and phenotypic sex (Paul et al., 2008).

SEX DIFFERENCES IN SLEEP REGULATORY MECHANISMS

Sex and reproductive hormones act on sleep regulatory mechanisms, the circadian system and the homeostatic drive for sleep, to influence both sleep and wake states (Paul et al., 2008; Van Cauter, Leproult, & Plat, 2000). (See Chapter 1, Physiological and Behavioral Aspects of Sleep.) Circadian gene expression influences the two chronotypes, morningness (tendency to prefer morning activity) and eveningness (tendency to prefer evening activity), that determine daily preferences for the timing of activity and rest (Paul et al., 2008). There is evidence that men are more predisposed to a circadian pattern of "eveningness," in which activity occurs during the evening, than are women (Lehnkering & Siegmund, 2007). In animals, sex influences the ability of several circadian genes to regulate sleep and wake, and circadian gene mutations appear to cause sex differences in sleep homeostasis, with females having more time awake (Paul et al., 2008). However, this does not appear to be so for humans.

Central nervous system sexual dimorphism is well documented (Armitage, Baker, & Parry, 2005; Goldstein, Jerram, Abbs, Whitfield-Gabrieli, & Makris, 2010; National Institutes of Health, 2003) and may explain sex-related differences in sleep. Examples of dimorphic structures and processes are circadian clock genes, growth hormone secretion, respiratory control,

the stress response and the HPA axis, the density of hypothalamic nuclei, and sex hormone receptors in the suprachiasmatic nucleus that influence the day–night cycle. These differences have implications for regulation of sleep and circadian rhythms (Armitage et al., 2005).

Researchers believe that sleep homeostasis is regulated differently in women than in men (Paul et al., 2008). Overall, women exhibit slightly more and higher frequency slow-wave sleep (SWS), a higher basal sleep pressure, and less age-related reduction in SWS (Armitage & Hoffmann, 2001). The observation that sleep deprivation increases the sex difference in SWS, with tendency to develop SWS accumulating more quickly in women during prolonged wakefulness than in men, suggests that the size of the sex difference is partially dependent on the magnitude of the stress or degree of sleep deprivation (Armitage & Hoffmann, 2001).

In mammals, reproductive hormones contribute to the sex effects on sleep and influence behavior (Paul et al., 2008). Gonadal hormones organize the stability of circadian rhythms in constant conditions and the ability of circadian rhythms to synchronize with the daily light–dark cycle. Female gonadal hormones promote wakefulness. However, in humans estrogen appears to promote sleep. The influence of exogenous estradiol on REM sleep is sensitive to stress. That is, anxiety or discomfort can interact with the ability of hormone replacement to improve women's sleep (Paul et al., 2008) and reduce its effects on sleep. Although some sex differences in infant sleep–wake patterns have been reported, the specific variables (genetic and gonadal or phenotypic sex) that are responsible are not known (Paul et al., 2008).

Endocrine hormones that stimulate release of other hormones involved in sleep regulation also have differential affects dependent on sex. For example, ghrelin, produced in the stomach lining and the brain, is responsible for hunger. Sleep deprivation results in increased ghrelin. Injection of ghrelin led to a significant increase of Stage N2 sleep, SWS, and non-REM sleep in men, but not in women. After ghrelin injections, cortisol and growth hormone increased and secretion patterns were comparable in both sexes, but sleep was affected in young and elderly men but not in young or postmenopausal women (Kluge et al., 2010). These findings may be due to the higher levels of SWS in women or the impact of female hormones. Further research is needed.

GENDER DIFFERENCES IN NORMAL SLEEP

Until the late 1980s, there was little recognition of gender-bound differences in sleep. Up to 75% of sleep studies included only men (Dijk, 2006; Lee & Kryger, 2008). Studies that included women had small numbers of women or failed to address sex or gender differences (NIH, 2003) and could, therefore, not contribute to understanding the reason for differences in prevalence and symptoms of sleep disorders. As a result, a cadre of sleep scientists, many of whom are nurses, focused their programs of research on exploration of sleep in women. Their work has revolutionized understanding of gender effects on sleep and led to recognition of the influence of women's biology on sleep and the bidirectional impact on women's social roles (Hale, 2010; Lee, Baker, Newton, & Ancoli-Israel, 2008; Lee & Kryger, 2008).

Gender Differences in Sleep Architecture Across the Lifespan

From childhood to early adulthood, sex differences are apparent (Armitage et al., 2005). Ohayon, Carskadon, Guilleminault and Vitello (2004) conducted a meta-analysis of 65 extant studies of quantitative sleep parameters in 3,577 healthy individuals ranging in age from 5 to 102 years. (See Chapter 2, Developmental Aspects of Normal Sleep.) Table 3.1 summarizes the gender differences found in the meta-analysis. Compared to age-matched men, women took longer to fall asleep, had more sleep time, and slept more deeply. Overall, the differences between men and women were modest and associations between sleep and aging were similar for both sexes. Findings regarding the lack of a linear change in REM sleep across the lifespan were supported by a later meta-analysis (Floyd, Janisse,

Table 3.1 ■ *Gender Differences in Normative Sleep Across the Adult Lifespan*

Stage N1 (light sleep) %	■ Increases with age, and these aging effects more important in women.
Stage N2 sleep %	■ Increases with age from 5 to 60 years; women have less percentage of Stage 2 sleep than age-matched men.
Deep (SWS) % sleep	■ Decreases at a rate of about 2% per decade but women have greater percentage of SWS than age-matched men. ■ Age effects are similar in men and women.
REM sleep %	■ Subtle decrease with age from young adulthood to late middle age. ■ Differences between men and women were modest.
REM onset latency	■ Negative association between REM onset latency and age. ■ Aging effects more important in women, although the differences between men and women were modest.
Sleep onset latency	■ Subtle increase with age, but women have longer sleep latency than age-matched men. ■ Differences between women and men were modest.
Wake after Sleep onset (WASO)	■ With aging, more awakenings during night (WASO). ■ Differences between men and women were modest.
Sleep efficiency	■ Decreases with age and aging effects more important in women.
Total sleep time (TST)	■ Women have longer TST than age-matched men. ■ Aging effects more important in women.

Source: "Meta-Analysis of Quantitative Sleep Parameters From Childhood to Old Age in Healthy Individuals: Developing Normative Sleep Values Across the Human Lifespan," by M. M. Ohayon, M. A. Carskadon, C. Guilleminault, and M. V. Vitiello, 2004, *Sleep, 27*(7), pp. 1255–1273.
Abbreviation: SWS, slow-wave sleep.

Jenuwine, & Ager, 2007). Subsequent reports have also confirmed sex differences in sleep duration and sleep architecture. Women had higher sleep efficiency, more SWS, less Stage 1 sleep, and longer REM latency (all $p < .01$) than men, but there were no differences in sleep latency or Stage 2 sleep (Bixler et al., 2009). SWS decreased to a greater extent in men than women with aging. Women were also less responsive to the stress associated with nocturnal blood draws than men. The finding of increased SWS in women was consistent with another study (Walsleben et al., 2004).

Infants

Ongoing research efforts are focused on elucidating sex differences to identify risk for disease. For example, the prevalence of sudden infant death syndrome (SIDS) is higher in infant boys. Maturation of the central nervous system and cortical function may occur more slowly in male infants. Researchers found that 2- to 4- week-old male infants were easier to arouse during quiet sleep than female infants, but at 2–3 months, the age of peak incidence of SIDS, there were no significant gender effects on arousability (Richardson, Walker, & Horne, 2010). The higher risk of SIDS in male infants was not associated with gender differences in cortical arousal. Other studies have shown that infant boys may have less total sleep and more frequent awakenings than infant girls, but the evidence is inconclusive (Armitage et al., 2005). (See Chapter 2, Developmental Aspects of Normal Sleep.)

Children and Adolescents

There are few gender-related sleep differences until after 8 years of age (Armitage et al., 2005). Some studies that matched children by pubertal development documented no significant sex-related differences in sleep, but the results contrasted with later studies (Campbell,

Higgins, Trinidad, Richardson, & Feinberg, 2007; Feinberg & Campbell, 2010; Feinberg, Higgins, Khaw, & Campbell, 2006; Laberge et al., 2001; Lee, McEnany, & Weekes, 1999). Girls between the ages of 11 and 12 tend to wake up earlier on school days and sleep later on weekends than boys (Laberge et al., 2001; Lee et al., 1999). Studies of small samples of early adolescent girls and boys have shown that EEG delta power (slow-wave or deep sleep) begins to decline by 12 years of age and declines more rapidly after that age. Delta power density was lower in girls than boys in middle adolescence (Feinberg & Campbell, 2010).

Reorganization of the neurohormones of the hypothalamic–pituitary–gonadal axis at puberty influences homeostatic and circadian regulation of sleep (Paul et al., 2008). These hormonal fluctuations combined with environmental stress and the onset of many psychiatric illnesses, particularly affective disorders that are more common in girls and women, may contribute to changes in sleep quality during adolescence (Krishnan & Collop, 2006) and may be associated with the development of insomnia.

Much remains to be learned about sex differences in sleep and homeostatic regulation to improve understanding of the role of sleep in normal development, maturation, and adaptation. For example, little is known about how changes in sex hormones during the menstrual cycle influence sleep physiology in adolescents or their relationships with dysphoric mood and dysmenorrhea.

Adults and Older Adults

Women often report more difficulty in initiating sleep than men (Lichstein, Durrence, Riedel, Taylor, & Bush, 2004), although some studies have found no difference in sleep latency (Bixler et al., 2009), and evidence is inconsistent regarding difficulty maintaining sleep and early morning awakening in women. On the other hand, in studies comparing young and middle-aged adults, women reported longer total sleep time (TST) (Lichstein et al., 2004) and the Sleep Heart Health Study (Redline et al., 2004) found poorer sleep in older men compared to younger men. Women had longer sleep time and twice the amount of SWS than men after controlling for age and other covariates such as body mass index (BMI).

Men increased the rate of regular napping as they aged, but women decreased their napping in the middle years and increased it as they became older adults (Lichstein et al., 2004). Overall, greater napping occurred among men. The investigators concluded that in general, age is a more important influence on sleep over the lifespan than gender or ethnicity. (See Chapter 2, Developmental Aspects of Normal Sleep.)

"Successful agers" (healthy older men and women) (Driscoll et al., 2008) aged 75 and older reported high subjective sleep quality. Healthy older women had more TST, greater sleep efficiency, and more SWS than healthy older men. Consistent with other findings (see Chapter 2, Developmental Aspects of Normal Sleep), these studies suggest that aging or gender per se are not determinants of poor sleep in older adults.

Although older women have few age-related changes in SWS and more SWS than age-matched men (Paul et al., 2008), analysis of EEG data using spectral methods (an alternative to the standard method of measuring SWS) revealed less slow-wave activity than older men. This is consistent with EEG patterns of wakefulness and lower rates of release of growth hormone at sleep onset in older women than older men. EEG spectral power findings may explain the female vulnerability to developing insomnia (Vaz Fragoso & Gill, 2007) and may lead to reconsideration of sex differences in SWS.

Sleep–wake homeostasis (slow-wave sleep) is a biological marker of aging in men, but circadian aspects of sleep (sleep timing, sleep consolidation, and distribution of REM sleep) appear to be well preserved until late life. Reduced SWS is partly responsible for reduced growth hormone secretion in middle and late life (Van Cauter et al., 2000). The largest growth hormone secretion occurs during sleep in men, but not in women (Andersen & Tufik, 2008; Dijk, 2006).

SEX HORMONES AND SLEEP

Hormonal Changes and Sleep in Men

The quality of sleep, sleep efficiency, and SWS deteriorate with aging (Dijk, 2006). Andropause, a gradual decline in testosterone production, begins between 45 and 60 years of age (Paul et al., 2008) in men. In contrast to age-related reductions in estrogen secretion in women, the reduction of testosterone secretion is only about 1%–2% annually (Andersen & Tufik, 2008; Paul et al., 2008) and may not be associated with symptoms.

Testosterone release has a diurnal rhythm identified in boys as young as 4–5 years old. In adults, sleep is critical for testosterone regulation, with maximum hormone concentrations during REM sleep in the latter hours of the sleep period and minimum levels during waking (Andersen & Tufik, 2008; Axelsson, Ingre, Akerstedt, & Holmback, 2005). Lower testosterone levels are associated with decreased sleep efficiency, reduced frequency of REM sleep episodes, and altered REM sleep latency. This may explain some of the decrements in sleep quality in older men. Testosterone is a safe and effective replacement therapy for men who are androgen deficient, but some men self-administer massive doses for nonmedical purposes, such as in power sports and body. This can lead to reduction in TST, sleep efficiency, and percentage of non-REM sleep along with an increase in Stage 2 sleep (Andersen & Tufik, 2008). Sleep deprivation and shift work, either alone or in combination with exercise or stress, lead to reductions in circulating androgens, including testosterone, in healthy men (Andersen & Tufik, 2008). Lower morning testosterone levels found in dissatisfied shift workers when compared with satisfied workers were associated with greater sleep need, disturbed sleep-wakefulness, and increased need for recovery after working (Axelsson, Akerstedt, Kecklund, Lindqvist, & Attefors, 2003).

The higher prevalence of sleep apnea in men and its exacerbation by androgen treatment imply a role for testosterone in control of breathing (Andersen & Tufik, 2008; Behan & Wenninger, 2008). Conversely, decrease in testosterone levels that result from obstructive sleep apnea have important implications for sexual and reproductive function (Andersen & Tufik, 2008). Further examination of the relationship between sleep and regulation of testosterone may contribute to better understanding of health problems in men associated with sleep disorders, aging, and shift work (Andersen & Tufik, 2008).

Hormonal Changes and Sleep in Women

Menstrual Cycle

The menstrual cycle is characterized by fluctuations across a 25–35-day period in hormone levels (gonadal steroids, pituitary hormones, melatonin, and cortisol) and core body temperature control; these fluctuations are associated with sleep changes (Krishnan & Collop, 2006; Lee et al., 2008). In women with regular menstrual cycles, estradiol levels increase during the follicular (proliferative) phase and are lowest at the end of the luteal (secretory) phase, while progesterone levels peak in the luteal phase and are low in the follicular phase.

About one-third of menstruating women complain of sleep disturbances during the premenstrual week or menses, but studies with objective measures have not consistently found poor sleep, and a great deal of evidence suggests that sleep onset latency, sleep efficiency, and SWS are fairly stable across the menstrual cycle (Lee et al., 2008). In a recent study, women in the mid-follicular phase compared with women in the mid-luteal phase had elevated nocturnal core body temperature, a reduction in circadian amplitude of core body temperature, and reduced REM sleep with no alteration in SWS, an indicator of sleep homeostasis (Shechter, Varin, & Boivin, 2010). REM latency was also shorter during the luteal, compared to the follicular phase, but there was no difference in the circadian phase of core body temperature or melatonin.

The effects of progesterone on the regulation of breathing have been well characterized. During sleep, upper airway resistance is lower in the luteal phase when progesterone is high

compared to the follicular phase (Behan & Wenninger, 2008). Withdrawal of the effects of progesterone (and estrogen to some extent) during menopause may partially explain the increased prevalence of obstructive sleep apnea after menopause. (See Chapter 7, Sleep-Related Breathing Disorders.) Greater understanding of the role of sex hormones in sleep-related breathing disorders is needed (Behan & Wenninger, 2008).

Overall, sleep has stable characteristics throughout the menstrual cycle in healthy women. Women with clinical problems related to the menstrual cycle, such as dysmenorrhea, premenstrual syndrome, and premenstrual mood disorders, are more likely to experience disturbed sleep (Lee et al., 2008).

Pregnancy and the Postpartum Period

Disrupted sleep is common during pregnancy, evident as early as 11–12 weeks gestation, and continues into the postpartum period (Lee et al., 2008). In healthy women, rapid eye movement sleep remains stable throughout pregnancy, but pregnant women have less time in deep sleep, more time in light sleep, and more awakenings during the night compared with sleep prior to pregnancy (Lee et al., 2008). (See Chapter 16, Sleep Promotion in the Childbearing Family.)

Menopause

Menopause begins between 45 and 55 years of age and is defined by the cessation of the menstrual cycle with decrease in ovarian secretion of estradiol (Paul et al., 2008). Sleep disrupting vasomotor symptoms such as hot flashes and night sweats are the primary complaints of women during the menopausal transition (Lee et al., 2008; Minarik, 2009). However, objective measures of sleep often do not consistently reflect these changes (Minarik, 2009; Young, Rabago, Zgierska, Austin, & Laurel, 2003).

Investigators for the SWAN study of "Women's Health Across the Nation" examined changes in sleep over the menopausal transition (Kravitz et al., 2003; Kravitz et al., 2008). Among 12,603 White, African American, Chinese, Japanese, and Hispanic women between 40 and 55 years of age, 38% reported sleep difficulty that was least prevalent in the premenopausal group (31%) and most prevalent in women with natural or surgical menopause group (46%–48%) (Kravitz et al., 2003). Follow-up of the SWAN cohort revealed that difficulty falling asleep and maintaining asleep increased from the late perimenopause to the postmenopausal period (Kravitz et al., 2008), and these sleep symptoms were associated with menopausal symptoms, bleeding characteristics, and endogenous hormone levels (e.g., ratio of estradiol to testosterone).

The SWAN study also demonstrated associations between White race, higher education, psychological symptoms, perceived stress, poorer self-perceived health, lower quality of life, less physical activity, current smoking, and arthritis and self-reported sleep (Kravitz et al., 2003), as well as association between stable marital status and good sleep (Troxel et al., 2010). Subsequent ancillary analysis (Hall et al., 2009) revealed that self-reported and polysomnographic sleep were significantly worse and lighter in African American women compared with Caucasian and Chinese participants. Race and financial strain, but not educational attainment, were independent correlates of sleep quality, duration, continuity, and architecture.

The Penn Ovarian Aging Study, a longitudinal 8-year study of late reproductive aged women (Pien, Sammel, Freeman, Lin, & DeBlasis, 2008), revealed that hot flashes, depressive symptoms, and lower levels of the reproductive hormone inhibin B were associated with poor sleep quality. A more recent study (Woods & Mitchell, 2010) confirmed the multivariate nature of factors influencing women's self-reported difficulty initiating and maintaining sleep. These factors included multiple symptoms, stress, perceived health, mood, alcohol use, and a history of sexual abuse, among others.

The physiological stress response and thermoregulation are important to midlife women's sleep. Sleep, especially SWS, appears to have an inhibitory influence on the stress system, including the hypothalamic–pituitary–adrenal (HPA) axis; SWS is associated with declining

plasma cortisol levels (Vgontzas & Chrousos, 2002); shorter sleep time and arousal are associated with elevated cortisol levels and activation of the stress system (Vgontzas et al., 1998; Vgontzas & Chrousos, 2002).

Stress is also implicated in the experience of hot flashes during menopause that are associated with autonomic arousal. Studies have shown that EEG arousals occurring prior to hot flashes explain poor sleep rather than the hot flashes per se (Freedman, 2005a; Freedman, Benton, Genik, & Graydon, 2006; Freedman & Roehrs, 2004), but hot flashes were suppressed in the second half of the night, when there is a higher proportion of REM sleep and no thermoregulation (Freedman & Roehrs, 2006). Hot flashes are triggered by small elevations in core body temperature that occur within reduced thermoneutral zones in women with menopausal symptoms (Freedman, 2001, 2005a). Core body temperature is maintained within a wide thermoneutral zone in premenopausal women, but the zone is narrowed in symptomatic postmenopausal women. At the upper limit of the thermoneutral zone, sweating removes heat, and at the lower limit, shivering conserves heat. The more restricted thermoneutral zone may result partially from low estrogen and elevated sympathetic activation (Freedman, 2005b). Stress may affect sleep quality directly and indirectly by narrowing the thermoneutral zone. These findings suggest that the occurrence of sleep disturbance at menopause may not be solely attributable to hot flashes or that amelioration of hot flashes improves sleep (Freedman, 2005b). Stress appears to play an important role. It is possible that behavioral strategies, such as relaxation may widen the zone, reduce hot flashes, and improve sleep (Minarik, 2009).

Taken together, evidence suggests that multiple factors (e.g., stress, depression, other symptoms, marital relationships) may contribute to poor sleep at the menopausal transition. Because midlife is also a time of increased onset of chronic psychiatric (e.g., depression) and medical (e.g., diabetes, heart disease, obesity) conditions, it is especially important to incorporate a comprehensive assessment of these potential influences on sleep in nursing care. Patient education about these potential influences and behavioral strategies to improve sleep are most likely to be helpful. Further research is needed into the factors associated with poor sleep at midlife and effective sleep-promotion strategies.

GENDER DIFFERENCES IN SLEEP DISORDERS AND COMORBID CONDITIONS

There are gender differences in the prevalence and manifestations of sleep disorders. Each of these conditions is described in detail in separate chapters of this volume, but the gender differences are summarized here.

Obstructive sleep apnea has a higher prevalence in men (approximately 2–3 fold) until midlife (Phillips et al., 2008). However, the incidence and prevalence increases in women after menopause. This difference is thought to be associated with the loss of the respiratory stimulant effects of progesterone in women, but may also be associated with weight gain and changes in body fat distribution. There are also gender differences in the presentation of obstructive sleep apnea, with women less likely to report excessive daytime sleepiness and more likely to have insomnia (Phillips et al., 2008), restless legs, depression, nightmares, palpitations, and hallucinations (Valipour et al., 2007). (See Chapter 7, Sleep-Related Breathing Disorders.)

Women with polycystic ovary syndrome (PCOS), in which there is ovarian failure, often obesity, and excessive androgens, are at higher risk for obstructive sleep apnea. PCOS is associated with excessive daytime sleepiness and proinflammatory cytokines (Phillips et al., 2008). Women with PCOS should undergo assessment for the presence of sleep-disordered breathing.

Women complain more about difficulties with initiating and maintaining sleep and are more likely than men to be diagnosed with insomnia (Collop, Adkins, & Phillips, 2004), with one meta-analysis showing that women may have a 41% increase in risk (Zhang & Wing, 2006). Gender-related differences often begin at puberty (Paul et al., 2008). Studies of the extent to which insomnia contributes to

conditions that disproportionately affect women (e.g., anxiety, depression, and fatigue) and the gender-related effects of insomnia treatment are urgently needed. Restless legs syndrome is about twice as common among women as men (Berger et al., 2005). Its incidence and prevalence increase during pregnancy and is more common in women in older age groups (Collop et al., 2004). (See Chapter 8, Sleep-Related Movement Disorders and Parasomnias.)

IMPLICATIONS FOR NURSING

Differences in the gender-related incidence and prevalence of sleep disorders, as well as their presentation, suggest the critical importance of accounting for sex and gender differences in sleep and its consequences in assessing and treatment sleep in a variety of health care settings. Observed gender differences in sleep–wake across the lifespan, especially at critical developmental and transitional phases (e.g., aging, menopause, pregnancy) in women and over the course of the lifespan in men and women, suggest the importance of these critical time points in the trajectory of sleep–wake. Sleep-promotion strategies include teaching about expected changes in sleep, providing anticipatory guidance, and offering sleep hygiene strategies to assure regular patterns of sleep–wake and adequate sleep duration.

Although sleep complaints are often attributed to hot flashes in menopausal women, middle age is also associated with increases in the prevalence of chronic conditions, such as hypertension, depression, and cardiovascular disease, as well as stress related to role demands associated with work and family responsibilities. These stressors may lead to poor sleep. Conversely, sleep deprivation resulting from role overload may lead to higher levels of morbidity. These issues suggest the critical importance of a comprehensive view of sleep that addresses these multifactorial influences.

CONCLUSION

Research on gender and sleep has increased dramatically over the past two decades and revealed physiologically based sex differences in sleep and important information on sleep as a behavior embedded in the social environment. Although over the course of the human lifespan gender differences are modest, differences in presentation and prevalence of sleep disorders and their potential impact suggest the importance of sex- and gender-related factors in sleep promotion and assessment and management of sleep disorders.

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The Nature of Sleep Disorders and Their Impact

4

Nancy S. Redeker and Geoffry Phillips McEnany

"Sleep, like insomnia, is not just about what happens at night: it's about what happens to the day"

(Greene, 2008, p. 48)

At least 50–70 million U.S. adults have difficulty with sleep (Centers for Disease Control and Prevention, 2008). More than a quarter of adults report not getting enough sleep and 10% experience chronic insomnia (http://www.cdc.gov/sleep/). Although the prevalence of sleep disorders is more difficult to quantify in children, experts estimate that approximately 25% of children and adolescents have sleep disturbance at some time during these developmental periods (Owens, 2008).

Evidence accrued over more than 20 years now convincingly documents the associations between sleep loss and decrements in human performance, quality of life, medical and psychiatric morbidity, and mortality. Sleep loss has tremendous societal implications associated with lost productivity, injuries, accidents, and excessive financial costs. The purpose of this chapter is to discuss the nature of sleep loss and sleep disorders and their implications for human health and well-being. The epidemiological and societal consequences of sleep disorders are reviewed, and diagnostic classification systems and their implications for nursing are discussed.

NATURE OF SLEEP LOSS

Sleep loss results from obtaining less sleep than needed (about 7–8 hours in adults) and/or fragmented sleep that leads to deprivation of specific sleep stages (e.g., increased Stages N1 or N2 sleep at the expense of delta sleep) even in the presence of adequate total sleep. Complete sleep deprivation is rare in modern society. However, chronic sleep deprivation (decreased duration and continuity of sleep) is endemic and contributes to pathophysiology, daytime dysfunction, fatigue, sleepiness, morbidity, injury, mortality, and poor quality of life.

Many social, occupational, lifestyle, environmental, and disease-related factors contribute to sleep loss (sleep deprivation) and altered circadian rhythms of sleep-wake, as depicted in Figure 4.1. The contributions of specific sleep disorders and medical and psychiatric conditions to sleep loss are extensively documented in subsequent chapters of this book.

Volitional and nonvolitional social and behavioral factors that contribute to sleep loss include the demands of a 24/7 society in which work, role-related demands, and leisure time activity extend well into the nocturnal hours. Approximately 20% of workers are shift workers who often suffer from sleep loss as well as circadian misalignment (Institute of Medicine [IOM], 2006). Over the past 20 years, there has been a large increase in the percentage of adults sleeping fewer than 6 hours per night (Centers for Disease Control and Prevention, 2005) and concomitant decreases in children's sleep (http://www.sleepdex.org/deficit.htm). The widespread availability of technology and media (e.g., computers,

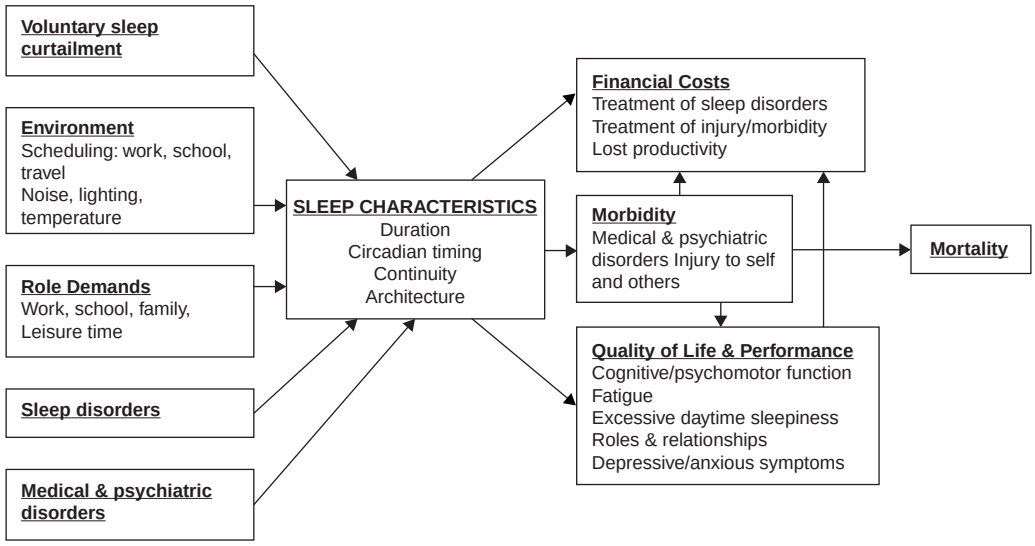

Figure 4.1 ■ Factors that contribute to sleep loss and its consequences.

television) and the perceived need to be productive throughout the 24-hour day contributes in many ways to decreases in total sleep time for both children and adults. (See Chapter 2, Developmental Aspects of Normal Sleep and Chapter 13, Pediatric Sleep Disorders.) Socioeconomic factors, African American race (and other minority status), and living in the inner city are also associated with short sleep duration (Hale & Do, 2007; Patel, 2007). However, the reasons for this are not completely clear. (See Chapter 15, Racial/Ethnic Health Disparities and Sleep Disorders.) Regardless of the cause, sleep loss is an important pathway to negative consequences for health, and many lifestyle factors are amenable to change.

CONSEQUENCES OF SLEEP LOSS

As discussed in Chapter 1, scientists do not fully understand the function of sleep, but there is plentiful evidence of the effects of insufficient sleep. Population-based studies have contributed to explaining the negative consequences of sleep deprivation across a wide range of populations and sleep disorders. The consistency in findings is notable, given the heterogeneity in populations studied, variability of individuals' responses, methods used to measure sleep, and outcomes examined. Less is known about the effects of strategies to decrease the negative consequences of sleep disorders, but there is growing evidence that treatment of some disorders improves selected outcomes. At the community and population levels, there is a need for better communication about the personal and societal risks associated with sleep loss.

Daytime Dysfunction

The most specific consequence of sleep loss is excessive daytime sleepiness. A recent survey revealed that 19.5% of U.S. adults are moderately to excessively sleepy. Nearly 18% reported falling asleep in situations that require a great deal of attention. The highest association was with obstructive sleep apnea, but people with insomnia and sleep duration less than 6 hours and those with depressive disorders were also at risk (http://www.sciencedaily.com/releases/2010/06/100608091852.htm).

Sleep loss also contributes to decrements in cognitive function, including attention, vigilance, memory, and decision making. Many individuals, including some who work in situations requiring high levels of vigilance (e.g., commercial drivers, health care workers), have involuntary micro-sleeps, variable performance,

increased errors, and slow response time (IOM, 2006). Although individual responses to sleep loss are highly variable, the cognitive effects of sleep loss are often subtle and many individuals and their health care providers are not aware that their performance is compromised.

Injury to Self and Others

Sleep loss contributes to excessive risk of injury to the person with sleep disorders and others. For example, obstructive sleep apnea is associated with a three-fold risk in the likelihood of motor vehicle crashes, but this relationship is not dependent on excessive daytime sleepiness (Rodenstein, 2009) and not dose-dependent. It may be secondary to more subtle effects of sleep-disordered breathing on cognitive function. Likewise, insomnia is associated with increased rates of nonmotor vehicle-related accidents (Daley, Morin, LeBlanc, Gregoire, & Savard, 2009). Sleep loss was associated with errors that led to major tragedies, such as the Exxon Valdez oil tanker spill, the nuclear reactor disasters of Chernobyl and Three Mile Island, and commercial and noncommercial motor vehicle crashes (IOM, 2006), among others. Health care workers, including nurses, are particularly vulnerable to the personal health (e.g., medical morbidity, motor vehicle crashes) and work performance (e.g., medication and other errors) consequences of prolonged work hours and shift work (Geiger-Brown & Trinkoff, 2010; Rogers, 2002; Scott et al., 2007). Although the number of hours per week that medical residents are permitted to work is limited by regulations, no such policies exist for nurses.

Morbidity

The public health burden of sleep disorders is enormous. Emerging evidence convincingly documents that sleep loss and sleep disorders contribute to highly prevalent chronic conditions in adults and children, including obesity, diabetes, and impaired glucose tolerance, cardiovascular disease, anxiety, depression mood, and alcohol use (IOM, 2006). The effects of specific sleep disorders and their treatment are explained throughout the chapters of this book.

Mortality

Sleep loss and sleep disorders also contribute to mortality. Several large prospective studies have documented a U-shaped relationship between short and long sleep durations and age-adjusted mortality, after controlling for relevant covariates (IOM, 2006). Less than 5 hours sleep was associated with a 15% increase in mortality risk, but long sleep durations (>9 hours) also confer risk. These findings continue to be replicated in more recent reports. For example, a prospective study of ethnic Chinese in Taiwan revealed a similar curvilinear relationship between sleep duration and all-cause mortality. Insomnia and short sleep duration were associated with deaths from cardiovascular disease (Chien et al., 2010). In a recent review of 23 studies (Gallicchio & Kalesan, 2009), both long and short sleep duration were associated with all-cause and cardiovascular and cancer-related mortality. Limitations to this body of research include frequent reliance on cross-sectional studies and inconsistent control for potential confounding variables. For example, depression and low socioeconomic status were associated with both long sleep duration and mortality (Patel, 2007; Patel, Malhotra, Gottlieb, White, & Hu, 2006).

Quality of Life

Sleep disorders and sleep loss are associated with decrements in multiple dimensions of quality of life. For example, obstructive sleep apnea was associated with vitality, and insomnia was associated with all dimensions of the Medical Outcomes Study. SF-36 in the Sleep Heart Health Study, a population-based study (Baldwin et al., 2001), and similar associations are present in many other studies, for example (McCall et al., 2010; Redeker et al., 2010; Rosen, Palermo, Larkin, & Redline, 2002; Wesstrom, Nilsson, Sundstrom-Poromaa, & Ulfberg, 2008). However, health care professionals have yet to fully recognize the problem.

Costs

Sleep loss and sleep disorders are responsible for billions of dollars of direct and indirect

costs, including the expenses of treatment, diagnostics, the services of health professionals, and hospital services. Indirect costs are attributed to morbidity, disability, injury, accidents, lost productivity, inability to cope with shift work, increased alcohol consumption, and absenteeism (Hossain & Shapiro, 2002), and errors (including medical). An extensive review of information on costs is provided in an Institute of Medicine report (IOM, 2006). Researchers in Quebec (Daley et al., 2009) found that the costs associated with health care consultations, prescription, and over-the-counter medications; the use of alcohol as a sleep aid; work absences; and decreased productivity cost an average of $5,000 Canadian per person among those meeting full criteria for insomnia and $1,400 for people with insomnia symptoms, with 76% of the cost associated with work absences and decreased productivity (Daley et al., 2009).

SLEEP TREATMENT

There is considerable information on the efficacy of treatment on sleep itself (i.e., effects detected with well-controlled, randomized clinical trials in homogeneous samples), much of which is reviewed in subsequent chapter of this book. However, there has been little research on the effectiveness (effects of sleep treatment in heterogeneous patients in usual clinical settings), and improvement in sleep characteristics *does not* necessarily lead to detectable improvements in morbidity, mortality, quality of life, daytime performance, and injury risk. Reasons for this are multifactorial and include the subtle and multidimensional nature of these consequences, as well as lack of consensus on the definition of daytime sleepiness/impairment related to sleep and lack of sensitive and specific methods for measuring these phenomena.

Studies of comparative effectiveness and costs of sleep disorders treatments are urgently needed (Espie, 2009; McDaid et al., 2009; Reeder, Franklin, & Bramley, 2007; Roth, 2009), but these types of analyses are just beginning. For example, CPAP treatment of moderate-to-severe sleep apnea was cost-effective over time, compared with dental devices and placebo (McDaid et al., 2009). Nurse-led CPAP titration in the home was less expensive and had comparable outcomes than traditional laboratory based physician-led treatment in Australia (Antic et al., 2009); in addition, a "stepped care" approach to behavioral insomnia treatment may be more efficient and cost-effective than full multi-session traditional cognitive behavioral therapy. These and other innovations may ultimately lead to improved outcomes and lower costs, but empirical evidence is needed.

Role of Nurses and Other Health Care Providers

Despite the dramatic increases in the science of sleep and sleep disorders, sleep disorders and their consequences remain underdiagnosed and undertreated by many who provide care for adult and pediatric patients (IOM, 2006). Early recognition of sleep-related symptoms is likely to lead to accurate diagnosis, effective treatment, and reduced likelihood of comorbidity—all of which may reduce the indirect and direct costs of sleep disorders. Therefore, there is a need for nurses to be proficient in the use of relevant sleep diagnostic classification systems (i.e., nosologies).

SLEEP DISORDERS DIAGNOSIS

Diagnostic classifications of sleep disorders have evolved concurrently with developments in the scientific basis of sleep and sleep disorders and increased sophistication of sleep disorders practice. There are four sleep disorders classification systems in common use that have evolved from different disciplinary perspectives: the *International Classification of Sleep Disorders, Second Edition (ICSD-2),* the *International Classification of Diseases (ICD),* the *Diagnosis and Statistical Manual of Mental Disorders (DSM-IV-TR),* and the *North Atlantic Nursing Diagnosis (NANDA) Taxonomy*. There are differences as well as significant overlaps between these classification systems that continue to be refined as clinicians acquire experience with their use and the diagnostic accuracy of the criteria is validated.

Because of the broad spectrum of populations and health care settings in which nurses provide care and the virtually universal need to assess and treat sleep in these settings, it is important

for nurses to be conversant with the diagnostic classification systems used in their settings. For example, specialized sleep centers use the *ICSD-2*. However, nurses and other health professionals who work in psychiatric-mental health settings are more likely to use the *Diagnostic and Statistical Manual for Mental Health* (*DSM*).

A significant challenge related to the widespread utilization of sleep disorders nosologies is clinical specialization and discipline-specific education and subcultures within the health care field. However, there has been significant movement toward interdisciplinary, transdisciplinary, and multidisciplinary practice that requires that members of various health care disciplines share terminology to facilitate diagnostic clarity and decision making.

International Classification of Sleep Disorders—Second Edition

The most specific taxonomy for sleep disorders is the *ICSD-2* (American Academy of Sleep Medicine, 2005) used primarily in specialized sleep centers. This diagnostic system (*ICSD-2*) emerged from the 1979 Association of Sleep Disorder's *Diagnostic Classification of Sleep and Arousal Disorders* and the *ICSD-I,* first published in 1990 and updated in 1997 (American Sleep Disorders Association, 1997). The *ICSD* was developed by a process of interdisciplinary consensus among sleep experts. The American Academy of Sleep Medicine commissioned the work that led to development of the *ICSD-2* in 2001, with subsequent open forums held at the meetings of the Associated Professional Sleep Societies (APSS) in later years to obtain consensus. The *ICSD-2* is a working diagnostic system and the nomenclature will continue to evolve as practitioners use it and new information emerges.

The goals of the *ICSD-2* are "to describe all currently recognized sleep and arousal disorders and to base the descriptions on scientific and clinical evidence; to present the sleep and arousal disorders in an overall structure that is rational and scientifically valid; and to render the sleep and arousal disorders as compatible with *ICD-9* and *ICD-10* as possible" (American Academy of Sleep Medicine, 2005, p. xiii). *ICSD-2* addresses both adult and pediatric sleep disorders and is organized into eight major diagnostic categories (see Table 4.1), including more than 90 specific diagnoses. For diagnoses within these broad categories, the reader should refer to the manual (American Academy of Sleep Medicine, 2005). Although the authors preferred to have a common overarching framework, this has not been possible; the long-term goal is to develop such a framework.

Table 4.1 ■ *Sleep Disorders Diagnosis Classifications in the* International Classification of Sleep Disorders, Second Edition (ICSD-2)

I.	Insomnias
	■ Adjustment insomnia
	■ Psychophysiological insomnia
	■ Paradoxical insomnia
	■ Idiopathic insomnia
	■ Insomnia due to mental disorder
II.	Sleep-related breathing disorders
III.	Hypersomnias of central origin not due to a circadian rhythm sleep disorder, sleep-related breathing disorder, or other causes of disturbed nocturnal sleep
IV.	Circadian rhythm sleep disorders
V.	Parasomnias
VI.	Sleep-related movement disorders
VII.	Isolated symptoms, apparently normal variants, and unresolved issues
VIII.	Other sleep disorders

Source: International Classification of Sleep Disorders (2nd ed.) by American Academy of Sleep Medicine, 2005, Westchester, IL: Author.

Each of the major diagnostic categories is based on common complaints, presumed etiology, and organ systems. For each disorder, there is a description of alternate names; essential features; associated features; demographics; predisposing and precipitating factors; familial patterns; onset, course, and complications; pathology and pathophysiology; polysomnographic and other objective findings; diagnostic criteria; clinical and pathological subtypes; unresolved issues and further directions; and differential diagnosis. There is also a brief bibliography in each section.

The authors of the *ICSD-2* anticipate that these codes will ultimately merge with the *ICD* system. However, the *ICD* requires that disorders be classified as organic or nonorganic, and this is often not possible with sleep disorders. The subsequent chapters in this volume use the framework of the *ICSD-2* and present information on the most prevalent examples of the many defined sleep disorders.

International Classification of Diseases

Developed by the World Health Association, the *ICD* is the international standard diagnostic classification for epidemiological and clinical use. It is used to classify diseases and other health problems recorded on many types of health records including death certificates and health records. These records are used for compiling morbidity and mortality statistics by World Health Organization member states, and the codes are used for categorizing disorders and insurance reimbursement in the United States, as designated by the Centers for Medicaid & Medicare Services. The most recent version is the *ICD-10* (World Health Organization, 2010). Sleep disorders are classified under mental and behavioral disorders as nonorganic sleep disorders (Codes: F51.0–F51.9) and under diseases of the nervous system as organic sleep disorders (Codes: G47.0–G47.9) (see Table 4.2). Other categories that are used include "personal history of unhealthy sleep-wake schedule" (Z91.3) and "other behavioral and emotional disorders with onset in childhood and adolescence" (e.g., enuresis and emotional problems) (F98), for example. If the sleep complaint is a symptom of a mental or medical condition and is not an overriding concern, the sleep code should not be used.

Diagnostic and Statistical Manual of Mental Disorders

The *Diagnostic and Statistical Manual of Mental Disorders (DSM)* is used by clinicians

Table 4.2 ■ *Classification of Sleep Disorders in the* International Classification of Diseases and Related Health Problems, Tenth Revision (ICD-10)

Nonorganic sleep disorders (F51)

- Nonorganic insomnia (F51.0)
- Nonorganic hypersomnia (F51.1)
- Nonorganic sleep-wake schedule disorders (F51.2)
- Sleepwalking (F51.3)
- Sleep terrors (F51.4)
- Nightmares (F51.5)
- Other nonorganic sleep disorders (F51.8)
- Nonorganic sleep disorder, unspecified (F51.9)

Organic sleep disorders (G47)

- Disorders of initiating and maintaining sleep (insomnias) (G47.0)
- Disorders of excessive somnolence (hypersomnias) (G47.1)
- Disorders of the sleep-wake schedule (G47.2)
- Sleep apnea (G47.3)
- Other sleep disorders (G47.8)
- Sleep disorder, unspecified (G47.9)

Source: International Statistical Classification of Diseases and Related Health Problems (10th Rev.) by World Health Organization, 2010, Geneva, Switzerland: Author.

and researchers from a variety of disciplines (e.g., psychiatrists, other physicians, advanced practice psychiatric and mental health nurses, other nurses, psychologists, social workers, occupational and rehabilitation therapists, counselors, and other health and mental health professionals) across settings and populations. Experts developed it through a process of consensus. The most recent version is the fourth edition *(DSM-IV-TR)* (American Psychiatric Association, 2000) that was updated in 2000 with additional text to accommodate changes in the ICD. *The DSM-5* is under development.

For each diagnosis, the *DSM-IV-TR* includes criteria, diagnostic features, associated features (health history and physical findings), cultural, age, and gender factors, prevalence, course, and differential diagnosis. There is also a discussion of the relationships of the sleep disorders diagnoses to the *ICSD* nosology (American Psychiatric Association, 2000). In the *DSM-IV-TR,* sleep disorders are categorized by presumed etiology into four categories (primary sleep disorders, sleep disorders related to other medical conditions, sleep disorders related to mental conditions, and substance-induced sleep disorders).

Primary sleep disorders include those that are not explained by another mental disorder, a general medical condition, or a substance and are presumed to arise from endogenous abnormalities in sleep-wake mechanisms. They are categorized as dyssomnias (abnormalities in the amount, quality, or timing of sleep) and parasomnias (abnormal behavioral or physiological events occurring in association with sleep, specific sleep stages, or sleep-wake transitions). Sleep disorders related to another mental disorder and sleep disorders related to another medical condition are those that relate to psychiatric (most commonly anxiety or depressive disorders) and medical conditions, respectively. Substance-induced sleep disorder is presumed to result from concurrent use or recent discontinuation of a substance (e.g., alcohol, tobacco, medications) (American Psychiatric Association, 2000). The *DSM-IV-TR* contains a section describing its relationship to corresponding disorders in the *ICSD* for each sleep disorder. An overview of the major *DSM-IV-TR* sleep disorders categories is presented in Table 4.3.

Sleep-related symptoms (e.g., difficulty initiating or maintaining sleep, hypersomnia, sleeplessness) are embedded within a large

Table 4.3 ■ *Classification of Sleep Disorders in the* Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR)

Primary sleep disorders
- Primary insomnia (307.42)
- Primary hypersomnia (307.44)
- Narcolepsy (347.00)
- Breathing-related sleep disorders (780.57)
- Circadian rhythm sleep disorders (327.xx)
- Dissomnia, not otherwise specified (307.47)

Parasomnias
- Nightmare disorder (307.47)
- Sleep terrors disorder (307.46)
- Sleepwalking disorder (307.46)
- Parasomnia, not otherwise specified (307.47)

Sleep disorders related to another mental disorder
- Insomnia (327.02)
- Hypersomnia (227.15)

Other Sleep Disorders
- Sleep due to a general medical condition (327.xx)
- Substance-induced sleep disorder

Source: Diagnostic and Statistical Manual of Mental Disorders (4th Text Rev. ed.) by American Psychiatric Association, 2000, Arlington, VA: Author.

number of diagnoses (e.g., mood disorders, anxiety disorders, schizophrenia, delirium, substance use disorders). These embedded criteria have utility for accurate psychiatric diagnosis, but their use as diagnostic criteria may result in overlooking important sleep disorders that may be comorbid with psychiatric illness. For example, patients who were ultimately diagnosed with narcolepsy associated daytime sleepiness, hypnagogic hallucinations, cataplexy, sleep paralysis, and disrupted nocturnal sleep received numerous psychiatric or neurological diagnoses prior to correct diagnosis of narcolepsy (Kryger, Walid, & Manfreda, 2002). These findings highlight differences associated with the disciplinary "lens" of the health care provider, as well as the importance of knowledge of sleep nosologies.

A task force is now preparing the fifth edition of the *Diagnostic and Statistical Manual of Psychiatric Disorders*, planned for publication in 2013. It will include significant modification in the sleep diagnostic categories that reflect advances in the field and consistency with the *ICSD-2*. In an effort to engage input from the disciplines that use the nosology and the public, the *DSM-5* Sleep-Wake Disorders Workgroup and Advisors (Reynolds & Redline, 2010) invited members of the sleep community, including nurses, to provide input into the revised criteria. These efforts may lead to dismantling of some of the diagnostic silos between disciplines who provide health care for people with sleep disorders.

North American Nursing Diagnosis Association Classification

The North American Nursing Diagnosis Association (NANDA) focuses on nursing diagnoses that facilitate "clinical judgments about individual, family, or community responses to actual or potential health problems/life processes" (Carpenito-Moyet, 2010). Within the NANDA taxonomy, sleep-related diagnoses are organized under the Functional Health Pattern "Activity-rest." The major sleep-related diagnoses and their definitions are listed in Table 4.4. Nursing diagnoses are not meant to duplicate "collaborative" diagnoses (e.g., sleep apnea), but to describe patient responses for which nurses have accountability. The sleep diagnoses are much broader than those described in the other taxonomies and allow for "diagnosis" of positive aspects of sleep, for example "readiness for enhanced sleep" guides health promotion strategies regarding sleep. Like the other nosologies, NANDA is an evolving framework. As more information is obtained about sleep and nurses' roles in assessing and diagnosing sleep-related concerns, the sleep diagnoses may become further specified.

Nurses may be conversant in one or more of the sleep nosologies, and these should be used to guide differential diagnosis and treatment. The use of particular classifications often depends on the nature of the clinical setting, including the nature of inter-disciplinary

Table 4.4 ■ *Definitions of Sleep Diagnoses from the North American Nursing Diagnosis Association*

Diagnosis	Definition
Readiness for enhanced sleep	A pattern of natural, periodic suspension of consciousness that provides adequate rest, sustains a desired lifestyle, and can be enhanced
Disturbed sleep pattern	State in which a client experiences a change in the quantity or quality of one's rest pattern that causes discomfort or interferes with desired lifestyle
■ Insomnia	The state in which a client reports a persistent pattern of difficulty falling asleep and frequent awakening that disrupts daytime life
■ Sleep deprivation	State in which a client experiences prolonged periods of time without sustained, natural, periodic states of relative unconsciousness

Source: Nursing Diagnosis: Application to Clinical Practice by L. J. Carpenito-Moyet, 2010, Philadelphia: Lippincott.

collaboration. Scope of practice and the need to document diagnoses for reimbursement purposes are also important concerns. Clearly the need for education of nurses in the various nosologies associated with sleep disorders will facilitate more accurate diagnosis and timely intervention.

CONCLUSIONS

There is no doubt that sleep disorders and sleep loss are major public health problems. The chapters in Unit II focus on the specific manifestations of sleep disorders, their consequences, and the implications for nursing. Sleep loss, its consequences, and strategies to prevent and/or treat it are important thematic underpinnings in these narratives. Proficiency in the use of relevant sleep diagnostic classification systems is necessary to interdisciplinary collaboration and sleep diagnosis and treatment. Unit III extends these ideas to provide specific strategies to promote sleep and prevent sleep loss across the many settings where nurses provide care for individuals, families, groups, and communities across the life span and states of health.

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Conducting a Sleep Assessment

5

Teresa M. Ward

INTRODUCTION

Sleep disorders are common in both children and adults. Untreated sleep disturbances pose significant, adverse daytime consequences and place individuals at considerable risk for adverse health outcomes. (See Figure 5.1 and Chapter 4, The Nature of Sleep Disorders and Their Impact.) Unfortunately, sleep disorders are too often undiagnosed. Changes in sleep are associated with specific sleep disorders (e.g., sleep-disordered breathing, restless legs syndrome), comorbid with acute and chronic illness states (e.g., arthritis, asthma, cancer), or manifested during developmental age-related transitions (e.g., childhood, adolescence, older adults). Regardless of etiology, sleep disturbances often present as reports of poor sleep quality, disrupted or fragmented sleep with frequent awakenings, inadequate sleep duration, and/or undesirable timing of sleep episodes throughout the 24-hour day.

Nurses are well positioned to assess and intervene with sleep disorders because they care for people across all cultures, age groups, and health conditions in community and clinical settings. There is widespread agreement among clinical and research experts that sleep assessment should be an integral component of all health assessment, with the specific focus and scope dependent on the situation. The purpose of this chapter is to familiarize nurses with evidence-based approaches to the assessment of sleep. The chapter addresses the basic components of sleep assessment for children and adults, including obtaining sleep-related history, detailed sleep evaluation, and physical examination. The chapter includes information on self-report and objective sleep measurement and resources on sleep assessment for nurses, patients, and their families. Information about assessment in specific settings and in relation to specific disorders is provided in other chapters in this book.

SCOPE OF SLEEP ASSESSMENT

All health care providers, regardless of setting, should ask patients about sleep and wakefulness during routine health assessments and encounters related to ongoing treatment or changes in health status or stressors (e.g., new onset of a medical or psychiatric condition, change in medication that may influence sleep, birth of a baby, acute hospitalization). However, health professionals, including nurses, often fail to ask patients about their sleep. This may lead to unnecessary morbidity and mortality, as well as health-related liabilities associated with sleep disorders.

The scope and comprehensiveness of the sleep assessment and the specific questions asked vary according to the setting and health status or developmental stage of the individual, the prevalence of specific sleep disorders in the specific target group, level of specialization, and scope of practice of the nurse, and priorities of care within the specific health care setting. For example, developmental issues around sleep (as well as assessment for sleep disorders) should be an integral component of a well-child assessment, but the priority for a child or adult undergoing anesthesia would be assessment for sleep apnea due to the dangers of respiratory depression in patients with this condition

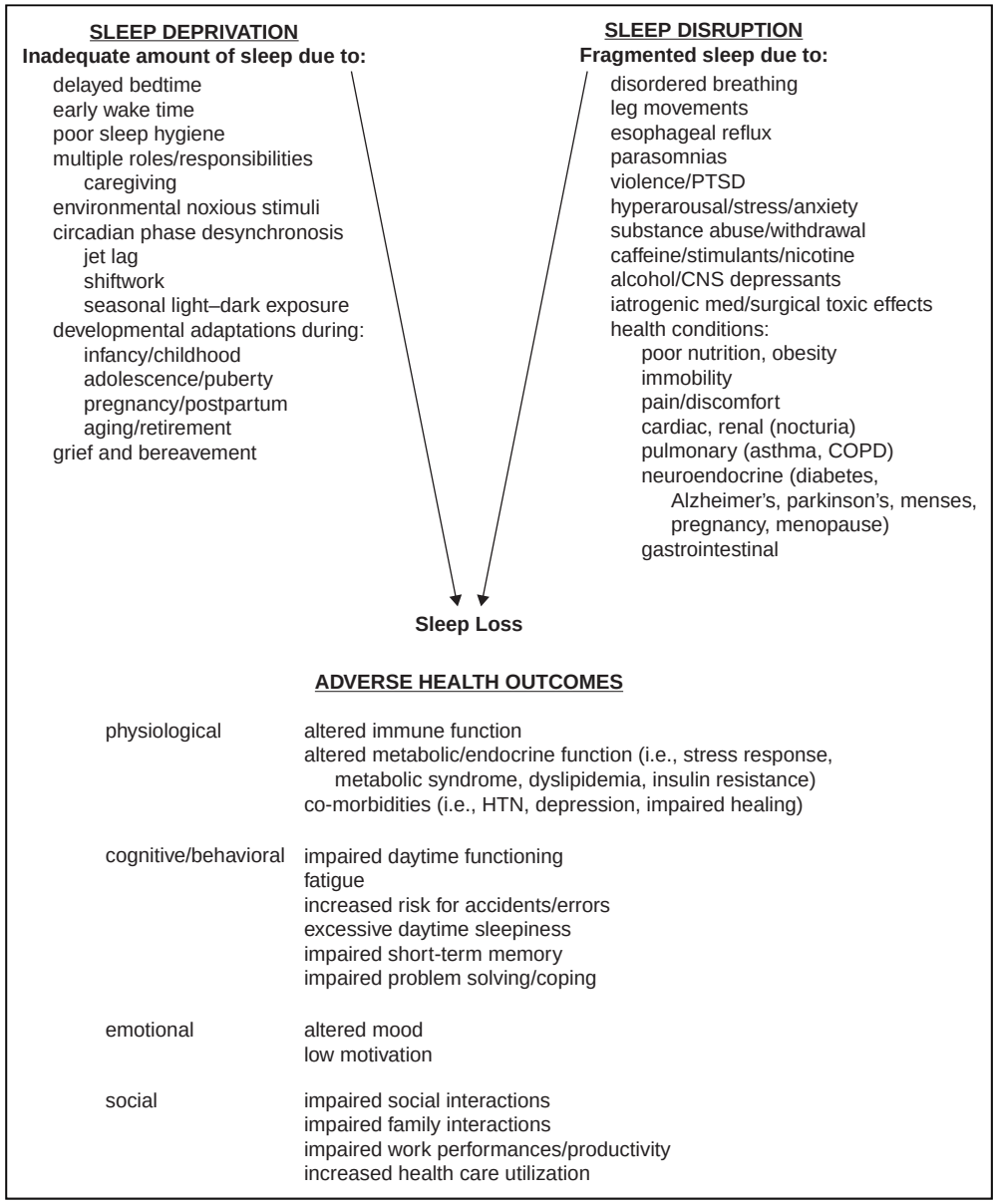

Figure 5.1 ■ Conceptual model of impaired sleep. *Abbreviations:* CNS, central nervous system; COPD, chronic obstructive aulmonary disease; HTN, hypertension; PTSD, posttraumatic stress disorder. *Source*: "Sleep and Chronobiology: Recommendations for Nursing Education," by Lee, K. A., Landis, C., Chasens, E. R., Dowling, G., Merritt, S., Parker, K. P., et al., 2004, *Nursing Outlook, 52,* pp. 126–133. Reprinted with permission.

who undergo anesthesia. (See Chapter 7, Sleep-Related Breathing Disorders and Chapter 20, Sleep in Adult Acute and Critical Care Settings.) A critical barrier to sleep assessment is lack of time within a typical patient encounter. Incorporation of a structured approach, as described in this chapter, is likely to facilitate incorporation of screening and assessment into routine patient care encounters.

The multidimensional nature of sleep disorders and their impact on a wide range of health-related problems necessitate a multidisciplinary

approach, including staff nurses, advanced practice nurses, neurologists, adult and pediatric primary care providers, geriatricians, psychologists, psychiatrists, pulmonologists, respiratory therapists, otolaryngologists, and dentists, depending on the population, setting, and nature of the sleep problem. Each brings a unique perspective to the problem.

Depending on the setting, the nurses' role, and the complexity of the sleep symptoms, staff nurses or advanced practice nurses may initiate the sleep assessment and then treat the sleep condition; refer patients to sleep specialists when more in-depth evaluation and treatment are needed; provide patient education regarding what to expect in a specialized sleep study; or be responsible for the care of patients after they have undergone specialized sleep evaluation and treatment. In the latter case, monitoring of efficacy and supporting adherence to treatment are important nursing roles. Specific aspects of sleep assessment, treatment, follow-up, and the need for referral for specialized treatment in various health care settings are discussed in Unit III. This chapter provides an overview of the basic components of sleep assessment, with the expectation that the approach to sleep will be modified in individual settings.

Challenges Associated With Sleep Assessment

The clinical evaluation of sleep and sleep disorders is challenging for a number of reasons, including patient and provider perceptions, knowledge, and the complexity of the interaction of sleep and sleep disorders with comorbid psychiatric and medical conditions. Due to the gradual onset of most sleep disorders, patients may become accustomed to abnormal sleep and may not seek assessment, a problem compounded by the failure of many health professionals to routinely initiate sleep assessment. Patients and health care providers, including nurses, may attribute the daytime effects of sleep disorders (e.g., daytime sleepiness, altered mood, inattention, and hyperactivity in children) to other causes. For example, older adults may view poor sleep as a normal consequence of aging. (See Chapter 2, Developmental Aspects of Normal Sleep.)

Assessment of sleep disorders that are related to medical or psychiatric conditions is complex due to the bidirectional relationship between these conditions and sleep (e.g., depression leads to poor sleep or poor sleep leads to depression). It is often not possible to determine cause and effect relationships and therefore both the sleep disturbance and medical or psychiatric conditions must be simultaneously addressed. (See Chapter 11, Sleep in Medical Disorders, and Chapter 12, Sleep and Psychiatric Disorders.)

COMPONENTS OF A CLINICAL SLEEP ASSESSMENT

Sleep assessment includes a thorough sleep history, addressing quantitative and subjective characteristics of sleep and sleep disturbances; related factors and consequences (e.g., mood, fatigue, excessive daytime sleepiness); a thorough medical, psychiatric, developmental, and social health history; medication history; physical examination; laboratory testing where indicated; and depending on the nature of the suspected sleep disorder, specialized sleep testing in a sleep laboratory setting. Specific sleep assessment methods used in clinical practice include sleep diaries, questionnaires, and objective measures (e.g., wrist actigraphy, polysomnography) of specific sleep characteristics. The impact of sleep disorders (e.g., sleepiness, cognitive function, and mood) should also be assessed.

Sleep History

A simple and useful guide to organize sleep assessment is the BEARS framework (Owens & Dalzell, 2005). The mnemonic "BEARS" refers to five domains of sleep: ***B***edtime problems, ***E***xcessive sleepiness, ***A***wakenings, ***R***egularity of sleep, and **S**leep-disordered breathing. BEARS questioning is relevant for infants, toddlers, preschool and school-age children, adolescents, and adults (see Table 5.1). Additional information on the approach to sleep assessment is given in Table 5.2. Questions in the BEARS screening tool can be completed by the health care provider, patient, parent, or child with

Table 5.1 ■ *"BEARS" Sleep Assessment*

B = Bedtime problems

- Do you have problems going to bed at night?
- Problems falling asleep at bedtime?
- How long does it take you to fall asleep after turning the lights out?
- Any problems at bedtime?

Note: Address sleep environment such as temperature, blinds, TV, or computer in the room or shares bedroom with siblings; resists going to bed, requires parental presence to fall asleep; consider mood or anxiety disorders; and use of caffeine or other stimulants.

E = Excessive daytime sleepiness

- Are you sleepy during the day? At school? At work? While driving?
- Do you have difficulty waking up in the morning?
- Do you fall asleep in school or at work? If so, how frequently? For how long? What activities are you doing when you fall asleep?
- Have you ever been in an accident because you fell asleep?
- How many servings of caffeinated food or beverages do you consume daily?

Note: Coworkers, spouses, teachers, or caregivers may have concerns regarding daytime sleepiness, behavior, and changes in school or work performance. The Epworth Sleepiness Scale is useful in adults as a measure of sleepiness.

A = Awakenings during the night

- Do you awaken during the night?
  - If yes, what awakens you?
  - How often?
  - How long are the awakenings?
  - Do you have trouble falling back to sleep?
- Do you have nightmares? Sleep terrors? Anxiety?

Note: Address behaviors during middle of the night awakenings. Consider medical and psychiatric conditions; medications; developmental transitions; menopausal transition; environmental factors; and caffeine consumption.

R = Regularity of sleep and duration

- How long is your typical nighttime sleep?
- What time do you go to bed? Wake up?
- What are these schedules on the weekend?
- Do these schedules vary according to work or school schedules?
- Do you work shifts? Which shifts? How often do you rotate?
- Nap patterns?
  - Where: Home? Child care? School? After school?
  - How often? Duration?

Note: Address consistency of bedtime schedules, extracurricular activities that may interfere with sleep schedule such as early swim practice or evening dance and music lessons, and shift work.

S = Sleep-disordered breathing

- Has anyone told you that you snore or breathe loudly while asleep?
- Do you have pauses in your breathing during sleep? Do you wake up choking?
- Do you have morning headaches?
- Are you restless or do you sweat during sleep?

Note: It may be necessary to obtain this information from bed partners or family members (e.g., parents of young children). Address medical conditions that may be related to sleep apnea (diabetes, obesity, hypertension, heart failure, ischemic heart disease, asthma); address growth and weight patterns in children (e.g., failure to thrive, obesity).

Source: Adapted from "Use of the 'BEARS' Sleep Screening Tool in a Pediatric Residents' Continuity Clinic: A Pilot Study" (Appendix A), by J. A. Owens and V. Dalzell, 2005, *Sleep Medicine, 6*, pp. 63–69.

Table 5.2 ■ *Critical Points of Inquiry in Sleep and Medical History*

General questions to begin the inquiry

- Review sleep habits including sleep schedule on weekdays and weekends, cosleeping, sleeping with pets, and sleep disruption because of a bed partner/pet. Sleep diary may be helpful in gathering this information.
- Gather information regarding consistency of bedtime routine including evening activities, feedings/eating prior to going to sleep, difficulty falling asleep, trouble staying asleep, and waking up too early.
- Inquire about the bedroom environment including questions about the temperature, comfortable bed, blinds and curtains on the window, and dark, quiet bedroom.
- Assess nocturnal behaviors including frequency of awakenings, uncomfortable or painful sensation in the legs, irresistible desire to move the legs, confusional arousals, sleep terrors, sleepwalking, enuresis, and seizures.
- Inquire about symptoms consistent with sleep-disordered breathing (SDB), such as snoring, pauses during sleep, noisy breathing while asleep, diaphoresis, restlessness, and morning headaches.
- Assess awakening symptoms including frequency of coughing or choking, shortness of breath, nasal congestion or runny nose, and heartburn.
- Assess daytime behaviors such as daytime sleepiness, feeling tired even after a full night's sleep, difficulty concentrating, naps, stress, and fatigue. In children, particular attention to daytime behaviors including hyperactivity, inattentiveness, and restlessness should also be assessed.

Questions specific to the medical and psychiatric history

- Pay particular attention to dimensions of the assessment that include functions related to cardiopulmonary (e.g., hypertension, heart disease, lung disease), neurological (e.g., seizure disorder, restless legs syndrome), immune disorders (i.e., rheumatoid arthritis), gastroenterology (e.g., gastroesophageal reflux [GERD], irritable bowel syndrome [IBS]).
- Screen for psychiatric conditions (e.g., anxiety, depression, substance abuse, alcoholism), as well as other pain-related conditions including juvenile idiopathic arthritis, fibromyalgia, and sickle-cell disease.

Questions specific to patients with presentations of insomnia

- For patients presenting with insomnia, a thorough history includes assessment of sleep-wake patterns, medical conditions, psychiatric illnesses, medication use (prescribed as well as over-the-counter, herbal remedies), substance use/abuse, family history, and screening for anxiety, depression, and posttraumatic stress disorder (PTSD).
- Other areas for clinicians to address include caffeine, alcohol, and stimulant consumption, smoking habits, school or work performance, family functioning, and diet. Difficulty with finances, anxiety about life events, school, or work, alcohol use before bed, trauma, or parental divorce may significantly affect sleeping patterns.

Questions specific to impressions from bed partners

- Questions for the bed partners, parents, and caregivers include frequency of snoring, loud breathing, observed apneas, repeated kicking of the legs or arms, thrashing of the body, teeth grinding, and whether or not these activities result in sleeping in separate beds.
- Frequently during a clinical evaluation, it is the spouse or bed partner who mentions the patient's nightly snoring or restlessness, which adds data to the clinical assessment to explain daytime sleepiness.

Questions specific for the family

- Obtain information about family members or relatives who snore or are diagnosed with obstructive sleep apnea [OSA], restless legs syndrome [RLS], narcolepsy, insomnia, or other problems such as fibromyalgia, depression, and anxiety.

pen and paper or by computer while waiting for a clinic appointment or during an inpatient admission. These questions provide the basis upon which to build a more detailed sleep history and physical assessment.

Within the BEARS framework, the first question, "***B****edtime problems*" focuses on sleep initiation, including the time it takes to fall asleep at night and perceived difficulty with falling asleep. Sleep latency (time from lights out to sleep onset) of 30 minutes or more is an indicator of insomnia in adults. However, patients with obstructive sleep apnea (OSA) usually do not have difficulty with falling asleep. Behavioral insomnia of childhood (e.g., requiring parental presence to fall asleep or being held or rocked) is common in young children.

It is important to ask parents about their infants or toddlers' bedtime routine to understand potential contributing factors and patients'

perceptions of these factors. For example, in young children with difficulty falling asleep, it is important to ask parents about the typical bedtime routine (e.g., what time the routine starts, who puts the child down at bedtime, and parental response). Initiation insomnia may be due to stress or anxiety, the patient's lack of knowledge about the stimulating effects of caffeine, and exercise near bedtime. Depression and anxiety disorders are common in school-age children, adolescents, and adults who have difficulty falling asleep (Chorney, Detweiler, Morris, & Kuhn, 2008; Hudson, Gradisar, Gamble, Schniering, & Rebelo, 2009). (See Chapter 6, Insomnia and Chapter 12, Sleep and Psychiatric Disorders.)

The second question pertains to "***E****xcessive daytime sleepiness*" and elicits the effects of sleep loss on daytime function. It is important to distinguish between excessive daytime sleepiness (EDS) and fatigue. Sleepiness is the tendency to fall asleep during the day, whereas fatigue refers to an overwhelming sense of tiredness, lack of energy, and a feeling of exhaustion associated with impaired physical and/or cognitive function (Agency for Healthcare Research & Quality, 2010). Although both fatigue and EDS may result from sleep loss, EDS is usually considered a more specific response to sleep loss in adults.

It is important to ask adults whether they feel sleepy during the day, whether they have fallen asleep, and to elicit the circumstances surrounding the sleepiness or sleep episodes. Explicitly inquiring about sleepiness and the specific situations in which it occurs is necessary because of the potential risk of injury to self or others (e.g., sleepiness while operating machinery or a motor vehicle). In addition, daytime sleepiness often has a gradual onset; many people may compensate over time and do not realize they are sleepy or that it is abnormal.

Although the exact prevalence of daytime sleepiness in adults is not known, 36% of adults reported driving while drowsy or falling asleep while driving, 29% fell asleep or became very sleepy at work, 20% lost interest in sex, and 14% reported missing family events, work functions, or leisure activities because of excessive sleepiness (National Sleep Foundation, 2008). Some may experience problems with cognitive performance or memory or have unexplained accidents (e.g., motor vehicle crashes due to falling asleep behind the wheel of a motor vehicle). Therefore, assessment of problems with daytime performance and history of injury or accidents are critical components of assessment.

Although 17%–21% of school-age children and adolescents report daytime sleepiness, the exact incidence and prevalence are not known (Hoban & Chervin, 2000; Kothare & Kaleyias, 2008). This may be partly explained by developmental or cultural differences or a child's ability to describe sleepiness. Clinical symptoms of EDS can be challenging to detect in children because of age differences, pubertal development stage, insufficient sleep (e.g., poor sleep hygiene), or increased sleep drive (e.g., narcolepsy) (Fallone, Owens, & Deane, 2002; Hoban & Chervin, 2000; Kothare & Kaleyias, 2008). Children may respond to sleepiness with hyperactivity and restlessness during the day and at night. Parents may report that children are tired, sleepy, cranky, wake up feeling unrefreshed, find difficult to awaken in the morning, nap during the day, or are inattentive or falling asleep in school (Chervin, Hedger, Dillon, & Pituch, 2000; Owens, Spirito, & McGuinn, 2000).

Consequences of sleepiness in children include behavioral problems, inattention, and hyperactivity (Fallone et al., 2002; Hoban & Chervin, 2000; Kothare & Kaleyias, 2008; Ward et al., 2010), as well as difficulty performing in school. These behavioral issues are an important component of assessments of sleepiness.

The primary cause of daytime sleepiness is sleep loss. Assessment should include evaluation for potential contributing factors, including voluntary sleep restriction (e.g., staying up late at night); shift work; caregiving responsibilities; primary sleep disorders (e.g., OSA, periodic limb movement disorder, narcolepsy; sleep phase disorder); medical (e.g., heart failure, Parkinson's disease); and/or psychiatric conditions (e.g., depression). Severe sleepiness can also be a sign of narcolepsy. (See Chapter 9, Narcolepsy.) Therefore, information about cataplexy, a sudden bilateral loss of skeletal muscle tone precipitated by an emotion (e.g., laughter,

surprise), lasting seconds to minutes, should also be obtained.

Most adults need between 7 and 8 hours of sleep each night; children and adolescents need between 8 and 10 hours. (See Chapter 2, Developmental Aspects of Normal Sleep.) However, when patients ask, "how much sleep should I be getting?" the health care provider's response should be, "enough sleep at night to allow you to stay awake and alert during the day. For most adults, this is between 7 and 8 hours per night." Most sleep experts believe that it is not normal for healthy older adults and children to be sleepy during the day. Therefore, any sleepiness at all is an indicator of potential sleep loss.

Sleepy people often consume large amounts of caffeine in coffee, tea, chocolate, or other caffeinated beverages as a strategy to cope with excessive sleepiness. They may not report sleepiness because it is masked by this substance. Therefore, assessment of caffeine consumption is an important component of assessment and differential diagnosis.

The 3rd question addresses "***A****wakenings*"—difficulty maintaining sleep during the night. A great deal of research indicates that sleep continuity (the degree to which sleep is uninterrupted during the night by awakenings or even brief arousals that may not be associated with full awakening) is an important indicator of sleep quality and more important in many cases than sleep duration alone. It is important to distinguish between time in bed (TIB) and sleep duration. Patients may not accurately recall the number of hours of sleep obtained each night. They may either confuse amount of sleep with TIB or fail to account for the wake time during the night. Therefore, specific information should be obtained on typical bedtime (differentiate lights out time from time spent in the bed before attempting to fall asleep for reading, sex, or TV), typical time taken to fall asleep, number of awakenings, and rise time. Calculating the sleep efficiency (SE) [(total amount of night time sleep/TIB) × 100] assists in determining the degree of sleep continuity. This information is useful in guiding and evaluating treatment, and sharing this information with patients assists them to monitor their own progress. Usually SE greater than 90% is the goal for sleep promotion. However, there is variation across the life span in "normal" SE, with lower SE associated with aging.

Patients may be aware of their awakenings and be able to report the frequency and approximate duration. However, some patients may have frequent brief EEG arousals that are not associated with full awakenings or awareness. EEG arousals my impair the quality of sleep and are often associated with sleep-disordered breathing or periodic limb movements during sleep. They are of serious concern since they often prevent the patient from reaching slow wave (delta) sleep—the stage in which the restorative functions of sleep occur. The bed partner or family member may be able to provide information on restless sleep associated with EEG arousals.

In young children, sleep terrors, confusional arousals, and sleepwalking result from an arousal during slow-wave sleep (SWS) (deep sleep, non-REM Stage 3) and do not involve dreaming. Sleep terrors occur 1–2 hours after sleep onset and involve dramatic events characterized by sudden arousal from SWS. Children appear terrified (e.g., shrieking screams), disoriented, and spontaneously return to sleep with no recollection of the event the following morning. Common triggers for sleep terrors include sleeping in a different environment (e.g., at grandparents house), skipping a nap, or a fever. In contrast, nightmares are associated with rapid eye movement (REM) sleep that typically occurs during the middle or latter third of the night (after 3:00 A.M.) when there are increased bouts of REM sleep. Distinguishing features of nightmares include arousal to a fully awake state and vivid recollection of the event. Children awaken scared and are often afraid to fall back to sleep. Nightmares are rare before 3 years of age and typically decrease over time. Parents of children with occasional nightmares should be reassured that these events are normal and often benign, but children with frequent nightmares may require psychological evaluation and intervention. (See Chapter 8, Sleep-Related Movement Disorders and Parasomnias, for information on these disorders in adults.)

Many factors influence sleep continuity, and these factors should be carefully addressed as a

component of assessment and differential diagnosis. It is helpful to use an organizing framework to guide assessment of the etiology of nocturnal awakenings. This includes medical and psychiatric conditions, medications and other substances, behaviors (e.g., voluntary sleep restriction, social activities, shift work), acute or chronic stress, primary sleep disorders (e.g., period limb movement disorders, sleep-disordered breathing), and developmental variations.

Over the life span and across chronic conditions, there are many causes of nocturnal awakenings. Common examples include nocturia, frequently associated with awakenings in older adults; dyspnea associated with cardiac or pulmonary conditions; hot flashes in perimenopausal and menopausal women; and caregiving responsibilities for a disabled adult or young child. Nocturnal awakenings are often a source of concern for parents of young children, although awakenings may be normal in many cases. (See Chapter 2, Developmental Aspects of Normal Sleep.) Environmental factors, such as noise, pets in the room, a restless bed partner, or excessive lighting are easily addressed, but often overlooked (National Sleep Foundation, *Healthy Sleep Tips*, Retrieved from http://www.sleepfoundation.org/article/sleep-topics/healthy-sleep-tips).

Excessive use of caffeine found in colas, coffee, tea, and chocolate is a very common source of frequent nocturnal awakenings, but many individuals are not aware of the large amounts of caffeine that they consume. Assessment should include the number of servings and proximity to bedtime, as well as substances used to self-treat sleep complains, such as over-the-counter medications, herbs, supplements, and alcoholic beverages. Although many adults use alcohol based on the assumption that it is a sleep aid, it only improves sleep onset, and usually leads to sleep fragmentation, including mid-sleep awakenings. Thus, patient education is needed to raise awareness about its negative impact on sleep, as well as its addictive properties.

The 4th question addresses "***R****egularity*" and the rhythm of daily rest and activity cycles. This permits understanding of the circadian variation in sleep and wake (see Chapter 1, Physiological and Behavioral Aspects of Sleep) and the consistency of these patterns with normal developmental sleep needs and patterning and social, behavioral, and environmental factors. The consistency in patterning of sleep-wake scheduling is often a determinant of good daytime function, and lack of consistent patterning or lack of synchronicity with work, school, or social schedules may contribute to poor daytime performance. (See Chapter 2, Developmental Aspects of Normal Sleep; Chapter 10, Circadian Rhythm Disorders; and Chapter 22, Sleep Promotion in Occupational Health Settings.)

Assessment of regularity includes information on consistency of bedtime and rise times from night to night, including weekends and weekdays. Frequency and timing of naps are important considerations. It is also necessary to assess the patterning of work, social, and family obligations and their fit with sleep-wake patterns. This fit and the individual's performance of roles and responsibilities or their perceptions of difficulty are important determinants of circadian rhythm disorders. (See Chapter 10, Circadian Rhythm Disorders.) For example, the earlier bedtimes and wake times sometimes seen in older adults may only be problematic if the individual desires to be engaged in activities during the evening hours.

The 5th question of the BEARS mnemonic represents "***S****leep-disordered breathing*," a condition associated with repetitive partial or complete closure of the upper airway during sleep, hypoxia, and sympathetic activation. (See Chapter 7, Sleep-Related Breathing Disorders and Chapter 13, Pediatric Sleep Disorders.) The symptoms of sleep-disordered breathing are listed in Table 5.2. Often, the patient is not aware of snoring or apneas occurring during sleep. Therefore, nurses should interview the bed partner (in the case of an adult) or a parent (in the case of the child) to elicit the presence of these symptoms. Snoring is the auditory evidence of a partially obstructed airway, while pauses in breathing are signs of total obstruction.

Physical Examination

A complete physical examination provides useful information about the general health of the

patient and specific conditions that may be causes or consequences of sleep disorders (e.g., heart disease, asthma, diabetes, Parkinson's disease, pain, depression, anxiety) and is guided by sleep history and differential diagnosis. For example, patients who are sleepy and snore loudly should be suspected of having OSA; patients with poor sleep and pain should have the sources of their pain evaluated. However, aside from craniofacial and airway structure that provides strong evidence supportive of the diagnosis of sleep-disordered breathing, most physical findings do not directly lead to diagnosis of sleep disorders.

Anthropometric data, including weight, height, body mass index, waist-hip profile, and neck size in adults are critical to assessing risk for sleep-disordered breathing. A body mass index more than 30 kg/m^2 and neck circumference as more than 17 inches in men or more than 16 inches in women are major risk factors for OSA in adults and waist circumference (Bixler et al., 2009; Li et al., 2010) and obesity (BMI > 95th percentile) are anthropometric risk factors in children (Epstein et al., 2009; O'Brien, Sitha, Baur, & Waters, 2006; Rosen et al., 2003). However, thin and normal weight individuals may also have this condition. Infants may present with failure to thrive (FTT), and young children may present with low percentile values of growth related to increased metabolic demands associated with SDB (Marcus, 2001).

Inspection of the head and neck includes careful assessment of the facial profile, oral cavity, and nasal passages. The facial profile should be assessed for anatomical anomalies such as enlarged tongue and high-arched narrow palate, micrognathia (smallness of the jaw, particularly the mandible), and facial hypoplasia that may obstruct the nasopharyngeal and oropharyngeal airways. (See Chapter 7, Sleep-Related Breathing Disorders.) Enlarged tonsils in children, but not necessarily in adults, are risk factors for sleep apnea (Epstein et al., 2009). The nurse should also inspect the nasal passages for swollen turbinates, deviated septum, polyps, and overall patency. Chronic sinusitis, allergies, or structural defects of the nose may contribute to sleep-disordered breathing.

Cardiovascular assessment includes a focus on blood pressure, heart sounds, and electrocardiography because dysrhythmias and hypertension are frequent consequences of sleep-disordered breathing. Extremities should be evaluated for edema. Assessment of the pulmonary system should focus on structural abnormalities (e.g., scoliosis) that may impair pulmonary function and breath sounds that may reveal congestion. Neurological and musculoskeletal (muscle tone and motor assessments) systems should also be conducted and accompanied by questions about restless leg syndrome (RLS) and periodic movements. Children may present with difficultly falling asleep secondary to limb pain and/or discomfort (e.g., pins-n-needles, fizzing, popping, tingling, burning, crawling like bugs) at bedtime, frequent kicking during sleep, and leg aches in the morning (Arbuckle et al., 2010).

Laboratory Findings

Because of the linkages of sleep-disordered breathing with cardiovascular and metabolic consequences, it may be useful to obtain data on glycemic control (e.g., fasting glucose, HgbA1c) and cardiovascular risk (e.g., cholesterol). However, these findings are not specific to the diagnosis of sleep apnea. Electrocardiography may provide important information on dysrhythmias and cardiac enlargement associated with sleep apnea. Pulmonologists may obtain pulmonary function tests, and in specialized sleep laboratory settings, measures of oxygenation and End-Tidal C02 may be obtained to evaluate sleep-disordered breathing. However, these indicators are not usually a component of routine screening for sleep disorders in primary care settings.

Health History and Medication Review

A detailed health history provides important information about the many medical and psychiatric conditions and associated symptoms and treatments that contribute to poor sleep or may be consequences of poor sleep. Many medications can also contribute

to sleep difficulty. Therefore, these should be carefully reviewed. (See Chapter 6, Insomnia; Chapter 12, Sleep and Psychiatric Disorders; and Chapter 11, Sleep in Medical Disorders.)

SPECIALIZED SLEEP MEASUREMENT

Sleep Diaries

Sleep diaries are frequently used to evaluate the patterning of sleep over time for clinical assessment and research purposes. This is particularly important because of the variability in sleep patterns. This information is usually used by the clinician to guide treatment, and it is very helpful for patients to see the information and use it to guide changes to their sleep-related behaviors. At a minimum, sleep diaries are used to elicit bedtimes, lights out times, wake times, out of bedtimes, and daytime naps. These data are used to calculate important quantitative sleep characteristics used to guide treatment (sleep latency, sleep duration, SE) and the patterning of sleep throughout the 24-hour day. Most diaries also elicit information on behaviors associated with sleep, such as meal times, caffeine consumption, and medications. They are also used to elicit perceptions about the quality of sleep. Depending on the needs of the specific patient population, diaries may also elicit frequency and/or severity of sleep-related symptoms, such as pain or dyspnea. Of course, increasing the amount of information obtained increases the time needed to complete the diary and may increase patient burden. Sleep diaries are reliable and valid measures of sleep-wake cycle. (See Chapter 1, Physiological and Behavioral Aspects of Normal Sleep.)

Sleep diaries are particularly useful in assessing changes in sleep over time. Although the amount of time needed varies considerably, especially in research, clinicians usually ask patients to complete the sleep diaries for 2 weeks. This permits adequate evaluation of weekend and weekday patterns of sleep. Usually patients are instructed to complete the diary each morning upon awakening to permit adequate recall of the past night's sleep and events over the past 24 hours. However, diaries can be completed more often, depending on the purpose. For example, completing the diary in the evening may permit recall of daytime events, if those are of interest.

Sleep diaries are available in several formats. Some include blocks that the patient colors in to indicate wake or sleep time. An example used for sleep assessment in adults is in Chapter 1. Additionally, Chapter 1 contains an adult sleep diary from the American Academy of Sleep Medicine. An example of a child sleep diary can be found at http://www.sleepforkids.org/pdf/SleepDiary.pdf. Many styles that can be printed are available online. Computerized versions are also available and can be used electronically through computers and PDAs.

Wrist Actigraphy

Actigraphs are electronic accelerometers that record motion and acceleration. Electronic computerized algorithms are used to estimate sleep patterns from these data. These algorithms are validated based on polysomnography. When used to evaluate sleep in adults and children, actigraphs are typically worn on the nondominant wrist. In infants or toddlers, actigraphs are worn on the ankle. Actigraphy has been used extensively in a variety of settings for research purposes. However, actigraphs are also used in clinical settings for assessment and evaluation of treatment effects. There is a CPT code that can be used to bill for actigraphy. However, reimbursement varies considerably across insurance companies. (See Chapter 1, Physiological and Behavioral Aspects of Sleep.)

Questionnaires

Questionnaires are useful in clinical settings in diagnosing sleep disorders in children or adults in many settings. However, because sleep is a multidimensional biobehavioral phenomenon and each sleep disorder has very specific attributes, it is important to select instruments that measure the characteristics of sleep that are of primary interest. For example, if the goal is to elicit symptoms of sleep apnea, the instrument must be a valid measure of these symptoms,

rather than global sleep quality alone. Some questionnaires directly elicit the characteristics of sleep. Others (e.g., Functional Outcomes of Sleep Scale) measure the consequences of poor sleep.

When selecting instruments for clinical purposes, it is also necessary to consider the language, reading level, and developmental level of the patient. Commonly used instruments have been translated into languages, in addition to English. Brevity, sensitivity and specificity are important feasibility factors to consider when selecting instruments for clinical purposes. Table 5.3 presents examples of pediatric and adult sleep questionnaires that have established reliability and validity and are widely used in both clinical and research settings. They are used in research and clinical practice, and many are available on the Internet. Authors, citations, Web site availability, concepts measured, and target population are described in the table. Additional information on reliability and validity is widely available in the literature.

Pediatric Sleep Questionnaires

Numerous reliable and valid questionnaires are available to evaluate characteristics of children's sleep quality and symptoms of specific sleep disorders. As indicated in Table 5.3, questionnaires elicit either parents' reports of the child's sleep or the child's report.

Adult Sleep Questionnaires

There are numerous questionnaires available to measure characteristics of sleep and sleep disorders in adults and older adults. A widely used measure is the Pittsburgh Sleep Quality Index (PSQI), a measure of global sleep quality (Buysse et al., 1989). This provides a broad view of sleep quality, but is not specific for particular sleep disorders. Other questionnaires, examples of which are measured in Table 5.3, measure specific sleep symptoms. Setting is also a consideration. For example, the Richards–Campbell Sleep Questionnaire (Richards, O'Sullivan, & Phillips, 2000) was designed to evaluate sleep in an acute care setting.

INTERPRETATION OF OBJECTIVE MEASURES OF SLEEP

Polysomnography

Polysomnography (PSG) is a technique that includes several "poly" (Greek) measures of biological indicators of sleep "somnus" (Latin). It primarily includes electro-encephalographic measures of brain waves, measures of eye movement (electro-oculography), and muscle tension (electromyelogram). Measures of airflow through the nose (and sometimes the mouth) are obtained with a thermistor or pressure transducer, and measures of respiratory effort (expansion of chest and abdomen) and pulse oxygen saturation (pulse oximetry) are obtained to determine the presence of sleep-disordered breathing. Usually microphones are use to record snoring. Electrocardiography is used to evaluate heart rate and rhythm disturbances that may correspond with sleep stages and respiratory events. Depending on the purposes of the study, additional measures, such as electromyographic measures of leg movement (to detect periodic limb movements during sleep), gastric pH, seizure activity, and other physiological variables may be obtained. In the sleep laboratory, video monitoring may also be employed for observation of behaviors (e.g., seizure activity, REM behavior disorder) that correspond with physiologic parameters. (See Chapter 1, Physiological and Behavioral Aspects of Sleep.)

PSG is the primary method used to diagnose sleep-related breathing disorders and diagnose other causes of EDS, such as narcolepsy, sleep fragmentation due to nocturnal arousals, and periodic limb movement disorders; delineate between parasomnias and nocturnal seizures; and to assess the efficacy of treatments for various sleep disorders (Kushida et al., 2005). Patients who report symptoms suggestive of the conditions should be referred to a sleep disorders center for specialized evaluation. PSG is not indicated for the diagnosis of insomnia, except to rule out other causes of sleep fragmentation, such as sleep-disordered breathing or periodic limb movements during sleep.

Table 5.3 ■ *Instruments Used for the Clinical Assessment of Sleep in Children and Adults*

Instrument Authors/Availability	Items, Time to Complete, Description of Measure	Population/Setting
Pediatric Questionnaires		
Children's Sleep Habits Questionnaire (CSHQ) (http://www.kidzzzsleep.org/pdfs/CSHQ.pdf) (Owens, 2000).	45-item, retrospective survey, completed by parents; Measures: bedtime resistance, sleep onset delay, sleep duration, sleep anxiety, sleep-disordered breathing, night wakings, parasomnias, and morning wakening/daytime sleepiness	Children, aged 4–10 with and without chronic conditions
Pediatric Sleep Questionnaire (PSQ) (Chervin, 2000)	22-item (5–10 minutes), parent report; Measures: sleep-related breathing, problems including snoring, mouth breathing, and trouble breathing; restless sleep including frequent leg kicking and growing pains; and sleep behaviors including difficulty falling asleep, parasomnias, and inattention/hyperactivity.	2–18 year-old children
Sleep Disturbance Scale for Children (SDSC) (Bruni et al., 1996)	27-item, parent retrospective report (past 6 months) Measures: disorders of initiating and maintaining sleep; sleep breathing disorders check in binder]; disorders of arousal/nightmares; sleep-wake transition disorders; five disorders of excessive somnolence; sleep hyperhidrosis	6–15 year-old children
Sleep Self-Report (SSR) (http://www.kidzzzsleep.org/pdfs/SleepSelfReport_Child'sForm_-9-02.pdf) (Owens, 2000)	26-item child retrospective report (previous week) Measures: bedtime routines; sleep behaviors; daytime sleepiness	7–12 year-old children with and without chronic conditions
School Sleep Habits Survey (SSHS) (http://www.sleepforscience.org/contentmgr/showdetails.php/id/93) (Wolfson et al., 2003; Wolfson & Carskadon, 1998)	63-item adolescent retrospective report (previous 2 weeks) Measures: school-night and weekend-night total sleep time; bedtime; rise time; sleep schedule regularity; weekend delay; weekend oversleep; school performance; daytime sleepiness; sleep-wake behavior problems; depressive mood	13–19 year-old adolescents
Measures of Consequences of Sleep Loss—Children		
Pediatric Daytime Sleepiness Scale (PDSS) (http://www.iowasleep.com/pdfs/Pediatric%20Daytime%20Sleepiness%20Scale.pdf) (Drake et al., 2003; Perez-Chada et al., 2007)	8-item child and adolescent retrospective report (previous 2 weeks) Measures: daytime sleepiness	11–15 year-old children and adolescents
Adult Questionnaires		
Pittsburgh Sleep Quality Index (PSQI) (http://www.sleep.pitt.edu/includes/showFile.asp?fltype=doc&fIID=2532) (Buysse et al., 1989)	19-item self-report (previous month) Measures: sleep disturbances; sleep quality; sleep latency; sleep duration; sleep efficiency; specific sleep disturbances; use of sleep medication; daytime dysfunction	Adults
Berlin Sleep Questionnaire (https://www.swclab.com/images/PDFS/Berlin-Questionnaire.pdf) (Netzer, Stoohs, Netzer, Clark, & Strohl, 1999)	10-item self-report of sleep apnea risk Measures: snoring behavior; wake time sleepiness; fatigue; obesity; hypertension	Adults

Continued

Table 5.3 ■ *Instruments Used for the Clinical Assessment of Sleep in Children and Adults (Continued)*

Instrument Authors/Availability	Items, Time to Complete, Description of Measure	Population/Setting
Richards-Campbell Sleep Questionnaire (RCSQ) (Richards et al., 2000)	5-item visual analog scale to assess sleep quality in acute care settings Measures: sleep depth; sleep latency; awakenings; percentage of time awake; quality of sleep	Adults; acute care
Insomnia Severity Index (https://www.myhealth.va.gov/mhv-portal-web/anonymous.portal?_nfpb=true&_pageLabel=healthyLiving&contentPage=healthy_living/sleep_insomnia_index.htm) (Bastein, Vallières, & Morin, 2001)	7-item self-report of perceived insomnia severity Measures: severity of insomnia; current sleep pattern; sleep interferences; noticeability of sleeping problem to others; concern about sleeping problems	Adults
Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS) (http://www.journalsleep.org/Articles/301114.pdf) (Espie, Inglis, Harvey, & Tessier, 2000)	10-item self-report of specific sleep-related cognitions about sleep and insomnia Measures: misconceptions about the causes of insomnia; misattribution or amplification of its consequences; unrealistic sleep expectations; diminished perception of control and predictability of sleep; faulty beliefs about sleep-promoting practices	Adults
Measures of Consequences of Sleep Loss		
Epworth Sleepiness Scale (ESS) (http://www.stanford.edu/~dement/epworth.html) (Johns, 1991)	8-item self-report of overall daytime sleepiness occurring during specific activities of daily life: sitting and reading; watching TV; sitting inactive in a public place; as a car passenger; lying down to rest in the afternoon; sitting and talking to someone; sitting quietly after lunch; in a car stopped at a traffic light	Adults
Sleep-Wake Activity Inventory (SWAI) (Rosenthal, Roehrs, & Roth, 1993; Rosenthal et al., 1997)	59-item self-report of sleepiness (previous week) Measures: excessive daytime sleepiness; psychic distress; social desirability; energy level; ability to relax; nocturnal sleep	Adults
Functional Outcomes of Sleep Questionnaire (FOSQ) (Weaver, 2001; Weaver et al., 1997)	30-item self-report on the effect of excessive sleepiness on activities of daily living Measures: activity level; vigilance; intimacy; sexual relationships; general productivity; social outcome	Adults

Interpretation of PSG reports requires an understanding of the terminology used and evaluation of normal versus abnormal findings that may indicate sleep disorders. A detailed description with examples of data is available (Kakkar & Hill, 2007). As with other clinical reports obtained on specialized procedures, the report of the polysomnogram usually includes the sleep specialist's interpretation of the findings and an impression of the patients' diagnosis and recommendations for treatment and follow-up care based on a full clinical sleep assessment. If treatment was provided (e.g., positive airway pressure titration for sleep-disordered breathing), the report will also provide the recommended pressure settings, type of positive pressure device, and prescribed patient interface (e.g., mask or nasal pillows). (See Chapter 7, Sleep-Related Breathing Disorders and Chapter 13, Pediatric Sleep Disorders.)

Careful review of the sleep recommendations is essential to assisting patients with needed follow-up care and adherence after the specialized sleep evaluation.

PSG reports include information on sleep duration, continuity, and sleep architecture. Sleep architecture includes the number of minutes and percentage of the sleep period and time of bed in each sleep stage (N1, N2, N3, and REM) and REM sleep stage. (See Chapter 1, Physiological and Behavioral Aspects of Sleep.) Measures of sleep duration and continuity include total sleep time (TST), TIB, wake after sleep onset (WASO) (percentage of time awake for the time from sleep onset until final awakening); sleep latency, (time from lights out to sleep onset); and SE (ratio of TST/TIB).

Reports include the number and type of abnormal respiratory events (and the number/hour). These include apneas (obstructive apnea [OA], central apnea [CA], or mixed apneas) or hypopneas. (See Chapter 7, Sleep-Related Breathing Disorders.) The average of apneas and hypopneas per hour of sleep (apnea-hypopnea index [AHI]) is used to classify the severity of OSA. In adults, OSA is classified as follows: (1) mild OSA: >5 to >15; (2) moderate >15 to <30; and (3) severe ≥ 30 events per hour (American Academy of Sleep Medicine Task Force, 1999). Usually the respiratory disturbance index (RDI) (average of the apneas, hypopneas, and respiratory-event related arousals [breaths with limited obstruction that may result in snoring but are not full hypopneas]) is also reported. Information is also provided on oxygen saturation throughout the night, including the minimum, maximum, and proportion of time spent below specific thresholds. These data are used to assist in determining the severity of sleep-disordered breathing.

The adult OSA criteria cannot be applied in children due to differences in etiology of sleep-disordered breathing, and clinical presentation. An apnea index more than 1 is abnormal in children and an AHI more than 5 events per hour indicates the presence of OSA (American Thoracic Society, 1999). Grigg-Damberger and colleagues (2007) provide detailed information about arousal, cardiac, respiratory, and movement criteria between children and adults.

The number of periodic limb movements (PLMS) and periodic limb movement events associated with an arousal are calculated and reported as an index/hour of TST (Grigg-Damberger et al., 2007). PSG reports also typically provide information about other physiological data obtained, such as cardiac dysrhythmias and seizure activity if present.

PSGs have several limitations that should be considered in interpreting the results. PSG data alone are not diagnostic of sleep disorders, but are used along with health history and physical assessment. Sleeping in a foreign environment leads to abnormalities in sleep architecture that can limit the diagnostic value of the test. In addition, many patients experience a "first night effect" or poor sleep (e.g., reduced SE, increased awakenings and arousals, prolonged sleep onset, decreased percentages of REM and non-REM that may preclude the detection of mild sleep disorders). There is also day-day variability in sleep-disordered breathing that may result in failure to detect the disorder on a single night study.

Nurses who work in primary care settings may care for patients who are referred for specialized sleep studies. It is important to explain the procedures to patients and their families. Usually patients are asked to come to the sleep disorder centers early in the evening to allow adequate time for setup of the equipment and explanation of procedures. Depending on the purposes of the test, they may be asked to follow their typical sleep-wake routine and other sleep-related behaviors (e.g., use of alcohol or hypnotics) in the days prior to the procedure. In other cases, they may be instructed not to do this. Most sleep laboratories ask patients to bring their own bed clothes and have shower facilities available so that patients can go to work or school immediately after the study in the morning. Adult patients found to have significant sleep apnea during the first half of a night of PSG may also undergo titration with continuous positive airway pressure (CPAP) to determine its efficacy. This is called

a "split night" study. Otherwise, patients with significant sleep apnea must return for a second night in the sleep laboratory to undergo CPAP titration. Accommodations are made for parents to sleep at the sleep center when young children undergo PSG. Patient information about what to expect during sleep testing is available for adults sleep testing online at http://www.nhlbi.nih.gov/health/dci/Diseases/slpst/slpst_during.htm http://www.talkaboutsleep.com/sleep-basics/viewasleepstudy.htm. Information for children is available at http://www.chop.edu/service/sleep-center/your-childs-study.

Multiple Sleep Latency Test

The Multiple Sleep Latency Test (MSLT) is a standardized and validated measure of the propensity to fall asleep during the day (Littner et al., 2005). The MSLT is usually used to diagnose or rule out narcolepsy (see Chapter 9, Narcolepsy), but it is not routinely used to evaluate EDS associated with suspected OSA, other medical or neurologic disorders, insomnia, or circadian rhythm sleep disorders (Littner et al., 2005).

An MSLT consists of five 20-minute nap opportunities at 2-hour intervals beginning from 1.5 to 3 hours after awakening from nocturnal sleep (Sullivan & Kushida, 2008). Patients are instructed to allow themselves to fall asleep *or* to resist falling asleep. The MSLT is designed to: (1) quantify sleepiness by measuring how quickly an individual falls asleep (e.g., sleep latency) on sequential naps during the day and (2) identify the abnormal occurrence of sleep-onset REM during the napping opportunities. The mean latency for all the naps and the number of naps from latency to sleep onset REM are calculated. Two or more sleep onset REM periods (SOREMPS) within 15 minutes of sleep onset are important in the diagnosis for narcolepsy and idiopathic hypersomnia. (See Chapter 9, Narcolepsy.)

In adults, a mean sleep latency of less than 5 minutes indicates pathologic daytime sleepiness. Sleep latencies between 5 and 10 minutes indicate moderate sleepiness with less well-defined pathology and consequences, and sleep latencies more than 10 minutes are normal (Carskadon et al.,1986). In children, the clinical cutoff value for EDS measured by MSLT and criteria for EDS have not been established (Fallone et al., 2002; Hoban & Chervin, 2000; Kothare & Kaleyias, 2008; Ward et al., 2010). Healthy school-age children rarely nap during the day and often remain awake throughout a 20-minute MSLT. Children typically have longer sleep latency than the cutoff criterion of 10 minutes used in the assessment of EDS in adults.

A PSG should be performed the night before the MSLT to assess sleep quantity and quality to assure that the daytime sleepiness is not a result of sleep deprivation. Other factors that may contribute to misinterpretation of MSLT results include medications that may cause sleepiness or excessive arousal (e.g., caffeine), sleep deprivation within a week prior to the MSLT, and circadian rhythm disorders, such as shift work disorder or delayed phase syndrome that may have an impact on the normal circadian pattern of sleep and wake. Assessment of these factors and normalizing them as much as possible is necessary to obtain an accurate result.

Maintenance of Wakefulness Test

The Maintenance of Wakefulness Test (MWT) measures a patient's ability to stay awake in a quiet, nonstimulating situation for a period of time. The MWT may be used to assess response to treatment with medications or in patients who need to demonstrate the ability to stay awake for safety or employment issues (Littner et al., 2005). The MWT is rarely used in children. At present, MWT testing is primarily used for research purposes. Normative data for the MWT are limited and more studies are needed to determine how performance on the MWT may affect actual performance (Doghramji et al., 1997).

SLEEP ASSESSMENT RESOURCES FOR NURSES

Numerous resources are available to guide screening and assessment of adults and children for sleep disorders (Bloom et al., 2009;

Carno & Connolly, 2005; Espie, 2009; Lee & Ward, 2005; McCurry, LaFazia, Pike, Logsdon, & Teri, 2009; Ward, Rankin, & Lee, 2007) (see Table 5.4). Extensive information including case studies and educational resources can be found on the AASM MEDSleep Educational Resource Web site.

Table 5.5 provides examples of clinical cases of sleep disorders that common clinical cases presented to nurses.

Table 5.4 ■ *Web-Based Resources*

- The National Sleep Foundation*: http://www.sleepfoundation.org
- The American Academy of Sleep Medicine. Sleep Educational Resource: http://www.aasmnet.org/MedSleep.aspx
- The National Sleep Foundation for Children: http://www.sleepforkids.org
- The Sleep Research Society: http://www.sleepresearchsociety.org

*Bilingual

Table 5.5 ■ *Examples of Clinical Situations in Which Sleep Assessment Is Needed*

1. Hyperactivity, attention problems, or restlessness in children may be secondary to unrecognized sleep-disordered breathing.
   a. Children may be misdiagnosed with attention deficit hyperactivity disorder (ADHD) and placed on stimulants, when in fact sleep patterns and problems were not assessed by the health care provider.
   b. Ask both the parent and child about their child's sleep routine (e.g., typical bedtime, wake time, napping patterns during the weekday and weekend) and sleep patterns (e.g., snoring, restlessness during sleep [frequent kicking during sleep, tossing and turning, sweating while asleep]).
   c. Ask parents about their child's school performance (e.g., teacher concerns).
   d. Refer the child to an accredited pediatric sleep disorders center for a thorough evaluation.
2. Daytime sleepiness in children and adults is a common consequence of undiagnosed sleep disorder (e.g., obstructive sleep apnea, narcolepsy) and/or insufficient sleep.
   a. Assess sleep routine (e.g., bedtime environment) and sleep patterns (e.g., typical bedtime and wake time during weekdays and weekends), frequency and duration of daytime napping.
   b. Evaluate for potential factors of daytime sleepiness (e.g., shift work, caregiving responsibilities; primary sleep disorders [obstructive sleep apnea, periodic limb movement disorder, narcolepsy, sleep phase disorder]; medical [e.g., heart failure, Parkinson's disease], and/or psychiatric conditions [e.g., depression]).
   c. Assess severity of sleepiness (e.g., sleepy during the day, at school, at work, or while driving), frequency of falling asleep at school or work and for how long, difficulty waking up in the morning.
   d. Evaluate the circumstances surrounding the sleepiness and the specific situations in which it occurs (e.g., sleepiness while operating machinery or a motor vehicle).
   e. Refer the individual to an accredited pediatric or adult sleep disorders center for a thorough evaluation.

CONCLUSION

Screening and assessment for sleep disorders is a necessary component of health assessment across patient populations and clinical settings. Nurses are in a unique position to screen patients for potential sleep disorders; treat specific types of sleep problems; refer patients to accredited sleep disorders centers for difficult or complicated sleep disorders; and to provide ongoing assessment and diagnoses over the course of development and changes in health.

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6 Insomnia

Carla Jungquist

Insomnia is the most common sleep disorder in adults. It is associated with repeated difficulty with sleep initiation, duration, consolidation, and quality that occurs despite adequate time and opportunity for sleep and results in some form of daytime impairment (American Academy of Sleep Medicine, 2005). It is associated with high levels of morbidity, injury, daytime functioning, and poor quality of life, as well as excessive health care costs. Although insomnia was previously considered to be just a symptom of other medical or psychological disorders, it has become clear that it is a multidimensional, biobehavioral disorder with a multifactorial etiology. This chapter addresses the prevalence, characteristics, risk factors, diagnosis, and treatment of insomnia.

CHARACTERISTICS/NOSOLOGY OF INSOMNIA

Primary insomnia is the subjective report of difficulty initiating and/or maintaining sleep, or the complaint of nonrestorative sleep that lasts for more than 1 month (Diagnostic and Statistical Manual for Mental Disorders-IV, American Psychological Association, 2000). The problem with the use of the label "primary insomnia" is that it does not account for sleep disruption secondary to other sleep disorders or comorbid medical or psychiatric conditions. In the past, clinicians assumed that treatment of a primary medical or psychiatric condition would improve insomnia, but in the vast majority of cases, insomnia persisted after the primary disorder was addressed. The labels of primary insomnia and secondary insomnia were developed to distinguish primary (not associated with other conditions) from secondary insomnia. Subsequently, sleep experts proposed using the term "comorbid" to replace "secondary" because of the difficulty in determining the causal sequence of these conditions. As part of the revision of the Diagnostic and Statistical Manual of Mental Disorders V, an expert panel on sleep is updating definitions for sleep–wake disorders. It is expected that the term "primary insomnia" will be replaced with the label "insomnia disorder" to avoid the problem with attributing causal relationships between insomnia and comorbid conditions. For example, a patient with both major depression disorder and an insomnia disorder is labeled with both conditions because both insomnia and depression should be treated. See Tables 6.1 and 6.2 for definitions of insomnia and classifications of insomnia severity, respectively.

Insomnia is acute if it lasts less than 1 month and chronic if the symptoms have occurred consistently for more than 1 month (AASM, 2005; Buysse, Ancoli-Israel, Edinger, Lichstein, & Morin, 2006; Mai & Buysse, 2008). Insomnia is also categorized by the nature of the sleep complaint: (1) initial, (2) middle, and (3) terminal (Dornberger, 2008; Mai & Buysse, 2008; Morin et al., 2009; Ohayon & Reynolds, 2009). The primary symptom of initial insomnia is sleep latency (time to fall asleep) in excess of 30 minutes. Maintenance insomnia, common in patients with comorbid medical conditions, is associated with frequent awakenings with wakefulness in excess of 30 minutes during the night. The hallmark

Table 6.1 ■ DSM IV-TR *Diagnostic Criteria for 307.42 Primary Insomnia*

A.	The predominant complaint is difficulty initiating or maintaining sleep, or nonrestorative sleep, for at least 1 month.
B.	The sleep disturbance (or associated daytime fatigue) causes clinically significant distress or impairment in social, occupational, or other important areas of functioning.
C.	The sleep disturbance does not occur exclusively during the course of narcolepsy, breathing-related sleep disorder, circadian rhythm sleep disorder, or a parasomnia.
D.	The disturbance does not occur exclusively during the course of another mental disorder (e.g., major depressive disorder, generalized anxiety disorder, a delirium).
E.	The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.

Source: Diagnostic and Statistical Manual of Mental Disorders (4th ed.) by American Psychiatric Association, 2000, Arlington, VA: Author.

Table 6.2 ■ *Severity of Insomnia* (International Classification of Sleep Disorders) *Definitions*

Mild Insomnia: an almost nightly complaint of an insufficient amount of sleep or not feeling rested after the habitual sleep episode. It is accompanied by little or no evidence of impairment of social or occupational functioning. Mild insomnia is often associated with feelings of restlessness, irritability, mild anxiety, daytime fatigue, and tiredness.
Moderate Insomnia: a nightly complaint of an insufficient amount of sleep or not feeling rested after the habitual sleep episode. It is accompanied by mild or moderate impairment of social or occupational functioning. Moderate insomnia is always associated with feelings of restlessness, irritability, anxiety, daytime fatigue, and tiredness.
Severe Insomnia: a nightly complaint of an insufficient amount of sleep or not feeling rested after the habitual sleep episode. It is accompanied by severe impairment of social or occupational functioning. Severe insomnia is associated with feelings of restlessness, irritability, anxiety, daytime fatigue, and tiredness.

Source: The International Classification of Sleep Disorders, Revised; Diagnostic and Coding Manual by American Academy of Sleep Medicine, 2005, Westchester, IL: Author.

of terminal insomnia is waking up 30 minutes before the alarm or planned wake time. Contrary to the term used to describe it, terminal insomnia is not associated with risk of mortality during sleep. Although these categorizations help to describe the patients' primary concern with sleep, insomnia often presents as a mix of the three types.

Daytime consequences of insomnia are clinically important and are required to formally establish the diagnosis. Common daytime consequences of insomnia include fatigue, decreased energy, decreased performance, and difficulty concentrating (Abdel-Khalek, 2004; Roth, 2007; Walker, 2008). Patients with insomnia may be excessively sleepy during the day, but some experience heightened arousal and are not as sleepy during the day as their nocturnal sleep impairment might suggest. The severity of insomnia often varies from night-to-night, with nights of refreshing sleep often followed by a sequence of nights of poor sleep (Perlis et al., 2009). Irregularity in sleep patterning often contributes to expectations of poor sleep that perpetuate insomnia.

Circadian rhythm disorders associated with jet lag, shift work, irregular sleep–wake scheduling and other conditions are frequently confused with insomnia. (See Chapter 10, Circadian Rhythm Disorders.) Careful assessment to differentiate these disorders from insomnia is important because the underlying etiologies and treatment differ substantially.

Insufficient sleep opportunity (either intentional or unintentional) is common in the American culture and is sometimes confused with insomnia. Excessive work hours, late night social activities, or use of the Internet or television may lead to decreased sleep time or partial sleep deprivation and daytime sleepiness, fatigue, and the general sense of nonrestorative sleep (Miyata et al., 2010; National Sleep Foundation, 2008). However, this is not insomnia. Insomnia occurs in the presence of sufficient opportunity for sleep.

EPIDEMIOLOGY OF INSOMNIA

Insomnia symptoms are highly prevalent, with 30%–34.5% of the adult population exhibiting difficulty initiating or maintaining sleep or nonrestorative sleep at least three nights per week (Buysse et al., 2006; Mai & Buysse, 2008; Morphy, Dunn, Lewis, Boardman, & Croft, 2007; Ohayon, Krystal, Roehrs, Roth, & Vitiello, 2010;

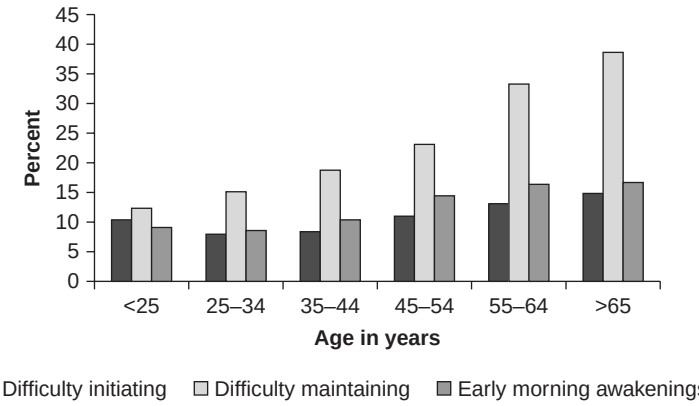

Figure 6.1 ■ Sleep disturbances by type over the lifespan. *Source:* "Epidemiological and Clinical Relevance of Insomnia Diagnosis Algorithms According to the DSM-IV and the International Classification of Sleep Disorders (ICSD)" by M. M. Ohayon and C. F. Reynolds, III, 2009, *Sleep Medicine*, *10*(9), pp. 952–960. Reprinted with permission.

Ohayon & Reynolds, 2009). The prevalence of primary insomnia (insomnia that is not associated with a comorbid psychiatric or medical condition) is much lower and estimated to be between 3% and 10% of the population (Buysse et al., 2008; Kao, Huang, Wang, & Tsai, 2008; Morphy et al., 2007; Ohayon, 2005; Ohayon & Reynolds, 2009). The annual incidence of insomnia is between 6% and 15% (Linton, 2004; Morphy et al., 2007; Ohayon, 2004b; Su, Huang, & Chou, 2004). Insomnia is more common in females (20%–21%) than males (13%–17%) (Doi, 2009; Ohayon & Lemoine, 2004b; Peretti-Watel et al., 2009), with women 1.6 times more likely than men to develop insomnia (Li, Wing, Ho, & Fong, 2002). As seen in Figure 6.1, difficulty maintaining sleep increases with age.

Low income, lack of family support, anxiety, depression, pain, or work–family–related stressors are all factors associated with insomnia (Linton, 2004; Morphy et al., 2007; Ohayon, 2004a; Paine, Gander, Harris, & Reid, 2004; Su et al., 2004). Even in the absence of a diagnosable mood disorder, 30% of patients with insomnia exhibit depressive symptoms, and 70% of patients with insomnia exhibit symptoms of anxiety (Ohayon & Reynolds, 2009).

CONSEQUENCES OF INSOMNIA

The consequences of insomnia are multifaceted and include difficulties in physical, emotional, cognitive, and spiritual functioning. Some of the associations between sleep and these multidimensional facets of a person's life are likely to be bidirectional; the presence of the disease contributes to the development of insomnia and conversely, insomnia may contribute to the development of these conditions. (See Chapter 11, Sleep in Medical Disorders, and Chapter 12, Sleep and Psychiatric Disorders.) This bidirectional relationship is particularly evident in the case of depression. Patients with insomnia have a 2.2- to 5.3-fold increased risk of depression (Benca & Peterson, 2008; Staner, 2010; Szklo-Coxe, Young, Peppard, Finn, & Benca, 2010; Taylor, Lichstein, Durrence, Reidel, & Bush, 2005), but insomnia or hypersomnia are also symptoms or consequences of depression. Insomnia is also associated with anxiety and many other psychiatric disorders. (See Chapter 12, Sleep and Psychiatric Disorders.) To further understand the associations between sleep and mood, neuroscientists are proposing that there is sufficient evidence to support the theory of sleep-dependent, emotional brain processing. Specifically, one theory posits that rapid eye movement sleep (REM) modulates affective neural systems (Walker & van der Helm, 2009). In patients with insomnia, the decreased proportion of REM sleep may explain associated mood disturbance, such as depression (Cervena et al., 2004; Feige et al., 2008).

There is considerable evidence documenting the negative health consequences of insomnia.

Insomnia is associated with decreased immune function, altered cognitive performance, altered sense of well-being, and mood disturbance (Altena, Van Der Werf, Strijers, & Van Someren, 2008; Kao et al., 2008; Taylor et al., 2005, 2007). Recent studies have provided evidence that insomnia may be associated with the development of important chronic disorders such as diabetes and hypertension. However, a problem with some of this work is failure to address the contributions of sleep-disordered breathing to difficulty maintaining sleep, a symptom shared by those with insomnia and sleep apnea. There is strong evidence that sleep-disordered breathing is associated with hypertension, cardiac disease, and altered glucose metabolism (Foster et al., 2009; Knutson, Ryden, Mander, & Van Cauter, 2006; O'Connor et al., 2009). (See Chapter 7, Sleep-Related Breathing Disorders.) However, two recent epidemiological reports (Vgontzas, Liao, Bixler, Chrousos, & Vela-Bueno, 2009; Vgontzas, Liao, Pejovic, et al., 2009) suggest that insomnia accompanied with short sleep duration may be associated with type 2 diabetes and hypertension even after statistically controlling for the contributions of sleep-disordered breathing. The mechanism for this relationship is thought to be activation of the hypothalamic–pituitary–adrenal axis. However, research into the associations between insomnia and cardiovascular and metabolic disorders and the mechanisms that explain them are ongoing.

Both experimental and chronic sleep deprivation contribute to decrements in cognitive performance. The effects of insomnia and sleep deprivation on brain function related to cognition occur in two main domains: memory and psychomotor vigilance performance. The hippocampus, a part of the limbic system, plays a critical role in long-term memory imprinting, olfaction, and dimensions of emotional experience. Walker (2008) found that one night of sleep deprivation markedly impairs hippocampal function, imposing a deficit in the ability to commit new experiences to memory. This finding is further supported by other studies of patients with chronic sleep disturbance that document impaired memory function (Bastien et al., 2003; Ellenbogen, Hu, Payne, Titone, & Walker, 2007; Fernandez-Mendoza et al., 2010; Murray & Ranganath, 2007; Ohayon & Lemoine, 2004a; Payne et al., 2009).

Subjects exposed to sleep deprivation exhibit decreased psychomotor and vigilance performance (Drummond, Brown, Salamat, & Gillin, 2004; Edinger, Means, Carney, & Krystal, 2008; Mander et al., 2008; Mednick, Drummond, Arman, & Boynton, 2008; Spiegelhalder, Espie, Nissen, & Riemann, 2008). Further exploration of the neural basis for decreased psychomotor and vigilance performance revealed that insomnia is associated with decreased frontal lobe function that resolves once treated with cognitive behavioral therapy for insomnia (Altena et al., 2008; Altena, Van Der Werf, Strijers et al., 2008; Walker, 2008). Frontal lobe function is important to vigilance, appraisal, and attention.

Researchers believe that insomnia is associated with immune function (Irwin, Clark, Kennedy, Christian Gillin, & Ziegler, 2003; Irwin, Wang, Campomayor, Collado-Hidalgo, & Cole, 2006; Savard, Laroche, Simard, Ivers, & Morin, 2003). For example, patients with normal sleep, compared with those with insomnia, had higher CD3+, CD4+, and CD8+ cells counts that are associated with a stronger immune response in presence of illness (Savard et al., 2003). Further research is underway to evaluate the impact of insomnia and its treatment on immune function and vulnerability to infection.

Etiology and Pathophysiology of Insomnia

The underlying physiological mechanisms for the development of insomnia are thought to be an imbalance of the two-process model of sleep regulation or dysfunction of the arousal systems in the brain. The two-process model states that there are two mechanisms (homeostatic and circadian) that interact to regulate the timing of sleep (Beersma, 2002; Beersma & Gordijn, 2007).

Pathophysiology

Proponents of physiologically based models of insomnia suggest that it develops as the result of imbalance of the sleep- and wake-promoting mechanisms in the brain (Richardson, Carskadon, Orav, & Dement, 1982; Richardson & Tate, 2000).

These mechanisms are regulated by physiologically complex homeostatic and circadian regulatory systems that involve various neurotransmitters and regions of the brain (see Chapter 1, Physiological and Behavioral Aspects of Sleep) (Altena et al., 2008; Drummond, Smith, Orff, Chengazi, & Perlis, 2004; Nofzinger, 2004; Nofzinger et al., 2004; Nofzinger, 2006; Nofzinger, 2008; Riemann et al., 2007; Smith, Perlis, Chengazi, Soeffing, & McCann, 2005). The sleep promoting mechanisms include neurons in the ventrolateral preoptic nucleus of the hypothalamus and melatonin produced by the pineal gland (Bruls, Crasson, Van Reeth, & Legros, 2000; Czeisler, Cajochen, & Turek, 2000; Krauchi & Wirz-Justice, 2001; Kripke, Youngstedt, Rex, Klauber, & Elliott, 2003; Leger, Laudon, & Zisapel, 2004; Nofzinger, 2008; Olde Rikkert & Rigaud, 2001; Riemann, Klein et al., 2002). The wake-promoting mechanism involves several areas of the brain and orexinergic, histaminergic, cholinergic, noradrenergic, and serotonergic neurons (Eggermann et al., 2003; Kilduff & Peyron, 2000; Kiyashchenko et al., 2002; Mignot et al., 2002; Nofzinger, 2008; Oberndorfer, Saletu-Zyhlarz, & Saletu, 2000; Ohayon, 2009). (See Chapter 1, Physiological and Behavioral Aspects of Sleep.)

Recent animal and human studies have also shown that primary insomnia is associated with physiological hyperarousal. This is manifested by high-frequency electroencephalography (EEG) activation, increased whole body and brain metabolism, elevated heart rate, cortisol, temperature, and sympathetic activity, manifested by increased levels of epinephrine and norepinephrine. This theory may explain some of the linkages between insomnia and depression, as well as its linkages with the development of diabetes and cardiovascular conditions (Bonnet & Arand, 2010). It is possible that treatments that reduce arousal may improve insomnia and its negative consequences.

Neurocognitive Model of Insomnia

Some patients with insomnia report that they do not sleep at all, but these patients do sleep when studied using polysomnography. This phenomenon is called sleep state misperception and can be explained by the neurocognitive model of insomnia. This model states that patients with insomnia experience high-frequency EEG activity (cognitive processing) at sleep onset that may blur the patients' perceptions that distinguish sleep and wake (Drummond, Smith et al., 2004; Smith et al., 2005). Further research is needed to fully understand the complexities involved with the patients' perception of their sleep.

Cognitive and Behavioral Mechanisms

Cognitive-behavioral models of insomnia are based on the theory that insomnia develops as the result of conditioning (Espie, 2007; Espie, Inglis, Harvey, & Tessier, 2000; Harvey, Sharpley, Ree, Stinson, & Clark, 2007; Harvey, Tang, & Browning, 2005; Morin, 1993; Morin, Daley, & Ouellet, 2001). Experts generally agree that patients with insomnia develop misperceptions and dysfunctional thinking about sleep that result in the inability to initiate and maintain sleep and to accurately perceive that they have slept (Espie, 2007; Harvey, Stinson, Whitaker, Moskovitz, & Virk, 2008).

Predisposing, Precipitating, and Perpetuating Factors for Insomnia

According to Spielman's (1987) "3-P" model (see Figure 6.2), patients who develop insomnia have premorbid or *predisposing factors* that are

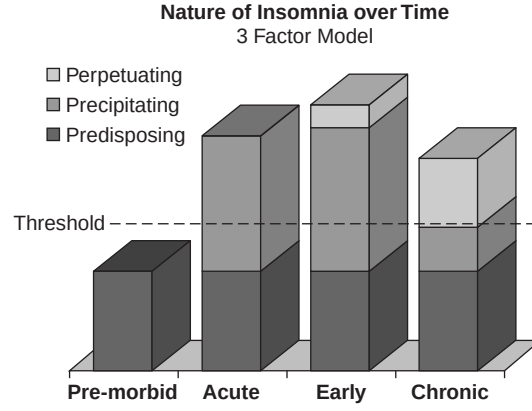

Figure 6.2 ■ Spielman three factor model of insomnia. *Sources: Cognitive Behavioral Treatment of Insomnia: A Session by Session Guide* by M. L. Perlis, C. R. Jungquist, M. T. Smith, and D. Posner, 2005, New York: Springer Science + Business Media. Reprinted with permission.

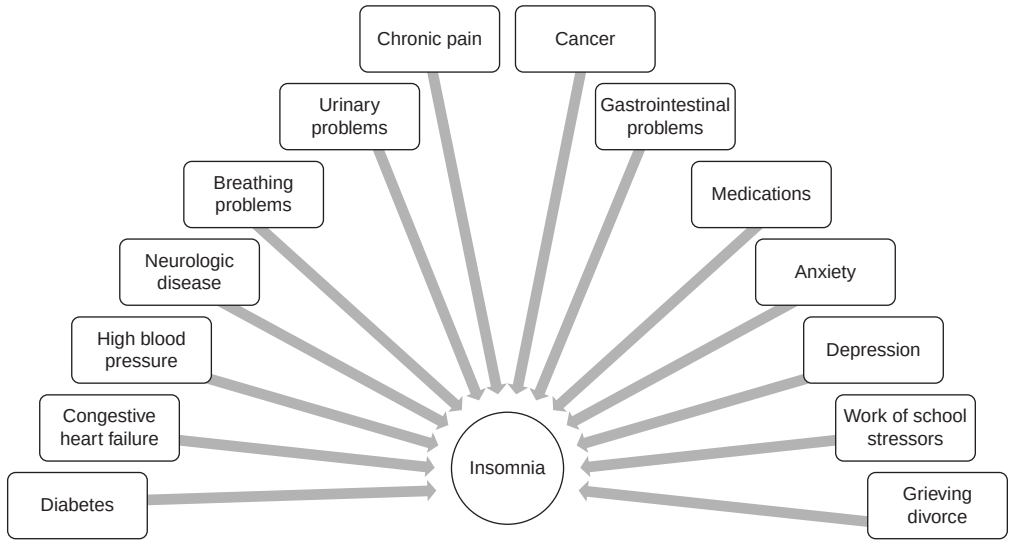

Figure 6.3 ■ Precipitating factors for insomnia.

aggravated by *precipitating factors*. In response, they use compensating strategies to correct sleep loss (Spielman, 1986; Spielman, Caruso, & Glovinsky, 1987). As the result of the failure of compensating strategies, dysfunctional beliefs develop that *perpetuate* the insomnia. Cognitive-behavioral therapy for insomnia, a multimodal treatment that includes sleep restriction, stimulus control, cognitive therapy, and sleep hygiene, is directed at reconditioning the dysfunctional beliefs and correcting the compensating strategies.

Predisposing factors include genetic factors and biological traits of hyperarousal such as hyperactivity, hypervigilance, increased metabolic rate/chronically elevated cortisol levels, or low melatonin levels. Psychological traits include proclivity to worry or ruminate. Social predisposing factors may involve discordance between the individual's sleep schedule and a bed partner or societal demands (Spielman, 1986; Spielman et al., 1987; Spielman, Nunes, & Glovinsky, 1996).

There are numerous medical and psychiatric conditions and medications that are associated with *precipitating* the development of insomnia (see Figure 6.3 and Table 6.3). Chronic insomnia is more common among patients with chronic medical conditions than those without. For example, rates of insomnia are higher in patients who have heart disease (44.1% vs. 22.8%), cancer (41.4% vs. 24.6%), high blood pressure (44.0% vs. 19.3%), neurologic disease (66.7% vs. 24.3%), breathing problems (59.6% vs. 21.4%), urinary problems (41.5% vs. 23.3%), chronic pain (48.6% vs. 17.2%), and gastrointestinal problems (55.4% vs. 20.0%) (Morin et al., 2009; Taylor et al., 2007). Stress may also contribute to the development of insomnia. Examples of stressors include death of a family member, loss of a job, stressors at work, illness, new baby, and move to a different time zone. The most

Table 6.3 ■ *Medications That May Contribute to Insomnia*

■ Calcium channel blockers
■ Corticosteroids
■ Beta blockers
■ Nicotine
■ Xanthines
■ Dopamine agonists
■ Decongestants
■ Antidepressants

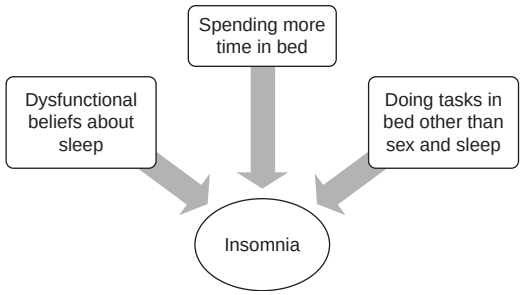

Figure 6.4 ■ Perpetuating factors for insomnia.

common precipitating events are related to family, health, and work-school events (Bastien, Vallieres, & Morin, 2004; Daley et al., 2009).

Perpetuating factors are cognitive and behavioral in nature (see Figure 6.4). Cognitive factors are dysfunctional beliefs and misperceptions about sleep. Common dysfunctional beliefs are thoughts that insomnia will lead to terrible consequences (e.g., loss of job, auto accident, illness, stress). Individuals develop dysfunctional behaviors, such as going to bed early and/or staying in bed for extended periods of time engaged in activities (watching TV, reading, playing cards, eating, paying the bills) that are not related to sleep (Espie, 2007; Spielman et al., 1987), to compensate for sleep loss.

ASSESSMENT OF INSOMNIA

Interview and health history are important tools in diagnosing insomnia (Ohayon & Reynolds, 2009), with a systematic approach critical to developing the diagnosis (see Table 6.4) (Chesson et al., 2000; Sateia, Doghramji, Hauri, & Morin, 2000). Once a patient has identified that he or she has poor sleep, the characteristics of this complaint (e.g., difficulty initiating or maintaining sleep, early morning awakening) should be elicited. When assessing sleep quality, it is important to realize that the meaning of sleep quality among individuals with insomnia and normal sleepers is broadly similar (Harvey et al., 2008). Along with assessing the characteristics of sleep and sleep quality, it is important to address daytime consequences. Careful questioning regarding factors that may contribute to the insomnia (e.g., medical, psychiatric disorders, medications, other sleep disorders, and other substances) may reveal modifiable factors. Identification of perpetuating factors, as well as factors that improve insomnia is necessary. Although not typically necessary to establish the diagnosis of insomnia, a physical examination may reveal physical conditions that may contribute to poor quality sleep.

Sleep diaries and actigraphy are usually used to assess insomnia and evaluate the effects of treatment. (See Chapter 5, Conducting a Sleep Assessment, and Chapter 1, Physiological and Behavioral Aspects of Sleep.) One or both of these methods are always used in insomnia research, often used by sleep specialists when assessing for and treating insomnia, and sometimes used in primary care practice. Validated questionnaires are available for diagnosing and treating insomnia in clinical practice and research. Polysomnography is usually not used to diagnosis of insomnia, except to rule out other sleep disorders, such as sleep-disordered breathing or periodic limb movements during sleep. Polysomnography will only be reimbursable by third-party payers if the patient meets the criteria for a sleep disorder other than insomnia.

Sleep Diaries

Sleep diaries are the standard instruments used to record daily sleep characteristics, including time taken to fall asleep, minutes awake during the night, number of awakenings during sleep, minutes awake before planned-awake time, and quality of sleep, as well as bedtime and arising time. (See Chapter 5, Conducting a Sleep Assessmen and Chapter 1, Physiological and Behavioral Aspects of Sleep.) Diaries are usually completed for 2 weeks, and the averages of the sleep characteristics are computed over that time frame. Therapists calculate sleep efficiency and use it as a measure of therapy success. Sleep efficiency is calculated by dividing the total minutes asleep by the total minutes of time spent in bed and is expressed as a percentage. A sleep efficiency of more than 90% is the goal for most patients. It is best to ask patients to fill out their sleep diary as a first thing in the morning when their recall is the most accurate.

Table 6.4 ■ *Assessment of Insomnia*

Sleep quality

- How would you describe your sleep?
- Do you feel rested when you awaken in the morning?
- Difficulty initiating sleep: How long does it take you to fall asleep?
- Difficulty maintaining sleep: How often do you awaken? What awakens you?
- Early morning awakening: Do you awaken in the morning before you are ready to get up? Can you get back to sleep?

Duration of sleep complaint

Consequences of poor sleep

- Fatigue?
- Sleepy?
- Difficulty with memory, learning, or other performance difficulty?
- Mood problems?

Typical sleep patterns (diary helpful)

- Bedtime
- Rising time
- Number of awakenings
- Daytime napping
- Variation between weekends and weekdays

Contributing factors

- Medical disorders and symptoms (e.g., pain, dyspnea, nocturia)
- Psychiatric disorders (e.g., depressive symptoms, anxiety)
- Medications
- Substances (caffeine, alcohol, illicit drugs, over-the-counter medications, herbs)
- Sleep disorders and their symptoms (e.g., snoring, leg movements)
- Other predisposing, precipitating, perpetuating factors (see Figures 6.3 and 6.4)

Actigraphy

Wrist actigraphs, motion sensors from which sleep can be inferred, are objective measure of activity/rest and are good surrogate measures of sleep when used with validated electronic sleep scoring algorithms. They are particularly helpful in examining changes in sleep over time (Ancoli-Israel et al., 2003; Paquet, Kawinska, & Carrier, 2007; Sadeh & Acebo, 2002; Sivertsen et al., 2006). (See Chapter 5, Conducting a Sleep Assessment.) Third party reimbursement for actigraphy has improved, but the cost of the actiwatches remains an obstacle to the use of actigraphy in insomnia assessment and evaluation in the clinical setting.

TREATMENT OF INSOMNIA

Treatment of insomnia is focused on addressing precipitating and perpetuating factors and preventing the development of chronic insomnia. The most frequently used and efficacious treatments are hypnotic medications and CBT-I. Many patients prefer to use nonpharmacologic behavioral interventions. These treatments are the most effective in providing sustained relief of symptoms (Jacobs, Pace-Schott, Stickgold, & Otto, 2004; Morin et al, 2006; Morin et al., 1999; Smith et al., 2002).

Acute Insomnia

Patients most frequently use over-the-counter sleep aids (National Sleep Foundation, 2008) to treat acute insomnia. Medical providers frequently prescribe hypnotics for the first-line treatment for acute insomnia, despite evidence that behavioral interventions are safer and more effective (Morin et al., 2006). One reason is the lack of providers trained in CBT-I.

Pharmacotherapy

Food and Drug Administration approved drugs used for insomnia targets gamma-aminobutyric acid (GABA) and benzodiazepine (BZ) receptors (Hausken, Furu, Skurtveit, Engeland, & Bramness, 2009). Other agents target 5HT2, histamine, and alpha-adrenergic receptors. The classes of medications used for insomnia are BZs, barbiturates, nonbarbiturate hypnotics, antihistamines, and antidepressants (see Table 6.5). Deciding on the appropriate class of medication for insomnia requires consideration of not only nocturnal symptoms/type of insomnia but also the daytime consequences of the insomnia and the adverse effects of the medication. Prescription of hypnotics is based on the nature of the sleep complaint, patients' comorbid conditions, provider professional specialty, antipsychotic use, and/or opioid use (Hausken et al., 2009).

Table 6.5 ■ *Hypnotic Medications*

Drug	Dose Range	Half-Life
Nonbarbiturate Hypnotics		
Zolpidem	10 mg	2.5 hours
Zolpidem CR	12.5 mg	2.8 hours
Zaleplon	5–20 mg	1 hour
Eszopiclone	1–2 mg in elderly	6 hours in adults, 9 hours in elderly
Chloral hydrate	500 mg	7–10 hours
Ramelteon	8 mg	1–2.6 hours
Benzodiazepines		
Estazolam	1–2 mg	10–24 hours
Flurazepam HCl	15–30 mg	2.3 hours
Triazolam	0.25–0.5 mg	1.5–5.5 hours
Temazepam	7.5–30 mg	8.8 hours
Antidepressants		
Trazodone	25–150 mg	IR 7 hours: ER 10 hours
Mirtazapine	15–30 mg	37 hours females; 26 hours males
Amitriptyline	25–100 mg	9–25 hours
Nortriptyline	25–100 mg	15–39 hours

Source: Microdex by Thomson Health Care, 2007, retrieved July 14, 2007, from http://thomsonhc.com/hcs/librarian. Adapted with permission.

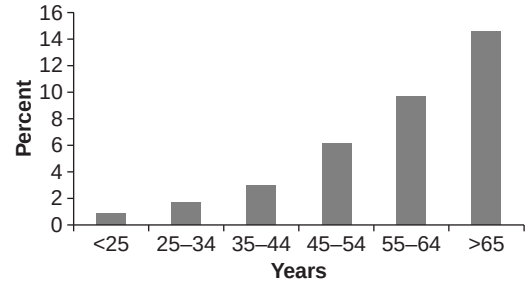

Figure 6.5 ■ Prevalence of sleep medication use by age. *Source:* "Pain Sensitivity, Depression, and Sleep Deprivation: Links With Serotoninergic Dysfunction" by M. M. Ohayon, 2009, *Journal of Psychiatric Research, 43*(16), pp. 1243–1245. Reprinted with permission.

The use of hypnotics increases with age (see Figure 6.5). Although use of hypnotic medications often improves nighttime sleep, the daytime complaints that occur as the result of sleep disruption do not always diminish with the use of medications (Vignola, Lamoureux, Bastien, & Morin, 2000), and patients often complain of daytime dysfunction, such as drowsiness resulting from the medications (Ohayon, 2009).

Nonbarbiturate Hypnotics

Zolpidem is a member of the imidazopyridine class of hypnotics, binds to the BZ receptor subunit of the GABA-A receptor and is metabolized by the cytochrome P450 system. It is known to interact with other central nervous system depressants. Formulations include short acting and controlled release. The controlled release version delivers the medication via two layers. The first layer dissolves immediately, and the second layer dissolves slowly to assist with sleep maintenance. This medication is widely prescribed because it has low potential to cause negative daytime sequela, and there are few adverse effects. The risk of lethality from this drug is low unless taken in combination with other central nervous system depressants (Thomson Healthcare, 2007).

Zaleplon is a nonbenzodiazepine hypnotic from the pyrazolopyrimidine class. The side effects profile is comparable to zolpidem and it is thought to be safe and well tolerated with little if any daytime carryover effects (Thomson Healthcare, 2007).

Eszopiclone is a nonbenzodiapine hypnotic drug and is thought to be a GABA receptor agonist. The usual dose ranges between 2 and 3 mg in adults and 1 and 2 mg in older adults. The adverse effect profile is minimal. The short half-life of 6 hours leads to a minimal risk of daytime sequelae (Thomson Healthcare, 2007).

Chloral hydrate is a central nervous system depressant, approved by the FDA for the treatment of insomnia. Due to the long half-life, it is likely to cause next day residual effects. The exact mechanism of action is unknown, but its actions are similar to barbiturates. Ingestion of more than 1.5 g is life threatening. This medication should be used cautiously in patients with suicidal tendencies and/or depression (Thomson Healthcare, 2010).

Ramelteon is a melatonin agonist. It acts at the melatonin M1 and M2 receptors and improves sleep by improving the circadian rhythm. It does not have affinity to other receptors that affect the action of hypnotics (e.g., GABA). The peak effect occurs within an hour. Lethality risk from the medication is low. In clinical trials, doses up to 160 mg were taken without negative effect. Adverse effects are also minimal. Increase in prolactin levels were not associated with reproductive effects (Richardson & Wang-Weigand, 2009; Thomson Healthcare, 2007; Thomson Healthcare, 2010).

Benzodiazepines

Benzodiazepines (BZs) have been used to treat insomnia for many years. Although effective, BZs are associated with tolerance, addiction, abuse, and daytime dysfunction, such as dizziness and somnolence. This may be particularly so for older adults who may be at increased risk for falls. With the increasing availability of the safer nonbenzodiazepine hypnotics, prescribing of BZs has decreased. However, BZs may be useful for people whose insomnia is associated with anxiety because they have anxiolytic effects (Vignola et al., 2000).

Estazolam is a short or immediate acting central nervous system depressant that is FDA approved for insomnia. Common adverse effects are asthenia, dizziness, and coordination problems. Its peak effect occurs at around 2 hours (Thomson Healthcare, 2010).

Flurazepam hydrochloride is a long-acting BZ that is FDA approved for insomnia. Its peak effect occurs within 30–60 minutes. This medication has active metabolites that are active for up to 120 hours and therefore should be used very cautiously in elderly as well as patients with renal disease (Thomson Healthcare, 2010).

Triazolam is a central nervous system depressant that has been used for many years. Due to adverse effects, it was taken off the market in Europe but remains available in the United States. Its peak effect occurs within 2 hours, and it is metabolized through the P450 CYP3A enzyme system and interacts with grapefruit juice (Thomson Healthcare, 2010).

Temazepam is a short to immediate action BZ that is FDA approved for the treatment of insomnia. Its peak effect occurs at 1.5 hours. Next day, sequelae are minimal (Thomson Healthcare, 2010). Temazepam should be used cautiously in the elderly due to the increased risk of falls (Glass, Sproule, Herrmann, & Busto, 2008).

Antidepressants

Although they are not approved as hypnotic medications, the somnolence-producing effects of some antidepressants may be useful in treating insomnia, especially when patients have comorbid psychiatric comorbidities. *Trazodone*, a triazolopyridine serotonergic antidepressant, has been used for many years due to sedating properties. Dosages ranging between 25 and 150 mg have been found effective for insomnia with and without cognitive behavioral therapy for insomnia and with and without comorbid depression (Bertschy et al., 2005; Mendelson, 2005; Saletu-Zyhlarz, Anderer, Arnold, & Saletu, 2003; Wichniak et al., 2007; Zavesicka et al., 2008). Noted adverse effects include next-day sedation, dizziness, and psychomotor impairment (Mendelson, 2005).

Mirtazapine is a potent antagonist at the 5-HT2 and 5-HT3 receptors. It is thought to have central noradrenergic effects and produces sedating effects by blocking the histamine H1 receptor (Thomson Healthcare, 2010). Mirtazapine was

effective for insomnia and nausea in patients with cancer (Biglia et al., 2007; Kim et al., 2008). Considerable weight gain and decreased daytime performance are adverse effects that preclude its widespread use for insomnia (Aslan, Isik, & Cosar, 2002; Schmid et al., 2006; Wingen, Bothmer, Langer, & Ramaekers, 2005). There is some evidence that mirtazapine may worsen sleep-disordered breathing through its effects on weight gain (Brunner, 2008; Carley, Olopade, Ruigt, & Radulovacki, 2007; Marshall et al., 2008). It is also associated with orthostatic hypotension (Sennef, Timmer, & Sitsen, 2003).

Stimulants

Stimulant drugs, such as modafinil and armodafinil (see Chapter 9, Narcolepsy) are usually not used to treat insomnia because insomnia is an adverse effect of these medications (Hou, Langley, Szabadi, & Bradshaw, 2007; Jha et al., 2008; Kumar, 2008; Lindsay, Gudelsky, & Heaton, 2006). However, it may be effective in treating daytime sleepiness/fatigue in people with insomnia and may assist with compliance to sleep restriction (Perlis et al., 2004) in patients who are undergoing sleep restriction therapy as a component of CBT-I.

Over-the-Counter Medications and Herbal Supplements

Diphenhydramine has been used for years to treat the inability to initiate sleep despite the lack of sufficient evidence to support its use. Diphenhydramine's soporific action is thought to occur as the result of blocking histamine attachment to cerebral histamine wake–promoting (H1) receptors (Tashiro et al., 2008; Thomson Healthcare, 2010). Modest, but not statistically significant, effects on sleep resulted from administration of 50 mg of diphenydramine in one study (Glass et al., 2008). In the second study, dose-dependent effects were associated with administration of three doses (12.5, 25, 50 mg) for sleep initiation in patients with psychiatric comorbidities (Kudo & Kurihara, 1990). Diphenhydramine's negative effects on memory, performance, and increasing risk of falls are similar to the effects of the BZs, temazepam, and lorazepam (Glass, Sproule, Herrmann, Streiner, & Busto, 2003; Glass et al., 2008; Turner, Handford, & Nicholson, 2006). Diphenhydramine also has anticholinergic effects and should be used very cautiously in the elderly due to its negative effects on cognition, performance, increased risk of falls, decreased daytime function, and impaired driving performance (Basu, Dodge, Stoehr, & Ganguli, 2003; Glass et al., 2008; Mansfield, Mendoza, Flores, & Meeves, 2003; Roehrs, Turner, & Roth, 2000; Weiler et al., 2000).

Melatonin is a synthetically produced endogenous substance that has been available in the United States since the early 1990s. It is an herb and its production and sale are not regulated by the FDA. Beta-methyl-6-chloromelatonin at 20 mg, 50 mg, and 100 mg are effective for sleep onset insomnia (Zemlan et al., 2005). More recently, a prolonged-release formulation of melatonin 2 mg was found to be effective for sleep onset insomnia, but not associated with next day impaired psychomotor performance, drug tolerance, or rebound symptoms when discontinued (Lemoine, Nir, Laudon, & Zisapel, 2007; Luthringer, Muzet, Zisapel, & Staner, 2009; Suresh Kumar, Andrade, Bhakta, & Singh, 2007; Wade et al., 2007; Wade & Downie, 2008; Zisapel, 2009). Supplemental melatonin may be a good choice for the elderly with insomnia because endogenous melatonin decreases with age (Leger et al., 2004; Zhdanova et al., 2001).

There is little evidence supporting the effectiveness or the adverse effects of herbal supplements commonly used for insomnia. Commonly used agents including lavender, chamomile, valerian, melatonin, SAMe, and tryptophan. (See Chapter 15, Racial/Ethnic Health Disparities and Sleep Disorders.)

Valerian-hops, an herb, is as effective as diphenhydramine for mild insomnia, and did not produce significant adverse effects in one study. However, other investigators found that the effects of valerian were no better than placebo (Glass et al., 2003; Morin, Koetter, Bastien, Ware, & Wooten, 2005). Doses obtained from the valerian plant range from 400 to 450 mg and valerian root range between 200 and 1,200 mg. The

mechanism of action is thought to be secondary to depression of the central nervous system and relaxation of smooth muscles (Thomson Healthcare, 2010).

SAMe (s-adenosylmethionine) is a naturally occurring substance. The supplement is thought to produce its effects by increasing dopamine and serotonin in the central nervous system. The usual daily dose is 600–800 mg for insomnia, depression, arthritis, fibromyalgia, and liver disease (Thomson Healthcare, 2010). However, there is no evidence supporting its effectiveness.

The *Chamomile flower* is thought to have antianxiety, antispasmodic, and antiinflammatory properties (Thomson Healthcare, 2010). Although it is used for insomnia, only one animal study suggests that it may improve sleep latency (Shinomiya et al., 2005). Chamomile is known to potentiate the effects of anticoagulant therapy (Thomson Healthcare, 2010), and its concomitant use may lead to bleeding. Although *lavender* is thought to be a sedative hypnotic (Thomson Healthcare, 2010) and it is readily available, there is insufficient evidence as to its safety or efficacy for the treatment of insomnia (Lee & Lee, 2006).

Tryptophan is an amino acid and naturally occurring substance in the human diet and is a precursor to serotonin (Thomson Healthcare, 2010). L-tryptophan is available as an over-the–counter supplement and is promoted for treatment of insomnia and depression. Studies of tryptophan conducted over more than 30 years have resulted in mixed evidence (Demisch, Bauer, & Georgi, 1987; Gnirss, Schneider-Helmert, & Schenker, 1978; Hudson, Hudson, Hecht, & MacKenzie, 2005; Meolie et al., 2005; Riemann et al., 2002; Schneider-Helmert & Spinweber, 1986) for its effectiveness. Overall, it appears that L-tryptophan may have some positive effects on decreasing time to sleep and time awake during the night, but it should be used cautiously as it interacts with medications having serotonin properties. Serious adverse effects such as eosinophilia-myalgia syndrome and serotonin syndrome have been seen with doses between 3 and 15 g daily. The usual hypnotic dose is 1–2 g at bedtime.

Evidence on the use of herbs and supplements for insomnia is very limited and does not support the effectiveness or the safety of using these agents. Additional concerns are the inconsistencies in preparations due to the absence of FDA regulation of these herbs/supplements. Certain supplements such as tryptophan have been found to cause serious interactions with psychiatric medications (National Institutes of Health, 2005) and may interact with prescription or nonprescription medications. Therefore, it is important to ask patients about their use of these substances, educate them about their effects and potential drug interactions, and provide alternatives where appropriate.

Chronic Insomnia

Behavioral for insomnia include CBT-I, exercise, music, and warm baths, among others. CBT-I, a multimodal group or individual-based treatment, has been the most widely studied and has been shown to be efficacious in numerous studies.

Cognitive Behavioral Therapy for Insomnia (CBT-I)

CBT-I employs a highly structured treatment protocol that addresses maladaptive behaviors and strategies as well as negative cognitions about insomnia and its daytime consequences. CBT-I is efficacious in both primary and comorbid insomnia (Jungquist et al., 2010; Morin et al., 2006; Morin et al., 1999; National Institutes of Health, 2005; Smith & Perlis, 2006). See Table 6.6 for components of therapy. Table 6.7 includes instructions for patients on sleep hygiene. CBT-I is as effective as hypnotic medications and its effects are more durable (Jacobs et al., 2004; Morin et al., 2006; Smith et al., 2002). The effects of CBT-I extend beyond 6 months after treatment and in most situations, sleep continues to improve for months post treatment. CBT-I is also effective in reducing the need for hypnotic medications.

Although the elements of CBT-I differ slightly between therapists and both group and individual formats may be used, the staple components

Table 6.6 ■ *Cognitive Behavioral Therapy for Insomnia*

Treatment Modality	Description
Stimulus Control Therapy	A set of instructions designed to reassociate the bed/control bedroom with sleep and to reestablish a consistent therapy sleep-wake schedule: (1) Go to bed only when sleepy; (2) get out of bed when unable to sleep; (3) use the bed/bedroom for sleep only (no reading, watching TV, etc); (4) arise at the same time every morning; (5) no napping.
Sleep Restriction Therapy	A method designed to curtail time in bed to restrict actual amount of sleep time. For example, if a patient therapy reports sleeping an average of 6 hours per night, out of 8 hours spent in bed, the initial recommended sleep window (from lights out to final arising time) would be restricted to 6 hours. Periodic adjustments to this sleep window are made contingent upon sleep efficiency, until an optimal sleep duration is reached.
Relaxation Training	Clinical procedures aimed at reducing somatic training tension (e.g., progressive muscle relaxation, autogenic training) or intrusive thoughts at bedtime (e.g., imagery training, meditation) interfering with sleep.
Cognitive Therapy	Cognitive psychological methods aimed at challenging and changing misconceptions about sleep and faulty beliefs about insomnia and its perceived daytime consequences. Other cognitive procedures may include paradoxical intention or methods aimed at reducing or preventing excessive monitoring of and worrying about insomnia and its correlates/consequences.
Sleep Hygiene	General guidelines about health practices (e.g., diet, education, exercise, substance use) and environmental factors (e.g., light, noise, temperature) that may promote or interfere with sleep. This may also include some basic information about normal sleep and changes in sleep patterns with aging.
Cognitive Behavioral Therapy	A combination of any of the above behavioral (e.g., behavior stimulus control, sleep restriction, relaxation) and cognitive therapy procedures.

Source: "Psychological and Behavioral Treatment of Insomnia: Update of the Recent Evidence (1998–2004)," by C. M. Morin, R. R. Bootzin, D. J. Buysse, J. D. Edinger, C. A. Espie, and K. L. Lichstein, 2006, *Sleep, 29*(11), pp. 1398–1414. Used with permission.

of CBT-I are sleep restriction, stimulus control, sleep hygiene, and cognitive therapy (Harvey et al., 2005; Harvey et al., 2007; Morin et al., 2006; Morin et al., 1999; Perlis, Jungquist, Smith, & Posner, 2005). Some therapists include relaxation therapy (e.g., progressive muscle relaxation and biofeedback) directed at decreasing somatic tension and arousal. Other adjunctive therapies (e.g., visual imagery and meditation) focus on decreasing the intrusion of negative thoughts and worry.

Although patients generally report high levels of satisfaction with CBT-I and it is usually efficacious, patients with severe depressive symptoms and those who report that their sleep duration is shorter than 4 hours are more likely to drop out of therapy (Ong, Shapiro, & Manber, 2009).

Therapists and Settings. CBT-I was developed and tested by psychologists and requires structured training, especially because of the potential for adverse effects and the potential need to address psychiatric comorbidities, such as anxiety or depression. Health care providers of various backgrounds, including psychologists, nurses, physicians, social workers, and counselors, have been trained in the delivery of CBT-I, but access to CBT-I continues to be poor due to the shortage of qualified, trained therapists. Currently, CBT-I is provided either to individuals or groups in the sleep disorders center setting, but there is growing evidence of its efficacy and effectiveness when delivered in primary care settings and to patients with comorbid conditions such as depression or cancer.

Espie (2009) advocated for a "stepped" approach to CBT-I, leveled according to the complexity of the insomnia, by health care professionals from a variety of disciplines, including nurses. Abbreviated forms of CBT-I have been delivered with positive results in a variety of settings, including primary care. However, the success of this approach may be limited by the lack of knowledge about sleep in general among many health care providers, including

Table 6.7 ■ *Instructions for Patients Regarding Sleep Hygiene*

1. **Sleep only as much as you need to feel refreshed during the following day.**
   Restricting your time in bed helps to consolidate and deepen your sleep. Excessively long times in bed lead to fragmented and shallow sleep. Get up at your regular time the next day, no matter how little you slept.
2. **Get up at the same time each day, 7 days a week.**
   A regular wake time in the morning leads to regular times of sleep onset and helps to set your "biological clock."
3. **Exercise regularly.**
   Schedule exercise times so that they do not occur within 3 hours of when you intend to go to bed. Exercise makes it easier to initiate sleep and deepen sleep.
4. **Make sure your bedroom is comfortable and free from light and noise.**
   A comfortable, noise-free sleep environment will reduce the likelihood that you will wake up during the night. Noise that does not awaken you may also disturb the quality of your sleep. Carpeting, insulated curtains, and closing the door may help.
5. **Make sure that your bedroom is at a comfortable temperature during the night.**
   Excessively warm or cold sleep environments may disturb sleep.
6. **Eat regular meals and do not go to bed hungry.**
   Hunger may disturb sleep. A light snack at bedtime (especially carbohydrates) may help sleep, but avoid greasy foods.
7. **Avoid excessive liquids in the evening.**
   Reducing liquid intake will minimize the need for nighttime trips to the bathroom.
8. **Cut down on all caffeine products.**
   Caffeinated beverages and foods (coffee, tea, cola, chocolate) can cause difficulty falling asleep, awakenings during the night, and shallow sleep. Even caffeine early in the day can disrupt nighttime sleep.
9. **Avoid alcohol, especially in the evening.**
   Although alcohol helps tense people fall asleep more easily, it causes awakenings later in the night.
10. **Smoking may disturb sleep.**
    Nicotine is a stimulant. Try not to smoke during the night when you have trouble sleeping.
11. **Do not take your problems to bed.**
    Plan some time earlier in the evening for working on your problems or planning the next day's activities. Worrying may interfere with initiating sleep and produce shallow sleep.
12. **Do not try to fall asleep.**
    This only makes the problem worse. Instead, turn on the light, leave the bedroom, and do something different like reading a book. Do not engage in stimulating activity. Return to bed only when you are sleepy.
13. **Put the clock under the bed or turn it so that you cannot see it.**
    Clock watching may lead to frustration, anger, and worry, which interfere with sleep.
14. **Avoid naps.**
    Staying awake during the day helps you to fall asleep at night.

nurses. This underscores the importance of including sleep-related information in all levels of nursing curricula. Advanced practice nurses (APNs), in particular, may be well suited to delivering CBT-I with appropriate training. Postgraduate course work is available to APNs in CBT-I (see Chapter 23, The Role of Advanced Practice Nurses (APNs) in Specialized Sleep Practice) and several treatment guides have recently been published to guide the therapist in delivering CBT-I (Edinger, 2008; Morin & Espie, 2003; Perlis et al., 2005).

Self-Help Resources

Over the past 10 years, the number of self-help books and Web sites offering CBT-I has increased. There is sufficient evidence to show Web-based and self-help bibliotherapy are effective (Belleville, Guay, Guay, & Morin, 2007;

Riley, Mihm, Behar, & Morin, 2010; Ritterband et al., 2009; van Straten & Cuijpers, 2009; van Straten, Cuijpers, Smit, Spermon, & Verbeek, 2009). However, little is known about the effects of self-help approaches compared with direct treatment by experienced therapists.

Complementary/Alternative Therapy for Insomnia

Patients use many complementary therapies for insomnia, such as exercise, relaxation, music, acupuncture, bright light therapy, hypnosis, relaxation, and body temperature manipulation. Although these are increasing in popularity, empirical evidence in support of efficacy is lacking for many of these methods. (See Chapter 14, Complementary and Alternative Medicine and Sleep.)

Exercise. In general, research findings on the effects of exercise on sleep are equivocal. However, several studies have shown small effects of exercise on sleep. Elavsky and McAuley (2007) found no significant effects of exercise on sleep quality after 4 months of moderate walking or yoga in middle-aged women. Results of studies of exercise in older adults (>55 years) also have been equivocal to support exercise as a primary therapy for insomnia (Elavsky & McAuley, 2007; King et al., 2008; Montgomery & Dennis, 2002; Oda, 2001; Payne, Held, Thorpe, & Shaw, 2008).

Relaxation. There is sufficient evidence that relaxation techniques improve insomnia symptoms and they can be used as solo treatments or components of CBT-I in all ages regardless of comorbidity (Friedman, Bliwise, Yesavage, & Salom, 1991; Greeff & Conradie, 1998; Gustafson, 1992; Jacobs et al., 1993; Lichstein & Johnson, 1993; Means, Lichstein, Epperson, & Johnson, 2000; Ziv, Rotem, Arnon, & Haimov, 2008). However, relaxation appears to be more effective for decreasing time to sleep than decreasing the number of awakenings (Jacobs et al., 1993).

Music Therapy. Music therapy may also be useful in improving insomnia symptoms. Ziv et al. (2008) found that music therapy was more effective for older adults than progressive muscle relaxation in improving sleep quality and decreasing anxiety.

Body and Skin Temperature Manipulations. As one of the natural sleep promoting effects of the circadian rhythm is a change in body core temperature, therapies using temperature manipulation may be effective strategies in treating insomnia. This theory is based on the fact that slow-wave sleep increases as core body temperature decreases (Raymann, Swaab, & Van Someren, 2007; Raymann & Van Someren, 2007; van den Heuvel, Ferguson, & Dawson, 2006). Difficulty maintaining sleep is thought to be the result of dysfunction of the thermoregulatory system that results in nocturnally elevated core body temperature (Lack, Gradisar, Van Someren, Wright, & Lushington, 2008). Several studies have found that a warm bath taken 60–90 minutes before bedtime (Kanda, Tochihara, & Ohnaka, 1999; Liao, 2002; Sung & Tochihara, 2000) resulted in shorter sleep latency. However, others (Liao) found no effects on polysomnographic measures of sleep. It is thought that peripheral vasodilation results in heat loss from the body and reduces core body temperature. Similarly, insomnia symptoms diminished when the dialysate temperature was reduced in patients undergoing hemodialysis (Parker, Bailey, Rye, Bliwise, & Van Someren, 2008). It is possible that the decrease in core body temperature may decrease sympathetic activation and assist in maintaining the normal circadian rhythm-driven changes in body temperature.

CONCLUSIONS

Insomnia is a highly prevalent condition that is known to be associated with important pathophysiological, cognitive, and functional consequences. Adults of all ages and all states of health frequently suffer from insomnia. Therefore, nurses are likely to encounter patients with insomnia in many health care and home settings, including primary care, specialty practices, and acute and long-term care settings. Assessment for insomnia should routinely be incorporated into nursing practice in these settings. Although

hypnotics are of some use, CBT-I offers the best long-term results in improving sleep and the daytime sequela resulting from poor sleep. Nurse at all levels play an important role in assessment of insomnia, education about the importance of insomnia, and offering strategies for prevention of chronic insomnia (Krishnan & Hawranik, 2008). Nurses with advanced training can be effective therapists in the delivery of CBT-I (Jungquist et al., 2010). Effective diagnosis, prevention, and treatment of insomnia is likely to have important effects on reducing its deleterious effects on pathophysiology, functional performance, and quality of life.

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Sleep-Related Breathing Disorders

7

Amy M. Sawyer and Terri E. Weaver

Obstructive sleep apnea, the most common sleep-related breathing disorder, was first described in 1976 after the first symposium on sleep-related respiratory problems held in Italy (Guilleminault, Tilkian, & Dement, 1976). The condition included snoring, abnormal nighttime behaviors, altered states of consciousness during sleep, "fogginess" upon awakening from sleep, enuresis or nocturia, and frequent unexplained arousals (Guilleminault et al., 1976). Since that time, there has been a tremendous increase in public and scientific awareness of sleep-related breathing disorders and evidence of the high prevalence and negative health-related consequences associated with these disorders. The purpose of this chapter is to provide a comprehensive description of sleep-related breathing disorders, including obstructive sleep apnea (OSA), central sleep apnea (CSA), and Cheyne-Stokes breathing (CSB). The chapter will address the characteristics, epidemiology, related factors and consequences, pathophysiology, assessment, diagnosis, and treatment of these disorders. Implications for nursing are discussed.

SLEEP-RELATED BREATHING DISORDERS

Sleep-related breathing disorders include CSA, OSA, sleep-related hypoventilation/hypoxemic syndromes, and sleep-related hypoventilation/hypoxemia due to other medical conditions (see Table 7.1) (American Academy of Sleep Medicine, 2005). Sleep-related breathing disorders are characterized by partial or complete cessation of respiration during sleep, oxyhemoglobin desaturation and sleep fragmentation. Each has unique underlying mechanism(s) and presentations that are discussed separately in the following narrative.

Obstructive Sleep Apnea

OSA is the most prevalent sleep-related breathing disorder. It is characterized by partial and/or complete upper pharyngeal airway closures with persistent efforts to breathe that result in intermittent oxyhemoglobin desaturation and sleep fragmentation. Complete (apneas) or partial (hypopneas) airway obstructions lead to reduction or cessation of airflow. In OSA, respiratory events are associated with increased respiratory effort, as evidenced by increased thoracoabdominal motion. Mixed apneas (apneas associated both with and without respiratory effort) are also common (see Table 7.2). Although respiratory events may occur in any stage of sleep, apneas and hypopneas are more common in Stage N1 and Stage N2 (NREM) and REM sleep than in Stage N3 (NREM). When respiratory events occur during REM sleep, they are usually longer and associated with lower oxyhemoglobin saturation levels. Respiratory events are usually associated with arousals from sleep that terminate the respiratory events. Hypoxia and arousals from sleep lead to daytime symptoms (e.g., sleepiness, fatigue), impaired cognition and daily function, and significant morbidity and mortality (Patil, Schneider, Schwartz, & Smith, 2007; Weaver, 2001).

Table 7.1 ■ *Nosology of Sleep-Related Breathing Disorders*

Categorization	Disorder
Central sleep apnea syndromes	Primary central sleep apnea Central sleep apnea due to Cheyne-Stokes breathing pattern Central sleep apnea due to high-altitude periodic breathing Central sleep apnea due to medical condition not Cheyne-Stokes Central sleep apnea due to drug or substance Primary sleep apnea of infancy
Obstructive sleep apnea syndromes	Obstructive sleep apnea, adult Obstructive sleep apnea, pediatric
Sleep-related hypoventilation/hypoxemic syndromes	Sleep-related nonobstructive alveolar hypoventilation, idiopathic Congenital central alveolar hypoventilation syndrome
Sleep-related hypoventilation/hypoxemia due to medical condition	Sleep-related hypoventilation/hypoxemia due to pulmonary parenchymal or vascular pathology Sleep-related hypoventilation/hypoxemia due to lower airways obstruction Sleep-related hypoventilation/hypoxemia due to neuromuscular and chest wall disorders
Other sleep-related breathing disorder	Sleep apnea/sleep-related breathing disorder, unspecified

Source: International Classification of Sleep Disorders (2nd ed.) by American Academy of Sleep Medicine, 2005, Westchester, IL: Author.

Table 7.2 ■ *Commonly Used Terms and Definitions Related to Sleep-Disordered Breathing*

Apnea: Cessation or near cessation (>70% reduction) of airflow for at least 10 seconds using a valid measure of airflow (i.e., thermistor, thermocouple, or nasal pressure transducer).

Hypopnea: Greater than 30% reduction of amplitude in thoracoabdominal movement or airflow as compared to baseline with a greater than 4% decrement in oxyhemoglobin saturation.

Mixed Apnea: Airway closures that are initially without respiratory effort followed by a gradual increase in effort to breathe against the occluded upper airway.

Respiratory-Effort-Related Arousal (RERA): Episodes that include a clear drop in inspiratory airflow, increased respiratory effort, and a brief change in sleep state (arousal) but do not meet criteria for an apnea or hypopnea.

Apnea Hypopnea Index (AHI): The number of apneas and hypopneas divided by the number of hours of sleep. Severity of OSA is commonly determined by AHI. Mild OSA is defined as an AHI of 5–15 events per hour; moderate OSA is defined as an AHI of 15–30 events per hour of sleep; severe OSA is defined as an AHI of greater than 30 events per hour of sleep.

Respiratory Disturbance Index (RDI): The number of apneas, hypopneas, and other scoreable respiratory events such as RERAs divided by the number of hours of sleep.

Periodic Limb Movement (PLM): All or part of a rapid partial flexion of the foot at the ankle, extension of the big toe, and partial flexion of the knee and hip that occurs during sleep. The movements typically occur every 20–90 seconds in a relatively consistent pattern and last approximately 0.5–5.0 seconds.

Source: International Classification of Sleep Disorders: Diagnostic and Coding Manual (2nd ed.) by American Academy of Sleep Medicine, 2005, West Chester, IL: Author.

Cheyne-Stokes Breathing and Central Sleep Apnea

CSB and CSA are characterized by instability of the respiratory control system that leads to apneic periods without respiratory effort. CSB is associated with cyclical fluctuations in breathing in which central apneas or hypopneas alternate with periods of hyperpnea (increase in respiratory depth and rate) in a gradual waxing/waning pattern (American Academy of Sleep Medicine, 2005). CSB and CSA are often associated with severe heart failure (HF) or

cerebrovascular disease. CSA and CSB often occur during non-REM sleep, but may also occur during wake, especially in people with HF. The CSB diagnosis is established with polysomnography (PSG) conducted in the laboratory (see Table 7.3). OSA frequently co-occurs with CSB (and the occurrence of mixed apneas) because the cyclical fluctuations in respiratory effort associated with CSB predispose the upper airway to instability and obstruction due to reduced stimulation of the upper airway dilator muscles (American Academy of Sleep Medicine Task Force, 1999).

CSA is characterized by recurrent apneas without upper airway obstruction. The absence of upper airway obstruction differentiates CSA and CSB from OSA. Metabolic or neuromuscular disorders contribute to hypercapneic CSA, a sleep hypoventilation syndrome (see Table 7.1).

Normocapneic/hypocapneic CSA may be idiopathic or secondary to CSB or exposure to high altitudes. At high altitudes, hypoxia leads to hyperventilation, which, in turn leads to low carbon dioxide levels and alkalosis. Hypocapnia leads to ventilatory inhibition (Eckert, Jordan, Merchia, & Malhotra, 2007) and respiratory depression (American Academy of Sleep Medicine, 2005). CSA resolves with descent to lower altitudes and increased availability of oxygen. PSG should be conducted in a laboratory at a similar altitude to the altitude in which patients live in order to be sensitive to the degree of CSA (Patz et al., 2006). Conversely, high-risk individuals who plan to travel to high altitudes should be prescreened for OSA and CSA, and health care providers should provide anticipatory guidance regarding the occurrence of CSA at high altitudes. CSA without CSB does not exhibit a crescendo–descrescendo pattern of respiration.

EPIDEMIOLOGY

Obstructive Sleep Apnea

Epidemiologists estimate the prevalence of OSA at 2% in females and 4% in males in the United States when minimal diagnostic criteria are used (AHI > 5 event per hour and the presence of daytime sleepiness) (Young et al., 1993). Based only on the criterion of AHI greater than 5 events per hour, without the criterion of sleepiness, the prevalence may be as high as 9% among women and 24% among men (Young et al., 1993). Roughly, 1 in 5 adults with a body mass index (BMI) of at least 25 kg/m^2 has at least mild OSA and 1 in 15 has at least moderate OSA (Tishler, Larkin, Schluchter, & Redline, 2003; Young et al., 1993; Young et al., 2002). Similar prevalence rates in Spain, Australia, China, Korea, and India suggest that OSA is a worldwide problem (Lee, Nagubadi, Kryger, & Mokhlesi, 2008). The prevalence of OSA is 1.7- to 3.0-fold higher for persons aged 60 years and older (Ancoli-Israel et al., 1991; Bixler et al., 2001; Bixler, Vgontzas, Ten Have, Tyson, & Kales, 1998; Young et al., 2002).

Cheyne-Stokes Breathing and Central Sleep Apnea

Epidemiological data on CSB and CSA were obtained primarily in patients with HF. Traditional estimates of the incidence of CSB/CSA in systolic HF (ejection fraction less than 40%) range from 30% to 50% (American Academy of Sleep Medicine Task Force, 1999), but recent studies that have included both men and women of diverse ethnicity and both systolic and diastolic HF suggest that CSA/CSB may be less common (9%–15% of patients) in stable HF patients. However, a larger proportion of patients with HF also have OSA (Ferrier et al., 2005; Redeker et al., 2010).

RISK FACTORS

Obstructive Sleep Apnea

The primary risk factors for OSA are individual (BMI, sex, and age) and health/disease-related characteristics (comorbid conditions, health behaviors). Identification of patients who may be at risk for OSA is an important nursing role.

Table 7.3 ■ *Diagnostic Criteria: Obstructive Sleep Apnea, Central Sleep Apnea, and Cheyne-Stokes Breathing*

SRBD Classification	Diagnostic Criteria
Obstructive sleep apnea	Subjective criteria: ■ Describes unintentional sleep episodes during wakefulness, daytime sleepiness, unrefreshing sleep, fatigue, or insomnia ■ Wakes with breath holding, gasping, or choking ■ Bed partner reports loud snoring, breathing interruptions, or both during patient's sleep Polysomnographic criteria: ■ Five or more scoreable respiratory events (i.e., apneas, hypopneas, or RERAs) per hour of sleep ■ Evidence of respiratory effort during all or a portion of each respiratory event And: ■ Disorder is not better explained by another sleep disorder, medical or neurological disorder, medication use, or substance use disorder **OR:** Polysomnographic criteria: ■ 15 or more scoreable respiratory events (i.e., apneas, hypopneas, or RERAs) per hour of sleep ■ Evidence of respiratory effort during all or a portion of each respiratory event And: ■ Disorder is not better explained by another sleep disorder, medical or neurologic disorder, medication use, or substance use disorder
Central Sleep Apnea	Subjective criteria (one or more): ■ Describes excessive daytime sleepiness ■ Frequent arousals and awakenings from sleep or insomnia complaints ■ Awakening short of breath Polysomnographic criteria: ■ Five or more central apneas per hour of sleep And: ■ Disorder is not better explained by another current sleep disorder, medical or neurological disorder, medication use, or substance use disorder
Cheyne-Stokes Breathing	Polysomnographic criteria: ■ At least 10 central apneas and hypopneas per hour of sleep in which the hypopnea has a crescendo–descrescendo pattern of tidal volume accompanied by frequent arousals from sleep and altered sleep structure And: ■ Breathing disorder occurs in association with a medical illness, such as HF, stroke, or renal failure And: ■ Disorder is not better explained by another current sleep disorder, medication use, or substance use disorder Subjective criteria (optional): ■ Excessive daytime sleepiness ■ Frequent arousals and awakenings from sleep ■ Insomnia complaints ■ Awakening short of breath

Source: International Classification of Sleep Disorders: Diagnostic and Coding Manual (2nd ed.) by American Academy of Sleep Medicine, 2005, Westchester, IL: Author.

Abbreviations: HF, heart failure; RERAs, respiratory-effort-related arousals.

Obesity

The most notable risk factor for OSA is obesity. The risk for moderate or higher OSA (apnea hypopnea index (AHI) ≥ 15/hour) is especially high at a BMI of 31 kg/m^2 or higher). With each unit increase in BMI, the odds ratio for developing OSA is 1.14 (95% CI 1.10–1.19) (Lee et al., 2008). Even patients who are mildly overweight are at risk for OSA.

Changes in weight are also associated with OSA severity. A 10% weight gain predicted, on average, a 32% increase in AHI and a 6-fold increase in the odds of developing moderate to severe OSA. Conversely, a 10% weight loss predicted a 26% decrease in AHI (Peppard, Young, Palta, Dempsey, & Skatrud, 2000). The associations between increasing AHI and weight gain were higher than the associations between weight loss and decreasing AHI (Newman et al., 2005) in the Sleep Heart Health Study that included over 6,000 patients. Weight increases were more closely associated with AHI among men than women (Newman et al., 2005). Neck circumference (≥ 17 inches in men; ≥ 16 inches in women) (White, 2006) and waist/hip ratio (ratio of 1 in men and 0.85 in women) (Seidell, 2010) are also associated with increased risk if OSA. These consistent findings underscore the importance of identifying the potential risk for OSA in patients who are overweight or obese. Prevention of weight loss may lead to reductions in the prevalence of OSA.

Aging

Older adults may have a higher prevalence of OSA than middle-aged adults. Approximately, 24% of persons 65 years or older had an AHI of 5 or higher and 62% had an AHI of 10 events per hour or more (Ancoli-Israel et al., 1991). However, prevalence, rather than severity of OSA, increases with age (Bixler et al., 1998), but seems to level out in the sixth decade of life (Ancoli-Israel et al., 1991; Young et al., 2002). To better evaluate the association between aging and OSA, further research is needed that includes larger cohorts and controls for potential confounding factors, such as obesity and gender. Aged persons, particularly those with symptoms consistent with OSA (see Table 7.3) should be further evaluated for OSA.

Gender

OSA is 1.5–3 times more common in men than women, but this gender gap decreases after menopause (Bixler et al., 2001; Young et al., 1993). Postmenopausal women are 3.5 times more likely to have OSA than premenopausal women, even when age and BMI are statistically controlled (Young, Finn, Austin, & Peterson, 2003). The narrowing of the gender gap at middle-age may be due to increased central adiposity, postmenopausal hormonal changes, that is, withdrawal of the respiratory stimulant effect of progesterone or the influence of testosterone on fat distribution (i.e., central adiposity) (Lin, Davidson, & Ancoli-Israel, 2008). The lower prevalence of OSA in premenopausal women may also be explained by differences in the manifestations of OSA. Women who have OSA tend to report more symptoms of insomnia, restless legs, nightmares, palpitations, and depression than men who are more likely to report snoring and apneas (Lin et al., 2008). It is important to consider the possibility that women who have sleep complaints have OSA, despite the absence of snoring or witnessed apnea.

Genetics

OSA increases with the number of affected family members (Redline & Tishler, 2000). Approximately, 30%–35% of the genetic variance in OSA is explained by heritability, while 40% is explained by obesity (Al Lawati, Patel, & Ayas, 2009; Redline & Tishler, 2000). Craniofacial morphology, specifically the volume of important upper airway soft tissue structures may be a heritable trait that confers excess risk (Schwab et al., 2006).

To date, investigators have not identified a single gene as causative of OSA. Because OSA is a complex disorder with many influential factors, it is likely that multiple interacting genetic and environmental factors contribute to

the risk (Palmer & Redline, 2003; Patel, 2005). Numerous studies are underway to identify the patterns of gene or protein expressions in cells or tissues related to OSA (Arnardottir, Mackiewicz, Gislason, Teff, & Pack, 2009) and to identify areas of genetic susceptibility through genome-wide association studies. Both approaches may lead to further description of OSA phenotypes and specific target cells, molecular products, and physiological outcomes that will "personalize" risk assessment and treatment response in OSA.

Race/Ethnicity

African Americans and Asians are at higher risk for OSA than Caucasians. (See Chapter 15, Health Disparities and Sleep Disorders.) The relative risk for severe OSA in older African Americans compared with Caucasians was 2.13 (Ancoli-Israel et al., 1995). After controlling for age, BMI, and sex, African Americans are 2.55 times more likely to have OSA than Caucasians (Ancoli-Israel et al., 1995). The high risk of severe OSA in younger African Americans (Redline et al., 1997) is of particular concern, given the associations of OSA with cardiovascular disease and diabetes and the high prevalence of these conditions in this population (Kaplan, 1998). Rates of OSA in Asians are similar to Caucasians, despite generally lower levels of obesity. Asians may be at increased risk due to craniofacial and posterior pharyngeal anatomical differences, even when BMI and neck are similar (Al Lawati et al., 2009).

Alcohol Consumption and Medications

Alcohol relaxes the upper airway dilator muscles and leads to increased upper airway resistance and complete or partial airway obstruction during sleep. Apnea hypopnea indices (AHI) were associated with moderate alcohol consumption among habitual snorers, but there was no association between alcohol consumption and minimum oxygen desaturation (Scanlan, Roebuck, Little, Redman, & Naughton, 2000). Each drink led to a 25% increased likelihood (OR = 1.25; 95% CI = 1.07–1.46) for OSA (Peppard, Austin, & Brown, 2007). However, in another study, women's risk of OSA did not increase with minimal to moderate alcohol consumption (Peppard et al., 2007). It is surprising that the risk associated with alcohol consumption was not associated with proximity to bedtime. Patients who have untreated OSA, including those awaiting diagnostic or treatment studies, should be advised to avoid consumption of alcoholic beverages until OSA is successfully treated.

Sedative and hypnotic medications, such as zolpidem and flurazepam, may increase the risk for respiratory depression and airway collapse in people who have OSA (Series, 2009). However, research findings are inconsistent. In general, experts recommend that use of sedatives and hypnotics should be avoided in people who have OSA or are suspected of having it, until the OSA is adequately treated. Experts recommend that individuals who have comorbid insomnia and require hypnotic or sedative medications with continuous positive airway pressure therapy (CPAP) or an alternative treatment should be studied with polysomnography while using both treatments to assure that the OSA is well controlled.

Several other classes of medications may worsen OSA. These include medications that modulate GABA function (Aurora et al., 2010; Gugger & Wagner, 2007) and the phenothiazine prochlorperazine. Although therapeutic, oral dosing of most narcotic agents is not associated with OSA, parenteral perioperative and postoperative dosing is believed to increase the severity of OSA. Persons undergoing procedures that require the use of general anesthesia or IV conscious sedation should be routinely screened for OSA, and CPAP should be available in the postoperative recovery area (Mulligan et al., 2009). (See Chapter 19, Sleep in Acute & Critical Care.)

Cigarette Smoking

Current, but not former, smokers are at increased risk for moderate–severe OSA, and the risk for OSA increases with the amount of tobacco smoked per day (Wetter, Young, Bidwell, Badr, & Palta, 1994). Although the underlying mechanisms are not well understood, the associations

between smoking and OSA may be the result of respiratory depression and reduced upper airway muscle tone associated with nicotine withdrawal during sleep, the effects of smoking on pulmonary function, or upper airway inflammation that further constricts a narrowed upper airway.

Comorbid Conditions

Medical conditions associated with increased prevalence of OSA include diabetes, polycystic ovary syndrome, refractory hypertension, coronary artery disease, HF (with systolic or diastolic dysfunction), and stroke (Lee et al., 2008), among others. Whether these associations are causal/contributory to or consequences of OSA is not known, but research is underway to further evaluate these relationships. Assessment for OSA should be a priority when providing nursing care to patients with these conditions in acute or primary care settings. (See Chapter 11, Sleep in Chronic Medical Conditions; Chapter 18, Sleep and Primary Care of Adults and Older Adults; and Chapter 20, Sleep in Acute and Critical Care Settings.)

Cheyne-Stokes Breathing and Central Sleep Apnea

Risk factors for CSB/CSA among people with HF include male gender, atrial fibrillation, older age, systolic HF, and hypocapnia (Sin et al., 1999). HF severity and systolic dysfunction were associated with CSB/CSA in some (Oldenburg et al., 2007), but not all (Redeker et al., 2010) studies. Apart from patients with HF, risk factors for CSB/CSA are not well understood (American Academy of Sleep Medicine, 2005; American Academy of Sleep Medicine Task Force, 1999).

CONSEQUENCES OF SLEEP-RELATED BREATHING DISORDERS

OSA has a significant impact on daytime function, cognitive performance, quality of life, injury, morbidity and mortality, and heightened risk for other medical conditions.

Economic Consequences

The economic costs associated with OSA are extraordinary and are associated with lost productivity, injuries, accidents, and treatment. The total economic burden of sleep disorders, including OSA, periodic limb movement disorder, and insomnia were estimated at $4,524 million (0.8% of gross domestic product) in Australia (Hillman, Murphy, Antic, & Pezzullo, 2006). The U.S. economic burden is estimated to be "greater than the economic burden of asthma and chronic obstructive lung disease and similar in magnitude to that of diabetes (132 billion dollars in 2002)" (AlGhanim, Commondore, Fleetham, Marra, & Ayas, 2008, p. 9).

Motor Vehicle Accidents

A review of 27 published studies concluded that OSA increased the risk of motor vehicle accidents, with odds ratios ranging from 1.3 to 13 (median OR = 3.1) (Ellen et al., 2006). People with severe apnea were at particular risk. Surprisingly, sleepiness was not consistently identified as a risk factor for accidents (Ellen et al., 2006). Although the reason for this is not known, it is possible that the Epworth Sleepiness Scale, a questionnaire that elicits self-reported sleepiness during everyday life and employed in most of the studies, was not sufficiently sensitive to important characteristics of neurobehavioral function that may be necessary for safe motor vehicle driving. (See Chapter 5, Conducting a Sleep Assessment.) Driving a motor vehicle is a complex task that incorporates neurobehavioral functions such as vigilance, visual acuity, cognitive function, and dexterity. Other objective or self-report measures of neurobehavioral function may be more sensitive to these factors. Nevertheless, OSA presents a critical risk of injury to self and others for individuals who operate motor vehicles or other machinery. Health professionals should query patients who are suspected of having OSA regarding their driving habits and occupational requirements and educate them about

the potential risks. It is advisable for patients suspected of having severe OSA to avoid driving or operation of heavy equipment/machinery until their OSA is treated. Patients who are untreated or insufficiently treated may need to be advised to make occupational changes if their work involves operating motor vehicles or machinery.

Neuropsychological Consequences

Decrements in neuropsychological function, including vigilance, executive function, and mood, are common consequences of OSA. A meta-analysis of norm-referenced and case-control studies revealed deficits in vigilance associated with OSA (Beebe, Groesz, Wells, Nichols, & McGee, 2003). This is not surprising because attention and concentration are particularly sensitive to sleep deprivation and loss (Durmer & Dinges, 2005). Persons with OSA also demonstrate substantially impaired executive function (working memory, mental flexibility, planning, organization, behavioral inhibition, and problem solving) (Beebe et al., 2003). These consequences have important implications for performance at work, school, and in everyday life.

The relationship between OSA and depressed mood is dose-dependent. OSA conferred a 60% increased risk for developing depression (Peppard, Szklo-Coxe, Hla, & Young, 2006). Moderate and severe OSA were associated with 2 and 2.6 times the risk of depression, respectively, in models controlling for relevant covariates (Peppard et al., 2006). Over 4-year follow up, individuals without OSA at baseline who progressed to minimal and/or mild OSA had an 80% increased risk of depression. Progression from mild to moderate or severe OSA was associated with a 3-fold risk of depression (Peppard et al., 2006). These findings, consistent with previous studies (Millman, Fogel, McNamara, & Carlisle, 1989; Sharafkhaneh, Giray, Richardson, Young, & Hirshkowitz, 2005), underscore the importance of OSA to the development of depression.

Health-Related Quality of Life

OSA is an important determinant of health-related quality of life (Moyer, Sonnad, Garetz, Helman, & Chervin, 2001), with significant decrements in both physical and mental domains (Engleman & Douglas, 2004). Impairment in social relationships, including sexual relationships (Reishtein, Maislin, & Weaver, 2010; Reishtein et al., 2006), may be explained by the impact of OSA-related sleepiness on ability to participate in family activities and sexual relationships (Reishtein et al., 2006). Sleepiness and intimacy/sexual relationships (Reishtein et al., 2010) improved with CPAP treatment. Improvement in their personal and intimate relationships may provide an incentive for adherence to CPAP treatment.

Cardiovascular Consequences

Even mild levels of OSA independently predict hypertension, stroke, and coronary artery disease (Lee et al., 2008; Morgan et al., 1998; Newman et al., 2001; Nieto et al., 2000; Redline et al., 2010; Shahar et al., 2001; Yaggi et al., 2005; Young, Peppard, & Gottlieb, 2002). For example, participants who had OSA (AHI $\geq$ 11 events per hour) had a 42% higher risk of coronary artery disease, stroke, and HF than those with an AHI less than 1.3 events per hour in the Sleep Heart Health Study, a multi-site longitudinal study of cardiovascular cohorts. Increases in cardiovascular risk were highest with AHI increments between 0 and 10 events per hour, independent of hypertension. Although hypertension has been widely recognized as a consequence of OSA, these findings suggest that, in addition to hypertension, other pathophysiological factors likely contribute to the increased cardiovascular risk associated with OSA.

Risk of stroke is more powerfully associated with OSA, including simple snoring, than with underlying cardiovascular disease (Shahar et al., 2001). OSA patients had significantly heightened risk for stroke and death (Hazard Ratio 1.97; 95% CI 1.12–3.48; $p = 0.01$) after controlling for relevant covariates. The duration and severity of disease may be important factors in risk of stroke and death (Yaggi et al., 2005), since this relationship was higher for patients with more severe baseline AHI. Further research is needed to better define the causal relationships between OSA and its cardiovascular consequences.

Metabolic Consequences

There is convincing evidence that OSA is associated with the development of insulin resistance/glucose intolerance, metabolic syndrome, and the development of type 2 diabetes (Chasens, Weaver, & Umlauf, 2003; Levy, Bonsignore, & Eckel, 2009; Morritt Taub & Redeker, 2008; Van Cauter, Speigel, Tasali, & Leproult, 2008). Although it is difficult to establish causality because of the associations between OSA, metabolic dysfunction, and obesity, the relationships between OSA and metabolic risk persist even when BMI is statistically controlled in observational studies (Ip et al., 2002; Punjabi et al., 2004; Vgontzas, Bixler, & Chrousos, 2003). Both sleep duration and sleep fragmentation, with associated activation of the sympathetic nervous systems and hypothalamic pituitary axes, appear to be associated with metabolic risk. The importance of these associations is underscored by the growing worldwide epidemic of obesity and Type 2 diabetes, which will likely contribute to an increasing prevalence of OSA.

Cheyne-Stokes Breathing and Central Sleep Apnea

CSB/CSA predicts mortality (Javaheri, Smith, & Chung, 2009; Lanfranchi et al., 1999) in HF patients. AHI and left atrial area (≥25 cm by echocardiography) were the only independent predictors of cardiac death. Median survival was 45 months in patients with CSA compared with 90 months in patients without CSA (Javaheri et al., 2009). Although these findings suggest that CSB/CSA is associated with excess mortality, whether CSB/CSA is a marker of severity of HF or an independent contributor to mortality is not known.

PATHOPHYSIOLOGY OF SLEEP-RELATED BREATHING DISORDERS

Obstructive Sleep Apnea

Partial or complete repetitive nocturnal upper airway pharyngeal closures that occur in OSA are associated with intermittent hypoxia and sleep fragmentation. Both anatomical and neurophysiological mechanisms contribute to the pathogenesis of OSA (see Figure 7.1).

Several pathophysiological factors contribute to airway collapse during sleep: (1) upper airway anatomy, (2) responsivity of upper airway dilator muscles to respiratory challenge during sleep, (3) propensity to awaken in response to increased respiratory drive (arousal threshold), (4) respiratory control stability, and (5) state-related changes in lung volume (Eckert & Malhotra, 2008).

Upper Airway Anatomy

The upper airway is flexible and composed of muscle and soft tissue that facilitate speech and swallowing. The cross-sectional area of the hard palate and larynx is reduced in patients with OSA (Eckert & Malhotra, 2008). Increased soft tissue in the structures surrounding the airway and thickened lateral pharyngeal muscular walls contributes to collapsibility of the flexible upper airway (Schwab et al., 1995).

Responsivity of Upper Airway Dilator Muscles

Upper airway dilator muscles are more active in OSA patients, even during the day, than in persons without OSA. This may be a protective mechanism for an already-narrowed upper pharyngeal space and the normal response of muscular relaxation during sleep (Eckert & Malhotra, 2008). The upper airway dilator muscles are responsive to negative pharyngeal pressure and possibly hypercapnia. During sleep, the responsivity of these reflexes decreases and may contribute to upper airway collapse (Eckert & Malhotra, 2008).

Arousals From Sleep

Electrophysiological cortical arousals usually accompany respiratory events and vary in duration relative to the degree of hypoxemia. The arousal mechanism promotes upper airway reopening and stimulation of the upper airway dilator muscles. Arousals are associated with ventilatory responses, such as hyperventilation

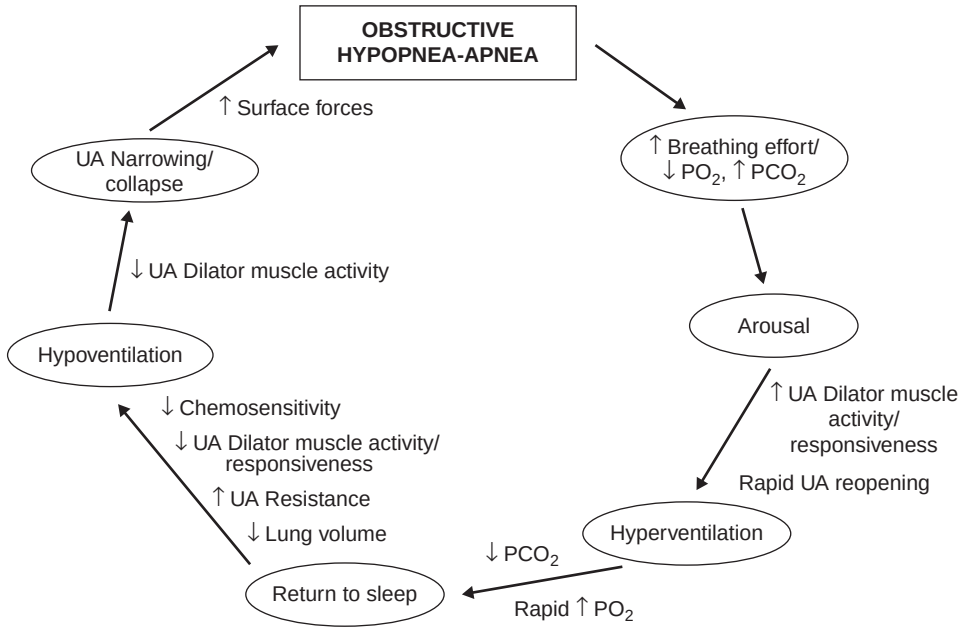

Figure 7.1 ■ Anatomical and Neurophysiologic Mechanisms of Obstructive Sleep Apnea. *Source*: "Pathophysiology of Adult Obstructive Sleep Apnea" by D. Eckert and A. Malhotra, 2009, *Proceedings of the American Thoracic Society, 5*, p. 148. Reprinted with permission from American Thoracic Society.

(Eckert & Malhotra, 2008) and may also contribute to the excessive sympathetic activation associated with some of the negative cardiovascular consequences of OSA.

Respiratory Control Stability

Chemical feedback contributes to the stability or instability (i.e., periodicity) of breathing (Eckert & Malhotra, 2008). In the presence of hypoxia or hypercapnia, peripheral chemoreceptors detect an alteration in CO_2 levels and stimulate upper airway muscle activation and respiratory effort. Decreased chemosensitivity (sensitivity to CO_2 levels) contributes to hypoventilation and reduced activation of the upper airway muscles. This may result in periodic breathing and apneas and/or hypopneas. Electrophysiological cortical arousals are associated with airway muscle stimulation and opening.

Lung Volume

Lung volumes are reduced in recumbent positions during sleep, and there is a caudal shift of intrathoracic structures that may increase the upper airway resistance and can cause collapse of the airway (Eckert & Malhotra, 2008). Although this occurs in healthy people and those with OSA, the reasons for the development of this problem in patients with OSA are not known.

Cheyne-Stokes Breathing and Central Sleep Apnea

Low cardiac output and prolonged arterial circulation time in people with CSA and CSB create delays in feedback from the pulmonary capillary bed to the central nervous system (Mookadam, Calvin, & Somers, 2008). There is also increased sensitivity to carbon dioxide and a heightened ventilatory response that lead to periodic breathing. For example, with central apneic events, the pCO_2 increases and leads to hyperventilation. This response quickly lowers the pCO_2 below the apnea threshold and central apnea recurs due to loss of the stimulation to breathe.

ASSESSMENT OF PATIENTS WITH SLEEP-RELATED BREATHING DISORDERS

Clinical Presentation

Obstructive Sleep Apnea

Patients with untreated OSA typically present with reports of snoring, witnessed apneas, and restless sleep, although this may vary somewhat for women, as described above. They may also describe decreased dreaming, frequent awakenings for unknown reasons, choking/gasping during sleep, unrefreshing sleep, dry mouth, headache, daytime sleepiness or excessive tiredness, and memory impairment (Epstein et al., 2009). OSA patients may report nocturia unrelated to other medical conditions and/or medications (Epstein et al., 2009). It is important to query patients about OSA symptoms because many patients will not necessarily recognize sleep-related symptoms or offer such information during a history and examination (see Table 7.4). Although not specific to OSA, patients are likely to present with obesity, history of cardiovascular disorders, or type 2 diabetes.

Cheyne-Stokes Breathing/Central Sleep Apnea

CSB/CSA is common in patients who have HF and may explain some of the persistent fatigue and reduced exercise capacity (Randerath, 2009). Persons with CSB/CSA do not snore, but may describe unrelieved fatigue, insomnia, restless sleep, daytime sleepiness, and unexplained nocturnal awakenings. A bed partner may describe prolonged pauses in breathing.

Evaluating Daytime Consequences of Sleep-Related Breathing Disorders

Because excessive daytime sleepiness is common and disabling in OSA, experts recommend that patients be evaluated for sleepiness (Epstein et al., 2009). Assessment of sleepiness should address the frequency of excessive daytime sleepiness, periods of difficulty fighting off sleep, and nonrestorative sleep (American Academy of Sleep Medicine Task Force, 1999; Epstein et al., 2009). Two instruments are most commonly used to measure subjective sleepiness: The Stanford Sleepiness Scale (SSS) (MacLean, Fekken, Saskin, & Knowles, 1992) and the Epworth Sleepiness Scale (ESS) (Johns, 1993). (See Chapter 5, Conducting a Sleep Assessment.)

Nurses should ask patients about their cognitive function, including memory and executive function (e.g., ability to plan and make decisions). They should also ask about history of accidents or lapses of attention in the workplace or while driving or operating heavy machinery. Because of varying presentations of sleep-disordered breathing, assessment of clinical consequences should be individualized and comprehensive (see Table 7.4).

Physical Assessment

Persons with OSA are often, but not always, obese or overweight, although thin people may have OSA. Therefore, obtaining anthropometric measures such as height, weight, BMI, waist–hip ratio, and neck circumference are an important component of the physical exam. Neck circumference should be measured because large neck size [(>17 inches (i.e., shirt collar size of 17) in men and (>16 inches in women)] is a risk factor. Measures of glycemic control and blood pressure are helpful in assessing the contributions of OSA to diabetes and cardiovascular risk.

Visual examination of the oronasopharynx often reveals one or more of the following: (1) large tongue, with or without scalloping pattern on lateral aspects; (2) posterior pharyngeal crowding, including reduction in the anterior–posterior and/or lateral diameter reduction; (3) erythematous and/or edematous posterior pharyngeal soft tissue structures; (4) retrognathia, a condition in which either or both jaws recede with respect to the frontal plane of the forehead; and (5) erythematous and/or edematous nasal mucosa, deviated nasal

Table 7.4 ■ *Assessment of Patients With Sleep-disordered Breathing*

Who should be evaluated for obstructive sleep apnea?

- Overweight or obese (BMI ≥ 25)
- Medical conditions: HF, atrial fibrillation, refractory hypertension, nocturnal dysrhythmias, stroke, pulmonary hypertension, type II diabetes
- Commercial drivers or individuals who operate machinery
- Preoperative bariatric surgery
- Severe snoring and/or witnessed apneas

Assess symptoms, signs, and consequences (elicit also from bed partner):

- Do you snore?
- Do you experience "stops in breathing" during sleep? Has anyone told you that you stop breathing in your sleep?
- Do you awaken from sleep for unknown reasons? Do you awaken from sleep feeling short of breath or with the feeling that you could not catch your breath?
- Do you feel refreshed when you awaken from sleep?
- Do you have a headache when you awaken in the morning?
- Do you have sleepiness during daytime that necessitates taking naps? Are your naps refreshing?
- Do you fall asleep when you do not intend to?
- Have you ever had an accident related to driving a motor vehicle or operating machinery because you fell asleep? Have you ever had a single-vehicle accident?

Assess associated factors and consequences:

- Sleep schedule (bedtime, awake time, consistency, naps, sleep latency, reasons for wakenings)
- Use of caffeine, alcohol, tobacco, illicit drugs with sedating properties
- Exercise, meal times
- Other sleep disorders (e.g., insomnia, periodic limb movements)
- Comorbid conditions (e.g., hypertension, stroke, cardiovascular disease, hyperlipidemia, diabetes, neuromuscular disorders, neurological disorders of vascular origin)
- Family medical history of OSA, snoring
- Medications including CNS depressants, GABA-ergic agents
- Blood sugar, HBA1C, blood pressure
- Occupation (e.g., heavy equipment operator, commercial driver)
- Driving habits
  - Number hours/day driven
  - History of fall asleep accidents/near misses
  - History of single vehicle accidents
- Mental health, including depressive disorders
- Social relationships (e.g., quality of interactions, feedback from friends/co-workers about interactions/productivity, and characteristics of intimate relationships)

Abbreviations: CNS, central nervous system; HBA1C, hemoglobin A1C; HF, heart failure; OSA, obstructive sleep apnea.

septum, and/or nasal polyps. Structural nasal abnormalities or sinus congestion associated with allergies or sinusitis may be present.

Posterior pharyngeal crowding can be assessed by visual examination and Mallampati scoring. The examiner asks the patient to open his/her mouth, either pushing the tongue forward (traditional Mallampati assessment) or retaining the tongue in a relaxed state in the oral cavity (modified Mallampati assessment). The appearance of the posterior pharynx is graded on a scale of I–IV (see Figure 7.2), with class III and IV highly suggestive of OSA.

There are no specific physical findings for CSB/CSA, but HF patients should be assessed for signs of fluid overload, including rales, peripheral edema, jugular venous distention, and atrial fibrillation.

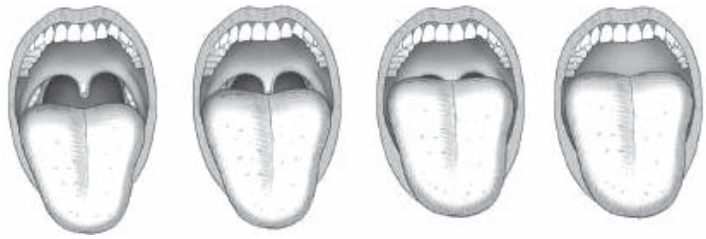

Figure 7.2 ■ *Mallampati Airway Classification.*[a] Assessment of Mallampati scores is conducted by asking the patient to open his or her mouth as wide as possible while pushing the tongue forward as far as possible. A modified Mallampati procedure includes only asking the patient to open his or her mouth as wide as possible, maintaining the tongue in a resting position. Ask the patient to avoid emitting any sounds during this assessment. Scoring is consistent for both Mallampati and modified Mallampati assessment. Class I: soft palate and entire uvula visible; Class II: soft palate and portion of uvula visible; Class III: soft palate visible (may include base of uvula); Class IV: soft palate not visible.

Source: "Physical Examination: Mallampati Score as an Independent Predictor of Obstructive Sleep Apnea," by T. J. Nuckton, D. V. Glidden, W. S. Browner, and D. M. Claman, 2006, *Sleep, 29*, pp. 903–908. Reprinted with permission of the American Academy of Sleep Medicine.

Diagnosis of Sleep-Related Breathing Disorders

Polysomnography

PSG is the gold standard for diagnosing sleep-disordered breathing. (See Chapter 5, Conducting a Sleep Assessment.) In addition to measures of EEG, EOG, and chin EMG necessary for sleep scoring, respiratory measures of respiratory effort (chest and abdomen), nasal airflow (pressure recorded with a transducer or temperature change recorded with a thermistor), and pulse oximetry are obtained. PSG characteristics associated with OSA include apneas, hypopneas, and respiratory event-related arousals (RERAs), intermittent oxygen desaturation, persistent respiratory effort, and electrophysiological arousals (see Figure 7.3). Clinical PSG reports usually include the apnea hypopnea index, respiratory disturbance index, the presence of central or obstructive apnea, and the degree of oxygen desaturation, in addition to associated changes in sleep architecture. These measures are used to establish the presence and severity of sleep-related breathing disorders.

Patients with OSA may also have periodic limb movements associated with respiratory events, but the diagnosis of periodic limb movement disorder (see Chapter 8, Movement Disorders) cannot be established until it is clear that the limb movements are not secondary to OSA. Cardiac dysrhythmias may also occur, especially in patients with oxygen desaturation. Criteria for the diagnosis of OSA are listed in Table 7.3.

PSG characteristics of CSB/CSA include a waxing and waning pattern of central apneas with periods of hyperpnea (i.e., abnormal increase in depth and rate of respirations) (Figure 7.4). (Diagnostic criteria for CSA/CSB are listed in Table 7.3.) Figure 7.5 illustrates the cyclical breathing pattern, absence of respiratory effort accompanying central apnea events, and EEG arousals that characterize CSA. Patients with OSA and CSA/CSB demonstrate sleep fragmentation and abnormal sleep architecture, as demonstrated by low levels of sleep efficiency, slow wave and stage R (REM) sleep, and increased stage N1 sleep.

Portable or Abbreviated Sleep Studies

Access to full attended PSG in sleep laboratory settings is often limited due to high demand, limited numbers of sleep centers, particularly in rural areas, and significant waiting periods (Agency for Healthcare Research and Quality, 2007). Unattended portable or abbreviated sleep studies may increase access and reduce the high costs of laboratory-based PSG (Agency for Healthcare Research and Quality, 2007).

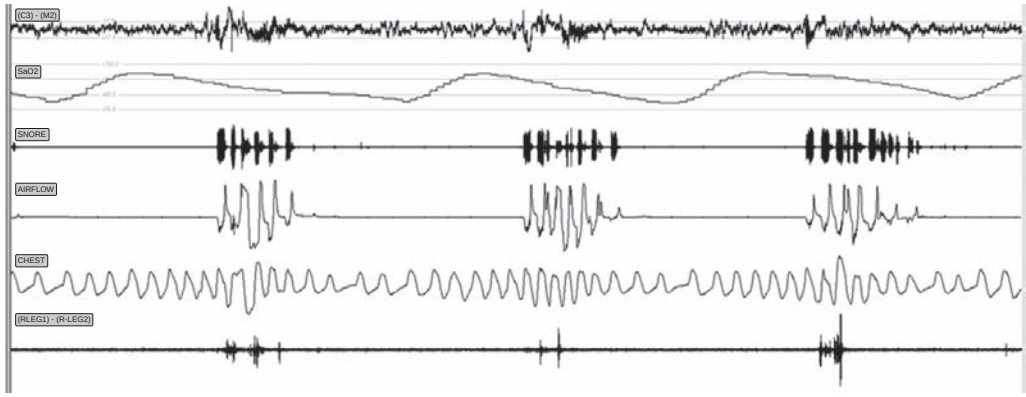

Figure 7.3 ■ *Polysomnographic recording of obstructive sleep apnea (120-second epoch).* The polysomnogram recording demonstrates the classic recording of obstructive sleep apnea. The recording includes (top to bottom): EEG (C3M2, a specific EEG-recording channel), oximetry (SaO2), microphone (snore), nasal airflow, chest plethysmography, and leg EMG. Note the lack of airflow on the nasal airflow channel and the persistent effort to breathe as recorded by a relatively robust waveform for chest plethysmography. Also note the intermittent pattern of oxyhemoglobin desaturation that follows the airflow decrement and intermittent leg movements as recorded on the leg EMG channel. Snoring is consistent on the microphone recording when there is a patent upper airway. There are also cortical arousals noted on the EEG recording that coincide with upper airway reopening and resumption of normal respiration during sleep.

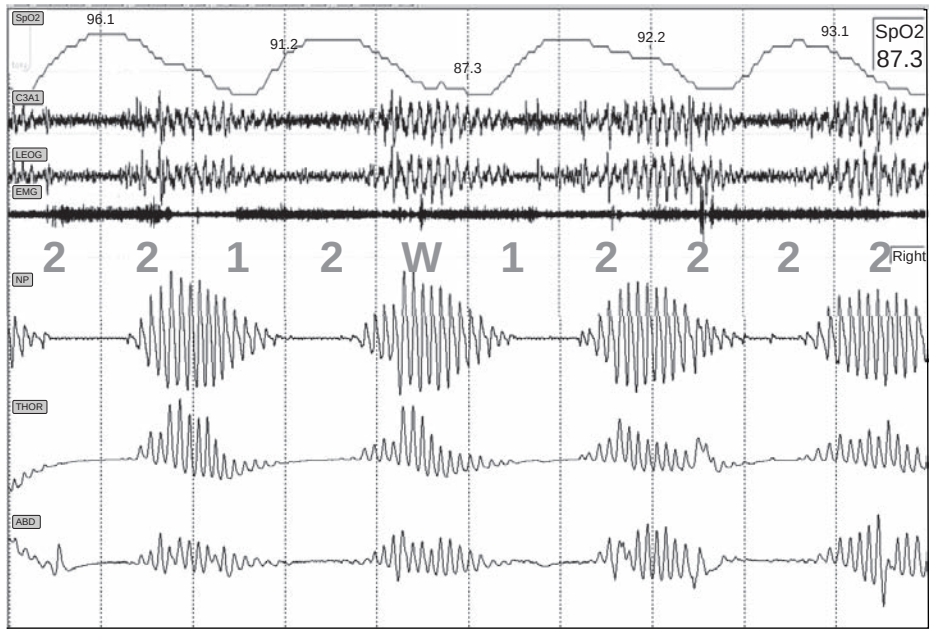

Figure 7.4 ■ *Polysomnographic recording of central sleep apnea with cheyne-stokes pattern (5-minute epoch).* The polysomnogram recording depicts central sleep apnea with a Cheyne-Stokes respiratory pattern. Polysomnogram recording includes (top to bottom): oximetry (SPO2), EEG (C3A1), electroculogram (EOG), chin electromyelogram (EMG), airflow by nasal pressure, chest plethysmography, and abdomen plethysmography. Note the complete absence of respiratory effort (thoracic and abdominal channels) in the absence of nasal airflow as detected by nasal pressure recording. With resumption of respirations, there is a gradual increase in respiratory effort and airflow and a gradual decrease with the onset of a central apneic event, representing the Cheyne-Stokes pattern of respiration. Oxygen desaturation occurs with each central event, with the nadir following the event in sequence.

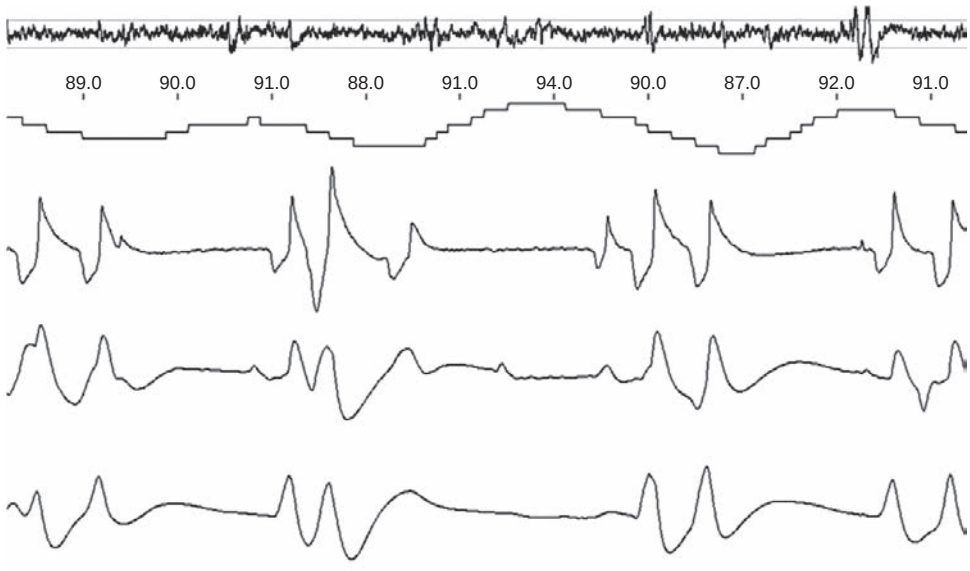

Figure 7.5 ■ *Polysomnographic recording of central sleep apnea.* The recorded polysomnogram depicts central sleep apnea. The polysomnogram recording includes (top to bottom): EEG, oximetry, nasal airflow, chest plethysmography, abdomen plethysmography displayed as a 50-second epoch. The recording demonstrates the complete absence of respiratory effort and upper airway airflow with the flattening of the channel recording for the nasal airflow and plethysmography. The central apneic event is followed by a desaturation in oxygen, which reaches its nadir with resumption of respiratory effort.

Abbreviated studies are valid measures of OSA, but not CSA/CSB (Collop, 2008; Collop et al., 2007), and some health insurers now reimburse for their use (Department of Health and Human Services, Center for Medicare and Medicaid Services, & 2008).

Different types of portable or abbreviated sleep studies are available, each of which measures a different set of physiologic parameters (see Table 7.5). The American Academy of Sleep Medicine (AASM) (Collop et al., 2007) recommends the use of at least Type 3 devices (see Table 7.6) for the diagnosis of OSA in people without significant comorbid conditions (Collop et al., 2007). Type 4 devices are not recommended because they do not include important cardiopulmonary variables. The AASM also recommends that portable devices be used only in people who have had a comprehensive history and physical exam and that the diagnosis be made by certified sleep specialists.

Treatment and Follow-Up Care

Obstructive Sleep Apnea

Effective treatments for OSA include continuous positive airway pressure therapy (CPAP), surgical treatments, oral appliances, and weight loss. These are reviewed in detail below.

Continuous Positive Airway Pressure Therapy

CPAP (Figure 7.6), the most commonly used treatment, involves the delivery of positive pressure into the airway through a nasal (Figure 7.7) or full face (Figure 7.8) mask or nasal pillows (Figure 7.9) to pneumatically splint the airway to maintain its patency. This leads to reduction in apneas and hypopneas during sleep and return of oxyhemoglobin saturation to nonapneic sleep levels and reduction in respiratory-related EEG arousals (Gay, Weaver, Loube, & Iber, 2006).

Table 7.5 ■ *Categorization of Sleep Study Devices*

Type 1 Sleep Study: Standard in-laboratory, technician-attended, overnight polysomnogram.
Type 2 Sleep Study: Type I polysomnography, not attended by a technician.
Type 3 Sleep Study: Record a minimum of four to seven physiologic variables that minimally includes two respiratory variables (airflow and effort), a cardiac signal (pulse or electrocardiogram), and oxyhemoglobin saturation by pulse oximetry.
Type 4 Sleep Study: Records only one or two variables, including oxyhemoglobin saturation.

Table 7.6 ■ *Guidelines for Use of Portable Sleep Studies in the Diagnosis of OSA*

■ Use only when incorporated with a comprehensive sleep evaluation
■ Patients best suited for portable monitor sleep studies are those with a high pretest probability of moderate to severe OSA
■ Not recommended for patients who have comorbid conditions or when coexistent sleep diagnoses are suspected
■ Airflow, respiratory effort, and blood oxygenation must be included in the portable monitor recording
■ Application of the recording device should be performed by an experienced sleep technologist or self-application instruction should be provided to patients by an experienced sleep technologist
■ Raw data from portable monitors must be available for diagnostic determination
■ Patients undergoing portable monitoring should have a follow-up clinical evaluation to discuss test results

Source: "Clinical Guidelines for the Use of Unattended Portable Monitors in the Diagnosis of Obstructive Sleep Apnea in Adult Patients," by N. A. Collop, W. M. Anderson, B. Boehlecke, D. Claman, R. Goldberg, D. J. Gottlieb, et al.; Portable Monitoring Task Force of the American Academy of Sleep Medicine, 2007, *Journal of Clinical Sleep Medicine, 3*, pp. 737–747. Adapted with permission.

Figure 7.6 ■ *Continuous positive airway pressure device with tubing and mask.* Continuous positive airway pressure device with humidifier chamber, permitting delivery of humidified air (warm or cool), with tubing and headgear/mask attached.

Although CPAP is highly effective in reducing respiratory events and associated oxyhemoglobin desaturation, particularly in severe OSA, data on its impact on the daytime consequences of OSA are less consistent (Gay et al., 2006). A meta-analysis of 11 randomized controlled trials (Patel, White, Malhotra, Stanchina, & Ayas, 2003) revealed that in comparison with placebo, CPAP improved subjective sleepiness, particularly in severe OSA (Patel et al., 2003). In a controlled trial of 45 patients, optimal levels of CPAP compared with placebo, improved vigilance and productivity (Montserrat, Ferrer, & Hernandez, 2001), measured with the Functional Outcomes of Sleep Questionnaire [FOSQ]) (Weaver et al., 1997). There were nonstatistically significant trends for improvements in the FOSQ and OSA symptoms (i.e., snoring, breathing pauses, nocturia, dry mouth, morning headaches, and unrefreshing sleep) improved. Unfortunately, neurobehavioral function and quality of life do not consistently improve with CPAP treatment (Gay et al., 2006).

Although CPAP acutely lowers blood pressure, evidence of its ability to reduce 24-hour blood pressure, and therefore improve hypertension and prevent coronary artery disease, HF, and stroke is less consistent (Gay et al., 2006; McDaid et al., 2009).

Figure 7.7 ■ *Continuous positive airway pressure therapy (CPAP) delivery by nasal mask interface.* CPAP nasal mask delivery interface shown on a mannequin. The nasal mask interface is a triangular-shaped mask that fits only over the nose. The mask is secured in place by a headgear system typically made of soft cloth material and Velcro for attachments. The mask connects to ventilator circuit tubing that attaches directly to the CPAP unit. The connection point from mask to tubing is generally flexible and often swivels to accommodate sleep movement.

Figure 7.8 ■ *Full-face mask interface for CPAP delivery.* A full-face mask and headgear for CPAP delivery are shown on the mannequin. This style of mask can be recommended for those with a persistent mouth leak when using a nasal interface.

Research is now underway to examine the effects of CPAP on reducing the negative cardiovascular consequences of OSA.

Due to the international epidemic of obesity and Type 2 diabetes, there is great interest in improving the metabolic consequences of OSA with CPAP. Although some studies have identified improved insulin sensitivity and glycemic control, glucose levels, HbA1c, and plasma cholesterol and HOMA indices (Levy et al., 2009), research findings have been inconsistent and obesity, sleep fragmentation and sleep duration are confounding factors.

A large body of studies documenting associations of OSA with these cardiovascular and metabolic disorders and the documented acute effects of CPAP suggest its potential to reduce morbidity. Therefore, experts recommend that patients with OSA who are at risk for these conditions use this treatment. However, there is a pressing need for randomized clinical trials to evaluate the long-term effects of CPAP.

Adherence to CPAP

The dose of CPAP required to normalize subjective and objective sleepiness and improve function relative to OSA is estimated at 4, 6, and 7 hours per sleep period, respectively (Weaver et al., 2007). Intermittent and short-duration CPAP users are not likely to experience improvements, and only about 50% of patients use it consistently. Patients generally make decisions to use CPAP on day 2 and 4 of treatment (Aloia, Arnedt, Stanchina, & Millman, 2007; Weaver et al., 1997), and skipping nights early in the course of treatment is associ-

Figure 7.9 ■ *Continuous positive airway pressure therapy (CPAP) delivery by nasal pillow interface.* The nasal interface with CPAP sits below the nose on the maxilla and includes soft, nasal pillows that interface with bilateral nares. The seal at the nares should not permit air leakage (i.e., loss of positive pressure). Similarly, the pillows should not place undue pressure on the nares so as to cause dermatologic damage (i.e., pressure ulceration).

ated with lower adherence (Aloia et al., 2007; Krieger, 1992; McArdle et al., 1999; Weaver et al., 1997). Therefore, the ideal time to intervene to promote adherence seems to be early in treatment and those who skip nights may need specific encouragement and coaching to promote adherence.

Like other areas of adherence to treatment for chronic conditions, adherence to CPAP is a multifactorial problem. Strategies that combine cognitive, supportive, and educational approaches may lead to higher CPAP adherence. For example, Hoy and colleagues (Hoy, Vennelle, Kingshott, Engleman, & Douglas, 1999) significantly improved CPAP use at 1 month with an intensive intervention that included in-laboratory CPAP titration for three nights, intensive CPAP training, inclusion of the patients' partners, and home visits. Although this intervention may be too resource-intensive for widespread adoption, group (Richards, Bartlett, Wong, Malouff, & Grunstein, 2007) and individual (Aloia et al., 2007) cognitive behavioral interventions that are somewhat less intensive improved CPAP use. There is a need for studies in larger, heterogeneous samples to better elucidate the intervention components that are effective for improving short- and long-term CPAP adherence (Weaver & Sawyer, 2009, 2010).

Interventions that clarify perceptions, promote realistic outcome expectations, and increase confidence in ability to use CPAP (i.e., self-efficacy) are most likely to be effective in newly diagnosed patients (see Table 7.7). Enlisting social support from family members or peers may also be helpful, especially during the patients' first exposure and acclimatization to CPAP (usually during CPAP titration). Significant others or CPAP experts available in the sleep clinic or the durable medical equipment company that supplies the device (Sawyer, Deatrick, Kuna, & Weaver, 2010; Weaver & Sawyer, 2010) may assist in trouble-shooting difficulties with the equipment and adapting to daily use. When patients return to their primary care provider after receiving specialized sleep treatment, it is important to elicit any problems and adherence with CPAP treatment. (See Chapter 17, Sleep Promotion Adult Primary Care Settings.)

Trouble-shooting problems with CPAP equipment. Patients may experience problems with use of CPAP including pressure points, discomfort, and skin irritation from the mask, nasal problems, and insomnia. The mask or nasal interface should not fit tightly, but a gentle seal at the mask-skin interface should be created once CPAP pressure is applied. If this cannot be achieved without excessive tightening of the headgear, the mask is probably not fitted correctly. The patient should be refitted for a different size or style mask by the sleep specialist's staff or by the durable medical equipment company.

Nasal and pharyngeal complaints that commonly occur with CPAP are dryness, sneezing, and/or congestion. Heated or cool humidifiers

Table 7.7 ■ *Patient Education and Follow-Up Care Related to Sleep-Disordered Breathing*

- Include bed partner, spouse, or a support person in patient education if possible
- Provide description of sleep-related breathing disorders
- Describe potential negative health consequences (e.g., hypertension, stroke, diabetes, diabetes, performance)
- Explain sleep testing and diagnostic process (e.g., PSG in sleep laboratory)
- Emphasize the need for follow-up after treatments are initiated
- Advise on managing treatments (CPAP, oral appliances, weight loss)
- CPAP-related issues
  - Cleaning & maintaining equipment
  - Correct application of headgear
  - Correct settings—pressure, humidification
  - Use of ramp function to promote sleep initiation
  - Procedures to follow when travelling, including use of adapters in foreign countries
  - Trouble-shooting problems: mouth leak, respiratory infections, dermatological problems, nasal discomfort/congestion
- Advise to avoid use of alcohol and GABA-ergic medication
- Advise to report recurrence of symptoms (sleepiness, fatigue, performance problems, snoring) to health care provider; symptoms will recur if treatment discontinued even temporarily
- Provide guidance on weight loss, including referrals to dietician
- Advise to bring & use CPAP (labeled with name) or oral appliance if hospitalized or when travelling
- Monitor and promote adherence, beginning in early treatment phase
  - Offer opportunities to discuss experiences of others diagnosed and treated for sleep-related breathing disorders
  - Provide concrete cognitive-behavioral strategies to promote adherence
  - Offer support groups if available
  - Use telephone contact, online chat rooms, and other mechanisms for support
  - Provide information in written handouts or on a Web site
  - Provide contact information for:
    - the sleep providers who diagnosed and initiated treatment recommendations
    - the durable medical equipment company who supplies and maintains the CPAP equipment

Abbreviations: CPAP, continues positive airway procedure; PSG, polysomnography.

are available and usually correct nasal problems. Patients with a history of chronic nasal congestion due to allergies, chronic irritant exposure, or recurrent sinusitis/rhinitis, may need to be evaluated for concomitant treatment with a nasal corticosteroid spray, antihistamines, and/or regular application of a nasal saline spray. Patients should be advised that they may not be able to tolerate CPAP during an upper respiratory tract infection and that OSA and its symptoms will recur while CPAP is temporarily discontinued. During periods without treatment, patients must be advised to avoid driving and operation of heavy machinery.

Dermatologic reactions to CPAP masks can occur, especially if the mask is not cleaned. Patients should be instructed to wash the tubing and mask with warm water and mild soap and let it completely dry on a weekly basis. The mask surface should be wiped clean each day with warm water. Skin pressure points can develop in some patients due to the mask fit, but this is not always avoidable. If the patient is able to apply the mask without excessive tightening of the headgear but continues to have persistent pressure points, it may be useful to provide an alternated style (e.g., face or nasal) mask for alternate use every few days.

Air leaks through the mouth or at the upper aspect of a nasal mask reduce the efficacy of CPAP treatment. Leaks near the eyes may cause eye irritation and significant optic damage if large and persistent. Leaks should be evaluated by observing the application of the mask/headgear while connected to CPAP pressure. If the application of mask/headgear is acceptable, but a seal cannot be established without leaks,

an alternative style mask should be considered. Chinstraps that gently keep the mouth closed can be used to correct mouth leaks for patients who use nasal masks. Use of a full-face mask (image 4) may prevent mouth leaks if the chinstrap does not prevent mouth opening.

Some patients may experience insomnia while using CPAP. To address this, CPAP can be set at a reduced pressure setting at the time the machine is powered on and then ramp up the pressure, permitting the patient to initiate sleep without full pressure delivery. The ramp function can be activated at the sleep onset and also at anytime during the night with a conscious awakening. Use of a relaxation method may improve sleep initiation and can also be used by patients who awaken during the night. Some patients may benefit from the use of hypnotics.

Many patients find it helpful to disconnect the CPAP at the hose–mask connection rather than completely removing the mask/headgear if they get out of bed to use the bathroom. This allows them to return to bed without having to reapply the mask/headgear and reestablish a mask interface seal.

Surgical Treatment

Surgical treatment for OSA focuses on increasing the upper airway area. Uvulopalatopharyngoplasty (UPPP) is most often used, but laser-assisted uvulopalatoplasty (LAUP), radiofrequency tissue ablation, and other surgical procedures are also employed. Unfortunately, the effectiveness of these procedures has not been established (Franklin et al., 2009). Adverse effects included death (0.2%), difficulty swallowing and nasal regurgitation (31%), voice changes (13%), and taste disturbances (5%) (Franklin et al., 2009). LAUP, surgical uvulopalatoplasty, and radiofrequency tissue ablation were also associated with significant adverse effects (Franklin et al., 2009).

Oral Appliances

Oral appliances are used to modify the position of the mandible, tongue, and other oropharyngeal structures to increase the cross-sectional dimension of the posterior pharynx (Hoekema, Stegenga, & deBont, 2004). They are usually prescribed and fitted by dentists specializing sleep medicine and include tongue-retaining or mandibular repositioning appliances (Hoekema et al., 2004) that are efficacious for mild–moderate OSA (Hoekema et al., 2004; Kushida et al., 2006) compared with UPP or placebo, but less efficacious than CPAP (Hoekema et al., 2004). Patients who decline CPAP or have mild–moderate OSA may be good candidates for oral appliances, but they are not useful in edentulous patients. Patients should undergo PSG testing to assure effective reduction in OSA while wearing the appliance.

Weight Loss

Since obesity is the major risk factor for OSA, weight loss may be useful. This may be accomplished through lifestyle changes, such as diet and exercise or through bariatric surgery for the morbidly obese. The added benefits of weight loss are its effects in reducing the negative consequences of other chronic diseases (e.g., diabetes and cardiovascular disease) and reducing weight-related disability.

An intensive lifestyle intervention (Foster et al., 2009) led to weight loss of 10.8 kg in diabetic patients in the experimental condition, compared with 0.6 kg ($p < 0.001$) in the placebo condition. This was associated with decreased in the AHI by 9.7 events per hour ($p < 0.001$) and remission of OSA (Foster et al., 2009) in the treatment group. A very low calorie diet and supervised lifestyle counseling over 12 weeks, compared with lifestyle counseling alone, led to significant weight loss and decreased severity of OSA (Tuomilehto et al., 2009). Although lifestyle change is difficult, it has multiple beneficial effects and may prevent OSA and its negative consequences.

Patients with OSA who are morbidly obese and have comorbid conditions may benefit from bariatric surgery. A recent meta-analysis revealed strong evidence for the effectiveness of surgical weight loss in improving OSA. On average, with significant postoperative weight reduction (BMI reduction 17.9 kg/m^2; 95% CI 16.5–19.3),

AHI decreased from 38.3 events per hour to 15.8 events per hour (Greenburg, Lettieri, & Eliasson, 2009). Because weight loss does not guarantee reductions in OSA severity, it is necessary to perform follow-up PSGs to reevaluate the need for continuing use of CPAP.

Cheyne-Stokes Breathing and Central Sleep Apnea

Treatments for CSB and CSA

Treatments for CSB/CSA include CPAP, bi-level positive airway pressure, noninvasive nocturnal ventilation, nocturnal oxygen, and adaptive servo-ventilation (ASV). Among HF patients, optimal medical management with beta blockers, ACE inhibitors and diuretics, and cardiac resynchronization therapy may also improve CSB/CSA (Redeker, 2008). Early studies suggested that CPAP improved central apneas, oxygen desaturation, and ejection fraction among HF patients. However, the CANPAP trial (Bradley et al., 2005), conducted in patients with systolic HF and an AHI greater than 15, found a trend toward better survival in the control group despite improvements in CSA/CSB, ejection fraction, and function in the treatment group. Comorbid OSA should be treated and several recent studies found that CPAP is safe and effective as a treatment for OSA in these patients.

Adaptive servo-ventilation (ASV) may be more sensitive than CPAP to individual ventilatory patterns and patients may prefer it to other devices (i.e., CPAP, bi-level PAP; Randerath, 2009). However, there is little data from which to draw conclusions regarding improvement in cardiovascular outcomes in HF patients with CSB/CSA.

NURSES' ROLES IN PREVENTING AND TREATING SLEEP-RELATED BREATHING DISORDERS

Patient teaching (see Table 7.7) is a major focus of nursing care with patients who have OSA. Patients at risk for sleep-disordered breathing are usually treated in a specialized sleep center, but are first screened in primary care settings and return to these settings once evaluated and treatment is initiated. Support for adherence is critical to successful treatment. Since sleep-related breathing disorders are chronic conditions and may change over time, long-term assessment for successful treatment, including evaluation for changes in weight, cognitive function, sleepiness, and changes in medical conditions, such as diabetes and hypertension are needed. Changes in these related conditions may signal the need for reevaluation in the sleep laboratory. Therefore, ongoing assessment and evaluation should be incorporated into plans of care.

Nurses in clinical, community, and public health settings where there are large numbers of patients who may be at significant risk for sleep-related breathing disorders could play a significant role in raising awareness about signs and symptoms, negative consequences, and the availability of treatment. In addition to in-patient acute care settings, nurses in emergency or trauma centers where patients with injuries that may be associated with sleep disorders are treated should incorporate assessment of the risks for sleep-disordered breathing. Sleep-disordered breathing is also growing in prevalence in children and adolescents. (See Chapter 13, Childhood Sleep Disorders.) Nurses' roles in specific settings are discussed in detail in Chapter 17 (Sleep Promotion in Adult Primary Care Settings), Chapter 19 (Sleep Promotion in Acute Care Settings), and Chapter 20 (Sleep Promotion in Adult Long-term Care Settings).

CONCLUSIONS

Sleep-disordered breathing is a chronic condition that has major implications for morbidity, mortality, and quality of life throughout the world. Although there is a need for additional rigorous research, efficacious treatments are available. Nurses, in collaboration with other health care providers, have important roles to play in assessment, treatment, and supporting long-term management and evaluation of this chronic condition. Additional information is available through the internet (see Table 7.8).

Table 7.8 ■ *Web Sites That Have Information About Sleep Disordered Breathing*

National Sleep Foundation—www.sleepfoundation.org
- Independent nonprofit organization
- Seeks to improve public health and safety through promoting an understanding of sleep and sleep disorders and by supporting sleep-related education, research, and advocacy
- Publishes annual Sleep in America Poll

American Sleep Apnea Association—www.sleepapnea.org
- Nonprofit organization
- Seeks to educate the public about sleep apnea and to serve people with this common disorder
- Promotes education and awareness about sleep apnea through the A.W.A.K.E. Network of voluntary mutual support groups, research, and continuous improvement in care.

American Academy of Sleep Medicine—www.aasmnet.org
- Professional society dedicated exclusively to the medical subspecialty of sleep medicine
- Sets standards and promotes excellence in health care, education, and research in sleep medicine

American Board of Sleep Medicine—www.absm.org
- Professional society that encourages the study, improves the practice, elevates the standards of sleep medicine, and issues certificates of special knowledge in sleep medicine to physicians and PhDs in related fields

Sleep Research Society—www.sleepresearchsociety.org
- Professional society that fosters scientific investigation, professional education, career development, public awareness, and evidence-based health policy in sleep science and academic sleep medicine

Academy of Dental Sleep Medicine—www.dentalsleepmed.org
- Professional society that promotes research, builds collaborations with clinical sleep centers and practices, and provides both public and professional awareness and education about dental treatment options of sleep-related breathing disorders

Sleep Education—www.sleepeducation.com
- From the American Academy of Sleep Medicine, this site is dedicated to providing patients and the general public with information about sleep disorders, diagnostics, treatment-related discussions, and the importance of sleep on health and everyday function

National Library of Medicine—www.nlm.nih.gov/medlineplus/sleepdisorders.html
- MedlinePlus is a publicly available Web site made possible by the National Library of Medicine
- The Web site provides information to patients and the general public about sleep and sleep disorders

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Sleep-Related Movement Disorders and Parasomnias

8

Norma Cuellar and Nancy S. Redeker

Several groups of sleep disorders are associated with abnormal movements and/or emotions that occur at sleep onset during sleep or at sleep–wake transitions. Movement disorders, including periodic limb movement disorder (PLMD) and restless legs syndrome (RLS), contribute to poor sleep, poor quality of life, and poor mental health as a result of abnormal limb movements (usually the legs). Parasomnias are undesirable emotional or physical events and behaviors that occur at the entry to sleep, during sleep, or during arousals from sleep (American Academy of Sleep Medicine, 2005). Although some parasomnias are benign and self-limiting, others are persistent and may be associated with emotional distress and risk of injury. Although both groups of conditions may contribute to sleep loss, morbidity, mortality, and poor quality of life, behavioral and pharmacological treatments are available. The purpose of this chapter is to describe the characteristics, epidemiology, pathophysiology, and treatment of movement disorders (RLS and PLMD) and parasomnias in adults and children and to suggest implications for nursing practice.

MOVEMENT DISORDERS

Periodic Limb Movement Disorder and Periodic Limb Movements During Sleep

PLMD is defined as periodic episodes of repetitive and highly stereotyped limb movements that occur during sleep and clinical sleep disturbance that is not explained by another sleep disorder (see Table 8.1). Limb movements (periodic limb movements during sleep [PLMS]) usually occur in the lower extremities and are characterized by extension of the big toe with partial flexion of the ankle, knee, or hip, but movements can occur in the upper extremities as well.

The symptoms of PLMD vary from night to night. Movements are involuntary, repetitive, and rhythmic and may occur every 20 seconds, significantly interrupting sleep. Severity is determined by the number of movements, with more than 15 movements per hour diagnostic in adults and more than 5 per hour diagnostic in children (American Academy of Sleep Medicine, 2005). However, the absolute cutoff score is not the sole criterion: The diagnosis must take into account sleep disturbance and daytime dysfunction (e.g., fatigue, memory impairment). Polysomnography documents movements that are typically of 0.5 to 5–10 seconds in duration and recur periodically in a series of 4 or more over a 6–90-second period (American Academy of Sleep Medicine, 2005). Patients often report difficulty initiating or maintaining sleep, but may not report symptoms because they are not aware that the limb movements occur. Therefore, it is essential to include information from bed partners or family members who may observe the activity when assessing patients for this condition.

PLMS are usually experienced during non-REM (NREM) sleep stages N1 and N2 (Allena et al., 2009) and are associated with

Table 8.1 ■ *Comparison of Movement Disorders in Sleep*

	Restless Legs Syndrome	Periodic Limb Movement Disorder
Definition (ICSD)[a]	1. Urge to move, usually accompanied by uncomfortable & unpleasant sensations in legs 2. Begins or worsens during periods of immobility or sitting 3. Urge to move or unpleasant sensations partially or totally relieved by movement (walking, stretching) 4. Worse or only occurs at night or in the evening 5. Condition not better explained by another current sleep or other disorder	1. Diagnosed by polysomnography: Repetitive, stereotyped limb movements (0.5–5 seconds duration; of amplitude ≥25% of toe dorsiflexion during calibration; in a sequence of 4 or more movements; separated by an interval of >5 seconds and <90 seconds) 2. PLMS index >5/hour (children) or 15/hour in adults 3. Accompanied by sleep disturbance or fatigue 4. Not explained by other conditions (e.g., apnea)
Sleep	Usually occurs before sleep	Usually occurs during sleep
Sensations and movements	Need to move legs due to sensations; May have uncontrollable movements (PLMS)	No awareness of the leg movements
Symptoms	Usually occur at bedtime but can start in the evening hours Delays sleep onset Intermittent or daily	No awareness of the symptoms as they occur
Outcomes	Daytime sleepiness	Absence of daytime sleepiness
Diagnosis	No definitive diagnostic tool Sleep history is critical 4 "diagnostic criteria" (above)	Electroencephalography (EEG) to measure movement during sleep
Age	May occur in children but the risk increases with age	Usually does not occur in children Symptom onset increases with age
Comorbidity associated with spinal cord involvement	80%–90% usually have PLMS during sleep sensory polyneuropathy	May not have RLS spinal cord lesions
	Depends on abnormal spinal sensorimotor integration at the spinal cord level and abnormal central somatosensory processing	Depends on increased excitability of the spinal cord and a decreased supraspinal inhibitory mechanism

Source: International Classification of Sleep Disorders (2nd ed.) by American Academy of Sleep Medicine, 2005, Westchester, IL: Author.
Abbreviations: PLMS, periodic limb movements during sleep; RLS, restless legs syndrome.

electroencephalogram activation (K complexes) and autonomic arousals (Portaluppi, Cortelli, Buonaura, Smolensky, & Fabbian, 2009; Siddiqui et al., 2007; Walters & Rye, 2009). Autonomic arousals, associated with alterations in cardiac, respiratory (e.g., increases in heart rate and/or respiratory rate, increases in blood pressure), and cerebral activity, are not evident in the EEG or associated with full awakening.

Epidemiology of PLMD and PLMS

PLMD occurs in adults and children and is thought to be quite rare (American Academy of Sleep Medicine, 2005). However, more than 6% of the adult population has PLMS. The prevalence increases with advancing age (Cuellar, Strumpf, & Ratcliffe, 2007) and PLMS may occur in as many as 34% of older adults (American Academy of Sleep Medicine, 2005). As many as 23% of children who reported nonspecific sleep disruption

had PLMS (Martinez & Guilleminault, 2004). African Americans are less likely to experience PLMS than Caucasians. (See Chapter 15, Racial/Ethnic Health Disparities and Sleep Disorders.) These ethnic differences may be genetic in origin, but there is wide variability in the criteria that have been used, and sleep disorders may be under-detected in this population. Therefore, there is a need for further systematic study.

PLMS may also occur in people with sleep-related breathing disorders or narcolepsy, but this does not indicate PLMD. Effective treatment of sleep-related breathing disorders usually addresses PLMS that are not associated with PLMD. While PLMD is a sleep disorder in itself, it is often linked with RLS, since 80% of persons with RLS have periodic limb movements in sleep (PLMS), and PLMS are considered a phenotype of RLS. For this reason, the primary focus of this chapter is on RLS and PLMD as a phenotype of RLS. For similarities and differences between RLS and PLMD, see Table 8.1.

Consequences of PLMD and PLMS

The consequences of PLMD are nonrestorative sleep that may result in excessive daytime sleepiness, fatigue, and performance decrements. The autonomic changes associated with PLMD, including arousals and sympathetic activation, suggest that PLMD may have long-term consequences for cardiovascular health (Allena et al., 2009; Siddiqui et al., 2007), including hypertension, coronary heart disease, or stroke. Daytime sleepiness, cognitive dysfunction, and associated poor quality of life may also be consequences of sleep loss associated with PLMS.

Assessment and Treatment of PLMD

Treatment of PLMD is similar to treatment for RLS (see below). However, levodopa and sedative hypnotics are more often beneficial in patients with PLMD. Because hypnotics may worsen sleep-disordered breathing in people who also have PLMS (Avidan, 2009), health care providers should avoid prescribing them until it is clear that sleep-disordered breathing is not present or that it is effectively treated.

Restless Legs Syndrome

RLS (Wittmaack–Ekbom's or Ekbom's Syndrome) is both a movement disorder and a sleep disorder (Willis, 1685; Wittmaack, 1861). It occurs in adults and children and is associated with discomfort of the legs that is described in various ways, but usually results in an urge to move the legs (see Table 8.1). Persons with RLS describe symptoms in a variety of ways, including "spiders crawling on the legs," "worms inside the legs," restlessness, "pin pricks," or "bubbles" in the legs. The symptoms occur at rest, usually in the evening or at night, at bedtime, or when lying in bed. The symptoms are typically relieved only with movement, such as getting out of bed and walking, kicking, or some other activity. While the symptoms usually occur in the legs, the uncomfortable sensations can also occur in the arms, torso, and phantom limbs. This movement prevents or delays the onset of sleep. Therefore, patients usually report difficulty initiating sleep.

Bed partners usually report that persons with RLS kick throughout the night, a description of PLMS. Since the symptoms occur at night and vary a great deal from patient to patient, and there is no objective measure of RLS, it is difficult to diagnose. The International RLS Study Group established four clinical criteria that are necessary to establish the diagnosis of RLS (see Table 8.1).

The severity of RLS symptoms can be intermittent, daily, or refractory. Intermittent symptoms are troublesome enough to require treatment, but not necessarily daily therapy. Daily symptoms require daily management. Refractory RLS is associated with lack of response to treatment, intolerable side effects from treatment, or uncontrollable rebound or augmentation (worsening of symptoms associated with tolerance to drug treatment) (Silber et al., 2004).

The severity of symptoms may determine the degree of sleep loss and resulting daytime fatigue, daytime sleepiness, poor daytime functioning, depression, poor quality of life, increased morbidity and mortality, and increased health care costs. Symptoms may also

interrupt social and occupation activities that require sitting for extended periods of time. Therefore, these symptoms are quite disabling and limit quality of life.

RLS in Children

Up to 25% of adults with RLS report that their symptoms started as early as of 10 years of age (Picchietti & Stevens, 2008). RLS has a higher prevalence than childhood diseases such as epilepsy and diabetes. The onset of symptoms in children implies a genetic association, with over 70% of children with RLS reporting one parent with the illness (Picchietti & Stevens, 2008; Rajaram, Walters, England, Mehta, & Nizam, 2004).

Approximately 20% of children with RLS have comorbid conditions, including parasomnias, ADHD, anxiety, or depression and/or obstinate defiant disorder (Picchietti et al., 2007). (See Chapter 4, The Nature of Sleep Disorders and Their Impact and Chapter 13, Pediatric Sleep Disorders.) Although children may report aches that may be described as "growing pains" associated with RLS, recent studies have minimized this association (Picchietti & Stevens, 2008). Many children with RLS may be misdiagnosed with attention deficit hyperactivity disorder (ADHD) (Horle & Wood, 2008). These findings underscore the importance of carefully assessing children who have discomfort in their legs for the presence of RLS. Criteria for RLS in children include the four adult criteria and use of developmentally appropriate words to describe leg discomfort; or the four adult criteria and two of the following: sleep disturbance, family history, or polysomnography showing a periodic limb movement index of greater than or equal to 5 per hour (Allen et al., 2003).

Pathophysiological Mechanisms of PLMD and RLS

Researchers believe that primary RLS originates in the central nervous system (CNS) and is the result of dysfunction in brain iron storage that leads to decreased iron in the brain and abnormalities in dopamine metabolism (Satija & Ondo, 2008). The "iron–dopamine connection" is a term used to explain this mechanism (Allen & Earley, 2007) (see Figure 8.1). Despite normal serum iron levels, low ferritin, high transferrin, and low iron concentrations are present in the cerebrospinal fluid (CSF) of patients with RLS. Neuroimaging revealed the presence of morphologic changes in the somatosensory cortex, motor cortex, and thalamic gray matter; has identified the role of iron and dopamine metabolism dysfunction in the pathogenesis of RLS; and has also identified the role of limbic and opioid systems in the pathophysiology of RLS (Connor et al., 2009; Snyder et al., 2009).

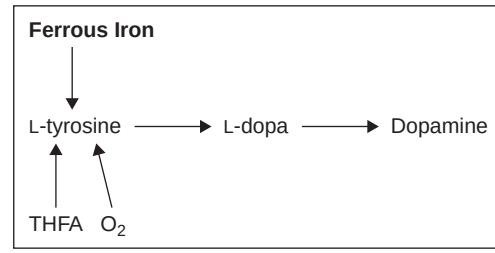

Figure 8.1 ■ Iron-Dopamine Connection in RLS.

Serum ferritin is the best measure of peripheral iron storage in persons with RLS because of the positive correlation with serum ferritin in the cerebral spinal fluid (O'Keeffe, 2005b; Sun, Chen, Ho, Earley, & Allen, 1998). A serum ferritin level of less than 50 ng/mL is correlated with increased RLS symptoms (O'Keeffe, 2005a; Sun et al., 1998), and serum ferritin concentration is used as the marker to initiate iron therapy in persons with RLS.

Dopamine, a monoamine neurotransmitter, is produced primarily in the substantia nigra, the area of the brain associated with movement and the pathophysiology of certain addictive disorders. Dopamine is responsible for the activation of five dopamine receptors and plays an important role in behavior and cognition, voluntary movement, motivation, and reward. Each of the dopamine receptor sites plays a role in dopamine metabolism, a clinically significant dimension of pathology in RLS.

The beneficial effects of dopaminergic drugs in patients with RLS suggest that it is caused by localized, abnormal function of the CNS,

rather than the peripheral nervous system. Symptoms of RLS, including PLMS, have been associated with spinal cord lesions. However, the finding that patients who have PLMD do not respond to dopaminergic agents as consistently as RLS patients suggests that the symptoms of these two conditions originate in different sites. Dopaminergic drugs differ in their mechanisms of action and may have affinity for specific dopamine receptors based on the pharmacokinetic profile of the drug. (See Chapter 13, Pediatric Sleep Disorders.) If it is known which dopamine receptor site is implicated in the pathophysiology of RLS, medications can be selected to specifically target that receptor site.

Genetics of RLS

Genetic factors play an important role in RLS. Researchers and clinicians have observed family associations in RLS since the 1800s. Geneticists believe that RLS is a Mendelian autosomal dominant disease with variable expressivity and high penetrance (Esteves et al., 2008; Kemlink et al., 2007; Mathias et al., 2006; Pichler et al., 2006; Winkelmann et al., 2002). This suggests that the affected person had one parent who was a genetic carrier of the disease. However, researchers have found other genetic patterns that may explain the occurrence of RLS (Desautels et al., 2005; Esteves et al., 2008).

It has been difficult to identify the genetic variations of RLS because there are several allelic forms (polymorphisms) (Schormair et al., 2008; Stefansson et al., 2007; Winkelmann, Polo et al., 2007; Winkelmann, Schormair et al., 2007). Although researchers have not identified a single gene as causative of the disorder, genome-wide associations have been found (Mignot, 2007), and at least one of the genes (BTBD9) has been linked with PLMS (Kemlink et al., 2008; Lohmann-Hedrich et al., 2008). BTBD9 is related to decrements in iron stores and ethnic differences in RLS prevalence (Trotti, Bhadriraju, & Rye, 2008).

Phenotypes of RLS

Previously RLS was categorized as primary (idiopathic or genetic) or secondary (associated with comorbidity and originally considered reversible) with no difference in symptom presentation (Cuellar, 2004; Gamaldo & Earley, 2006). RLS has several phenotypical presentations (see Table 8.2). Although this theoretical perspective of RLS may be changing based on advances in genetics and studies of mechanism of RLS, iron deficiency due to various causes may contribute to RLS. Symptoms of RLS may also occur during pregnancy (especially the last trimester) and in patients with end-stage renal disease, and are associated with iron-deficiency anemia and use of some types of drugs (see Table 8.3).

Table 8.2 ■ *Phenotypes of RLS*

- Age of onset separates primary versus secondary RLS
  (Allen & Early, 2000; Montplaisir et al., 1997; Walters et al., 1996; Winkelmann et al., 2000)
- Progression of symptoms (slow or fast)
  (Allen & Early, 2000)
- Variable presentation; mild symptoms often delay a definitive diagnosis of RLS (Montplaisir et al., 1997)
- Motor or sensory symptoms or both
  (Montplaisir et al., 1985)
- Family history
  (Allen & Early, 2000; Allen & Earley, 2001a; Desai, Cherkas, Spector, & Williams, 2004; Ekbom, 1960; Montplaisir et al., 1997; Ondo, He, Rajasekaran, & Le, 2000; Walters et al., 1996; Winkelmann et al., 2000)
- Iron metabolism
  (Allen & Early, 2000; Xiong et al., 2007)
- Presence of periodic limb movements
  (Bonati et al., 2003; Esteves et al., 2008; Montplaisir et al., 1997; Walters et al., 1996)

Table 8.3 ■ *Drugs Associated with Exacerbation of RLS Symptoms*

Antiemetics	Meclizine (Antivert, Bonine) Prochlorperazine (Compazine) Promethazine (Phenergan) Metoclopramide (Reglan) Trimethobenzamide (Tigan)
Dopamine antagonists	Haloperidol (Haldol) Loxapine succinate (Loxitane) Thioridazine (Mellaril) Molindone (Moban) Thiothixene (Navane) Fluphenazine (Prolixin) Risperidone (Risperdal) Mesoridazine besylate (Serentil) Quetiapine fumarate (Seroquel) Trifluoperazine (Stelazine) Chlorpromazine (Thorazine) Perphenazine (Trilafon) Triflupromazine (Vesprin) Olanzapine (Zyprexa)
Antianxiety agents	Hydroxyzine (Atarax)
Antidepressants	ALL **especially fluoxetine (Prozac) and sertraline (Zoloft)
Antihistamines	ALL **especially diphenhydramine, including over-the-counter medications that have combinations with diphenhydramine (Benadryl)

Prevalence of RLS

Between 5% and 15% of the general population may have RLS (Phillips, Hening, Britz, & Mannino, 2006). However, it is difficult to determine the exact prevalence due to variations in the criteria used to diagnose it (all four of the standard criteria vs. fewer). Although most studies report no difference by gender (Winkelman, Finn, & Young, 2006), approximately twice as many older women as men have this condition in some countries (Rijsman, Neven, Graffelman, Kemp, & de Weerd, 2004). There is considerable variability in prevalence of RLS by geographical areas that may be explained by genetics (see Table 8.4).

Although African Americans and Asians are least likely to have RLS or PLMD when compared with Caucasians and Native Americans, the majority of available data have been obtained in Caucasians. (See Chapter 15, Racial/Ethnic Health Disparities and Sleep Disorders.) However, one study reported the prevalence of RLS in Native Americans at 17.7% and another reported the prevalence at 4.7% in African Americans in Baltimore—higher than the 3.8% noted in Caucasians. Higher rates may be due to increased comorbidity in these populations. However, further study of racial differences, treatment, and barriers to access to care should be investigated (Lee et al., 2006).

Assessment and Diagnosis of RLS

A thorough sleep and health history and physical examination are necessary to diagnose RLS. (See Chapter 5, Conducting a Sleep Assessment.) Diagnosis of RLS is based on four criteria (see Tables 8.1 and 8.5—Nursing Implications) and supportive clinical features, including a positive response to dopamine agonist drugs, periodic limb movements, family history, and the exclusion of conditions that can mimic RLS, including akathesias, positional discomfort, leg cramps, peripheral neuropathy, arthritis,

Table 8.4 ■ *Prevalence of RLS by Geographic Region*

Canada	15%	Lavigne & Montplaisir, 1994
Norway and Denmark	11.5%	Bjorvatn et al., 2005
Sweden	11.4%	Ulfberg, Nyström, Carter, & Edling, 2001
Argentina	10.8%	Persi et al., 2009
Germany	10.6%	Berger, Luedemann, Trenkwalder, John, & Kessler, 2004
Italy	10.6%	Högl et al., 2005
United States	9.7%	Phillips et al., 2006
France	8.5%	Garbarino et al., 2002
Italy	8.5%	Tison et al., 2005
Ecuador	3.2%	Castillo, Kaplan, Lin, Fredrickson, & Mahowald, 2006
Turkey	3.2%	Sevim et al., 2003
Japan	1.5%	Kageyama et al., 2000
Korea	1%	Cho et al., 2008
Singapore	1%	Tan et al., 2001

and some anxiety disorders. These conditions have been described as "RLS-like syndrome" and result in 25% of false-positive diagnoses (Benes, Walters, Allen, Hening, & Kohnen, 2007; Benes, von Eye, & Kohnen, 2009; Hening, Allen, Tenzer, & Winkelman, 2007). If the patient responds to dopamine medications and has met the four diagnostic criteria, the sensitivity and specificity of correct diagnosis increases from 69% to 88% (Benes & Kohnen, 2009). A detailed algorithm designed to be used in primary care settings is available (Adler et al., 2004).

Older adults who have cognitive impairment have difficulty verbalizing RLS symptoms. Among patients with dementia, the most common risk factors for RLS were a periodic leg movement sleep index above 15 (54.55%) and use of selective serotonin reuptake inhibitors (SSRIs) (34.78%). The most common RLS-associated behaviors were repetitious mannerisms (56.52%) and general restlessness (34.78%) among patients with dementia (Richards, Shue, Beck, Lambert, & Bliwise, 2010). These findings suggest the importance of behavioral observation and the need for objective diagnostic evaluation in this population.

A major component of a health history is assessing the patient for factors that may mimic RLS (see above). A careful medication review may reveal the use of prescribed or over-the-counter medications that may contribute to RLS symptoms (see Table 8.3).

Serum testing for iron measures should be a priority, based on the acronym F.I.T. (ferritin, iron, and transferrin). Assessment of serum ferritin is the minimal requirement if cost is a concern. Laboratory tests used to rule out secondary RLS are listed in Table 8.6. Although objective measures are not needed to diagnose RLS, polysomnography, actigraphy, actometry, and the Suggested Immobilization Test (Montplaisir et al., 1998), a procedure that involves provoking the RLS symptoms by having the patient sit quietly may be useful.

Sleep diaries are essential for the description of RLS symptoms. A variety of screening tools have been developed to determine the severity of RLS as well as health outcomes related to RLS. See Table 8.7 for a description of these instruments.

Treatment of RLS and PLMD

Treatment of RLS includes behavioral and pharmacological modalities, is individualized based on the patient's frequency of symptoms, comorbid conditions, and medications, and focused on symptom reduction and improvements in sleep and quality of life (Cuellar & Ratcliffe,

Table 8.5 ■ *Nursing Implications Related to the Care of Patients With RLS*

- Include screening for RLS in all patient assessments across the lifespan, especially in:
  - Children and older adults who may not be able to verbalize symptoms
  - Children with ADHD symptoms
  - Pregnant women who may be at risk due to anemia
  - Patients with renal disorders/uremia
- Health and sleep history
  - Rule out conditions that mimic RLS
  - Medication history
  - Characteristics of RLS symptoms
  - Sleep complaints
  - Impact on daytime function: excessive daytime sleepiness, quality of life, fatigue, mental health, hyperactivity, social functioning
- Teach effective sleep promotion habits/sleep hygiene
  - Regularize patterns of sleep/wake
  - Adequate sleep duration
  - Avoid caffeine
- Consider nonpharmacological options (cognitive therapy, relaxation)
- Carefully titrate medications and evaluate response, including the potential for augmentation
- Teach patients and health care providers to avoid the use of over-the-counter medications (e.g., diphenhydramine) that may exacerbate RLS

Table 8.6 ■ *Laboratory Tests Used to Rule Out RLS*

- Anemia: serum for reticulocyte count, ferritin, CBC, iron, total iron binding capacity, iron saturation, transferrin, folate
- Diabetes: fasting plasma glucose, glucose tolerance test
- Renal disease: BUN, creatinine, protein, albumin, 24-hour urine, urine creatinine clearance
- Electrolyte imbalance: electrolyte panel, calcium, magnesium
- Neurological tests: electromyogram (EMG), nerve conduction studies, MRI (magnetic resonance imaging)
- Vascular tests: ultrasounds, Doppler studies, angiograms

2008; Satija & Ondo, 2008). It is imperative to treat and control any secondary comorbid conditions that may exacerbate RLS (e.g., diabetes, hypertension, thyroid problems, and electrolyte imbalances).

Nonpharmacological Treatment. Nurses play an important role in educating patients and their families regarding RLS and PLMD and assisting them to obtain accurate information. There are numerous resources available, including those on the Internet (see Table 8.8). However, patients often need assistance in identifying the Internet sites with the most credible information.

Promoting restorative sleep is a critical goal. Therefore, the use of substances that may interfere with sleep (e.g., caffeine and alcohol) should be evaluated and eliminated. Sleep hygiene is important and sleep behaviors should include a regular sleep routine in a comfortable environment, going to bed at the same time every night, as well as waking up at the same time each day, and obtaining sufficient sleep. Relaxation techniques and engaging in activities like reading may keep the mind stimulated and reduce the focus on the irritating symptoms. Moderate exercise should be encouraged. Behavioral modification includes learning coping strategies and attending support groups to learn how to manage the symptoms of RLS. Cognitive behavioral therapy, specifically for patients with RLS, is beneficial (Hornyak et al., 2008; Trenkwalder, 2006).

Table 8.7 ■ *Questionnaires Used to Screen for RLS*

Title of Instrument and Author	Description
Diagnostic Scales	
Single Question (Ferri et al., 2007)	Rapid screening of RLS Single question Diagnosis must be confirmed by diagnostic and supportive criteria
Hopkins Telephone Diagnostic Interview for RLS (TDI) (Hening, Allen, Washburn, Lesage, & Earley, 2008)	Structured diagnostic interview based on clinical features to guide accurate diagnosis of RLS Must be trained to use the scale
RLS Diagnostic Index (RLS-DI) (Benes & Kohnen, 2009)	Diagnosis of RLS by less experienced health care providers Weighted by standard and supportive criteria of RLS Can be used for patients with mimics of RLS Cutoff score for diagnostic certainty is reported
Cambridge-Hopkins Questionnaire (CH-RLSq) (Allen, Burchell, MacDonald, Hening, & Earley, 2009)	Identifies the four basic diagnostic features and provides differential diagnosis Reasonable level of sensitivity and specificity for population-based studies
L-Dopa Test (Stiasny-Kolster, Kohnen, Moller, Trenkwalder, & Oertel, 2006)	Standardized test procedure using a single oral dose of L-Dopa to evaluate response to dopaminergics as a supportive diagnosis criterion
Severity Scales	
John Hopkins RLS Severity Scale (Allen & Earley, 2001b)	Severity of RLS by time of day when symptoms start Single question Easy to use
International RLS Study Group Rating Scale (IRLSSG) (Walters et al., 2003)	Rating scale for severity of RLS symptoms Considered Gold Standard for assessing severity Might give false-positive score for mimics of RLS
RLS-6 Scale (Kohnen, Oertel, Stiasny, Benes, & Trenkwalder, 2004)	Severity of RLS and assessing sleep satisfaction and daytime sleepiness Can be used for the assessment of efficacy of drug treatment in RLS drug studies
Quality of Life Scales	
Restless Legs Quality of Life Instrument (RLS-QLI) (Atkinson et al., 2004)	Consequences of RLS that affect daily functioning, social functioning, sleep quality, and emotional well-being Not commonly used in research
Restless Legs Syndrome Quality of Life Questionnaire (QoL-RLS) (Kohnen, Benes, Heinrich, & Kurella, 2002)	Consequences of RLS on sleep, ADLs, mood, and social interactions
Hopkins RLS Quality of Life Questionnaire (RLSQoL) (Abetz, Arbuckle, Allen, Mavraki, & Kirsch, 2005)	Consequences of RLS on work life, as well as sleep, emotional well-being, and social life
Augmentation Scales	
Augmentation Severity Rating Scale (ASRS) (Garcia-Borreguero et al., 2007)	Quantify the severity of augmentation Determines the varying degrees of augmentation Used in clinical drug trials
Structured Interview for Diagnosis of Augmentation (SIDA) (Högl, Garcia-Borreguero, Gschliesser, et al., 2005)	Based on published criteria for augmentation

Table 8.8 ■ *Internet Resources on RLS*

Restless Legs Syndrome Foundation	www.rls.org	■ A nonprofit 501 (c)(3) agency ■ Goals are to increase awareness of RLS, to improve treatments, and, through research, to find a cure for persons with RLS ■ Provides information to lay persons on support groups and networks
National Institute of Neurological Disorders and Stroke	www.ninds.nih.gov	■ Mission of NINDS, out of NIH, is to reduce the burden of neurological disease—provides information to persons with neurological conditions including RLS, as well as information on research and funding in this area
WE MOVE: Worldwide Education & Awareness for Movement Disorders	www.wemove.org/rls.html	■ A nonprofit 501 (c)(3) agency ■ Comprehensive resource for movement disorder information and education ■ Developed to educate lay persons and health professionals on all movement disorders, provide resources and educational materials, and assist in the establishment and maintenance of support groups

Abbreviations: ADLs, activities of daily living; RLS, restless leg syndrome.

Herbs and nutritional supplements may be used to correct deficiencies in vitamins and electrolytes, such as folate, magnesium, and calcium. Valerian, an herb, is helpful in managing the symptoms of RLS (Cuellar & Ratcliffe, 2009). Patients report beneficial effects from acupuncture, massage therapy, meditation, music, cold or heat compresses, vibration, and transcutaneous electric nerve stimulation (TENS), but there is limited research on the efficacy of these treatments (Cuellar et al., 2004; Oertel et al., 2007). (See Chapter 14, Complementary and Alternative Medicine [CAM] and Sleep.)

Oral administration of iron supplements generally corrects iron deficiency and reduces RLS symptoms in those with ferritin levels below 50 ng/mL (Benz, Pressman, Hovick, & Peterson, 1999; Hening, 2007; Silber et al., 2004; Sun et al., 1998). Ferrous sulfate 325 mg with 500 mg of vitamin C should be given three times a day between meals, if tolerated. Increased augmentation (exacerbation of symptoms) may be seen in patients with low ferritin levels who are treated with dopaminergic drugs before iron therapy (Trenkwalder, Hogl, Benes, & Kohnen, 2008).

Pharmacological treatment. When lifestyle changes and nonpharmacological approaches cannot control symptoms, pharmacological treatment should be considered. Dopaminergics (dopamine precursors, dopamine receptor agonists), benzodiazepines, opioids, and other anticonvulsants are used in monotherapy or in combination in the treatment of RLS (see Table 8.9). An algorithm for the treatment of intermittent, daily, and refractory RLS using single or combination of a variety of pharmacological agents is available (Silber et al., 2004) and online (Adler et al., 2004) (see references). There are two major side effects of dopaminergic medications: augmentation and rebound (Satija & Ondo, 2008). Augmentation is the worsening of symptoms when symptoms are expected to get better, earlier onset of symptoms by at least 2 hours, or the spread of symptoms to additional limbs (the other leg or arms). Rebound is worsening of symptoms at the end of the dosing period, or when the medication is wearing off. Symptoms may be worse than before treatment started. A useful and detailed algorithm for use of these medications is available for primary care providers online

Table 8.9 ■ *Pharmacological Treatment of Restless Legs Syndrome*

Drug Class	Drug	Dose Range	Indications	Side Effects	Notes
Dopamine precursors	Levodopa	25–200 mg	Intermittent RLS	Nausea; libido; drowsiness; vomiting; dizziness; headache; tiredness; impulsivity related to gambling, spending; increased libido	Not recommended as first-line treatment; doses >200 mg may increase augmentation (worsening of symptoms)
Dopamine agonists nonergot derivative	Ropinirole (Requip)	0.25–4.0 mg	Intermittent, daily, or refractory RLS		Take 1–2 hours before onset of symptoms; rotigotine is formulated as patch for 24-hour wear
	Pramipexole (Mirapex)	0.125–2.0 mg			
	Rotigotine (Neupro)	0.5–4.0 mg/patch			
Dopamine agonist ergot derivative	Bromocriptine (Parlodel)	2.5–7.5	Daily or refractory RLS	Pergolide side effects: valvulopathies, valvular fibrosis, constrictive pericarditis, pleural pulmonary fibrosis; may need to discontinue use; Few patients develop addiction; Sedation; constipation, nausea; vomiting	
	Pergolide (Permax)	0.125–0.75			
	Carbegoline (Dostinex)	0.25–4 mg			
Opioid agonists	Tramadol	50–100 mg	Intermittent or daily RLS		Low potency; If not effective, use higher potentcy opioids
Opioids	Codeine	15–120 mg	Intermittent or daily RLS		2nd drug of choice; can be used for intermittent symptoms; not used for pain but for impact on dopamine system
	Oxycodone	2.5–20 mg	Daily RLS		Monitor for respiratory problems that may be worsened by comorbid conditions or other medications which may result in respiratory depression
	Methadone	5–40 mg	Refractory RLS		
Anticonvulsants	Gabapentin (Neurotonin)	300–1,200 mg	Daily or refractory RLS	Sedation, dizziness, fatigue, somnolence, ataxia	Use in addition to other treatments or as monotherapy; Gabapentin is beneficial in patients with RLS who complain of painful symptoms; not considered the first line of treatment; Efficacy of this drug class is modest compared to other possible options
	Carbamazepine (Tegretol)	50–800 mg			
	Valproic acid (Depakote)	500–1,000 mg			

(Continued)

Table 8.9 ■ *Pharmacological Treatment of Restless Legs Syndrome (Continued)*

Drug Class	Drug	Dose Range	Indications	Side Effects	Notes
Benzodiazepine receptor agonists	Zolpidem (Ambien)	5–10 mg	Intermittent or refractory RLS	Side effects are reversible and usually include morning sedation and daytime somnolence, or a hangover effect May develop tolerance	Short acting; promote sleep onset but not with nighttime awakenings; may be used as monotherapy or as combination treatment; reduce arousals; not as effective in treating the motor and sensory symptoms of RLS.
	Zaleplon (Sonata)	5–10 mg			
Benzodiazepines	Clonazepam (Klonopin)	0.5–2 mg	Intermittent or refractory RLS		
	Flurazepam (Dalmane)	15–60 mg			
	Temazepam (Restoril)	15–30 mg			
	Triazolam (Halcion)	0.125–0.5 mg			

(Adler et al., 2004). Implications for nursing practice are presented in Table 8.5.

PARASOMNIAS

Parasomnias are undesirable and abnormal movements, behaviors, sensations, emotions, and/or autonomic activity that occur during sleep and transitions between sleep and wake. They are classified as (1) disorders of arousal from nonrapid eye movement sleep, (2) parasomnias usually associated with rapid eye movement sleep, and (3) other parasomnias (American Academy of Sleep Medicine, 2005). Many parasomnias are infrequent and self-limiting. However, others, such as REM Behavior Disorder, may be associated with motor activity and agitation that may lead to injury to self or others. Some have resulted in violent behavior. Parasomnias may also lead to disrupted sleep for individuals who experience them or family members who may be awakened by the behaviors. Therefore, they may become a family concern (Avidan & Kaplish, 2010). Parasomnias are most common in children, but occur in approximately 4% of adults (Ohayon, Guilleminault, & Priest, 1999). An overview of parasomnias with detailed case studies and examples of polysomnographic findings is available (Avidan & Kaplish, 2010).

Disorders of Arousal

Disorders of arousal occur during slow wave sleep (SWS) and most frequently occur during the first half of the night. They are most common in children and least common in the aged. Common disorders of arousal include confusional arousals, sleepwalking, and sleep terrors. Genetic factors, as well as other sleep disorders, such as RLS and sleep-disordered breathing, and sleep deprivation contribute to the behaviors associated with these disorders (Bloomfield & Shatkin, 2009).

Confusional Arousals

Confusional arousals are associated with mental confusion, disorientation, or confused behavior following arousals from sleep. The individual is usually aroused from SWS during the first half of the night (American Sleep Disorders Association, 2005; Avidan, 2009). Patients show inappropriate behaviors (e.g., sexually aggressive behavior), have sleep inertia (grogginess or inability to function upon awakening), are slow to respond to questioning, and have retrograde amnesia (fail to remember things happening immediately before the event). Some individuals may respond with physically forceful behavior if awakened by another individual.

Confusional arousals are most common in infants and toddlers (Avidan, 2009) and occur in 17% of children between the ages of 3 and 13 years (Kotagal, 2009). They may be confused with nightmare or sleep terrors. Children may moan softly or cry, move around or become increasingly agitated, become difficulty to arouse and console, and then fall asleep (Bloomfield & Shatkin, 2009). Upon awakening, children have amnesia for the events. Confusional arousals usually diminish as the child grows older.

Therefore, education and reassurance of parents is a critical component of nursing care.

The prevalence of confusional arousals in people over the age of 15 is between 2.9% and 4.2% (American Sleep Disorders Association, 2005). They can be precipitated by sleep deprivation, fever, or circadian rhythm disorders, including shift work, and use of CNS depressants (sedative hypnotics, alcohol, antihistamines) (Avidan, 2009; Avidan & Kaplish, 2010). Sleep-disordered breathing and PLMS can also lead to behaviors similar to confusional arousals.

Somnambulism/Sleepwalking

Sleepwalking is characterized by ambulation during sleep and is associated with difficulty in arousal, mental confusion upon awakening, amnesia, routine behaviors at inappropriate times, inappropriate or nonsensical behaviors, and/or dangerous behaviors (American Sleep Disorders Association, 2005). The patient is usually calm and appears with open eyes and a glassy stare. Inappropriate, but apparently purposeful behaviors, such as eating or moving furniture, may occur, but violent behavior can also occur, especially in men, if the person is suddenly awakened. Injury can also occur to the individual who walks outside or performs other inappropriate behavior during episodes of somnambulism. Like confusional arousals, somnambulism typically occurs during the first half or third of the night and in SWS.

Sleep walking occurs in as many as 17% of children and occurs more often between the ages of 8 and 12 (American Sleep Disorders Association, 2005). Sleepwalking occurs in as many as 4% of adults. Although the prevalence does not significantly differ between genders, men are more likely than women to experience injury or violence. Acute sleep deprivation, use of sedatives, fever, and other sleep disorders may result in altered sleep architecture that then leads to somnambulism.

Sleep Terrors

Sleep terrors are characterized by sudden episodes of terror during sleep. An episode usually starts with a loud cry or scream accompanied by behavioral and autonomic manifestations (tachycardia, tachypnea, diaphoresis, and increased muscle tone), intense fear, and at least one of the following: difficulty in arousal, mental confusion, partial or complete amnesia for the episode, and dangerous or potentially dangerous behaviors (American Sleep Disorders Association, 2005). These episodes may become violent. Unlike nightmare, the individual has amnesia for the event (Avidan, 2009). Sleep terrors may be confused with epilepsy.

Sleep terrors occur in 1.1%–6.2% of children and 2.2% of adults (American Sleep Disorders Association, 2005). They may be associated with anxiety disorders and bipolar disorder in adults.

Diagnosis of Arousal Disorders

Because a cardinal feature of arousal disorders is their occurrence during SWS that most frequently occurs early in the sleep period, patients usually report symptoms of these conditions during the first portion of the nocturnal sleep period. The two principle characteristics used to establish the diagnosis are (1) the timing of the episodes relative to the nocturnal sleep period and (2) observer reports of behaviors consistent with each of the specific criteria. Polysomnography with video monitoring, sometimes performed in the home, is helpful in establishing the diagnosis and ruling out contributing sleep disorders, but is not usually indicated unless the parasomnias are persistent or associated with injury.

Treatment of Arousal Disorders

Often parasomnias are normal and self-limiting. Therefore, patient education and reassurance of patients and family members (e.g., parents of young children) is a very important component of nursing care. Given the high prevalence of occasional parasomnias in young children and the concerns that they may raise for parents, reassurance and education should be a standard element of well-child pediatric care. In most cases, awakening the individual in

the midst of an episode of parasomnia should be avoided due to the likelihood that it will increase agitation.

Given the high risk of injury resulting from somnambulism, safety is a paramount consideration. It may be necessary to add locks or alarms to doors or windows to prevent the sleepwalker from going outdoors. To avoid falling down the stairs, use of barriers to the stairs or sleeping on the first floor may be helpful.

Good sleep hygiene, including minimizing use of caffeine and late night stimulation through exercise or use of the television or computer should be encouraged. Normalizing sleep–wake schedules and providing adequate sleep is likely to reduce sleep deprivation and prevent some occurrence of parasomnias.

Treatment of other sleep disorders, such as sleep-disordered breathing, RLS, or PLMD that may contribute to parasomnias is indicated. Use of low-dose benzodiazepines (such as clonazepam or diazepam) or trycyclic antidepressants at bedtime may also be helpful (Avidan, 2009).

Parasomnias That Occur During REM Sleep

Several types of parasomnias occur during REM sleep, much of which occurs during the latter half of the night. Thus, these disorders typically are associated with that portion of the nocturnal sleep period.

Nightmares

Nightmares are vivid dreams that are intense or anxiety provoking and are a virtually universal experience on an occasional basis. Nightmare disorder encompasses episodes of awakenings with recall of intensely disturbing dreams that usually involve fear or anxiety, but may include anger, sadness, or despair. Patients are completely alert upon awakening and able to recall the dream content. The diagnosis of nightmare disorders must include either delay in returning to sleep after the episode or occurrence of the nightmare in the latter part of the night (American Sleep Disorders Association, 2005). Because nightmares occur in REM sleep, a stage during which there is muscle atonia, no movements are associated with them.

Eighty-five percent of adults have occasional nightmares or bad dreams with 8% reporting recurrent nightmares. As well, children frequently have nightmares with up to 50% of nightmares severe enough to disturb their parents (American Sleep Disorders Association, 2005). Nightmares that begin in childhood may persist into adulthood.

Recurrent nightmares are associated with anxiety and anxiety disorders in both children and adults. Physical and sexual abuse frequently leads to posttraumatic stress disorders, and nightmares are a cardinal feature of this condition. Persistent nightmares are associated with a three- and five-fold increase in the rate of psychiatric disorders in children and adults, respectively (Bloomfield & Shatkin, 2009). Nightmares are also associated with temperament and separation anxiety in children (Bloomfield & Shatkin, 2009).

Occasional nightmares do not require treatment and are considered normal. Therefore, reassurance of parents of young children is particularly important. Parents may find it helpful to reassure young children that their dream content is not real. Reduction in exposure to stressful events during the daytime and violent or disturbing media content, as well as writing or drawing dreams, and hypnotherapy, may also be effective. Rescripting techniques, writing or drawing dreams, and hypnotherapy are helpful (Bloomfield & Shatkin, 2009).

Persistent nightmares are a sign that suggests the presence of an underlying mental health condition. Therefore, reports of persistent nightmares should target the need for more in-depth psychosocial assessment. Psychotherapy and/or medications may be indicated.

Rapid Eye Movement Sleep Behavior Disorder

Rapid eye movement sleep behavior disorder (RBD) is the presence of REM sleep without atonia, as determined by electroencephalography obtained as a component of PSG. Patients have one or more of the following: "sleep related injurious, potentially injurious, or disruptive

behaviors by history" or "abnormal REM sleep behaviors documented by PSG monitoring" (American Sleep Disorders Association, 2005). These events occur in the absence of seizure activity or other sleep disorders. Often, the event that precipitates a health care consultation is a sleep-related injury either to the patients with RBD or their bed partner. Lacerations, fractures, bruises, and other injuries result from attempts to enact an unpleasant or violent dream in which the patient perceives himself/herself as being attached or chased by unfamiliar people or animals. Usually the individual awakens at the end of the episode and becomes alert, explaining the story. Physical behavior may include talking, laughing, swearing, and/or flailing of the arms and legs, but the individual usually does not get out of bed (American Academy of Sleep Disorders, 2005).

The frequency of RBD episodes is variable. RBD usually occurs at least 90 minutes into the sleep cycle because it occurs in REM sleep. (See Chapter 1, Physiological and Behavioral Aspects of Sleep.) It may occur early in the nocturnal sleep period in patients who have narcolepsy.

RBD is most common in middle-aged and older men, with a prevalence of 0.38% in the general population and 0.5% in the elderly population (American Academy of Sleep Disorders, 2005). More than half of the cases of RBD in adults are idiopathic. Other cases are associated with PLMS, neurological disorders, such as parkinsonism, dementia with Lewy bodies, stroke, and narcolepsy, as well as posttraumatic stress disorder (Avidan, 2009). Tricyclic medications, SSRIs, and MAOI drugs may trigger or exacerbate RDB. Predisposing factors in children and adolescents include narcolepsy, psychotropic medications, brainstem tumors, parkinsonism, and Tourette's syndrome (American Sleep Disorders Association, 2005). Acute RBD may be associated with drug toxicity or metabolic abnormalities, such as those associated with withdrawal from alcohol or sedative hypnotics.

Safety is a paramount concern for patients and bed partners of patients with RBD. There are numerous case reports of elaborate methods to avoid injuring their bed partners (e.g., handcuffs, constructing barriers in the bed); especially because they are not aware that RBD is abnormal and treatable. Barricading windows or sleeping on the floor in a sleeping bag may be needed to promote safety until pharmacological treatment is successful. Medication treatment is indicated in patients at high risk for injury. Clonazepam (0.25–1.0 mg orally before bedtime) is effective in reducing arousals. Imipramine, carbamezepine, pramipexole, and levodopa may also be helpful. Although melatonin may be effective, its use may be limited by the inconsistencies in available preparations due to lack of FDA regulation (Avidan, 2009).

Isolated Sleep Paralysis

Over half of the individuals experience occasional inability to perform voluntary motor activities at the onset of sleep and at the time of awakening. This is due to the intrusion of REM sleep (associated with atonia) into awakening. Although this may result in concern, treatment is usually unnecessary. Reassurance is usually sufficient. Patients should be encouraged to avoid irregular sleep schedules and avoid sleep deprivation. If persistent, anxiolytic medications may be useful.

CONCLUSIONS

Parasomnias and movement disorders are associated with many behaviors that occur in proximity to the sleep period during sleep stage transitions, or during REM or NREM sleep. Movement disorders, in particular, are associated with significant impairment in quality of life and possibly negative cardiovascular consequences.

Both groups of conditions occur in adults and children. Although some conditions are occasional, benign, and self-limiting, others are persistent and associated with significant sleep loss and/or the risk of injury to self and others. Some (e.g., restless legs, persistent nightmares, REM behavior disorder) may be signs of comorbid psychiatric or neurological disorders,

underlying sleep disorders, or the effects of toxic substances or metabolic abnormalities. Therefore, eliciting patients' or family members' (e.g., parents or bed partners) descriptions of these events is a critical component of nursing assessment. Nursing care for movement disorders and parasomnias is focused on patient education, providing appropriate reassurance regarding benign and self-limiting behaviors, a safe environment, and symptom control where necessary. Sleep hygiene, avoiding caffeine, and regularly scheduled sleep–wake cycle often reduces negative consequences. Treatment with medication is useful and available for many of these disorders, but should be used in combination with behavioral approaches as discussed in this chapter.

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9 Narcolepsy

Ann E. Rogers

Narcolepsy is a potentially disabling hypersomnia of central origin (American Academy of Sleep Medicine, 2005) that is associated with dysregulation of sleep and waking states. It affects 0.02%–0.5% of the U.S. population (see more on epidemiology below) and is associated with excessive daytime sleepiness, increased risk for injury, and poor quality of life. Although less prevalent than some other sleep disorders, such as sleep apnea and insomnia, narcolepsy is very important because studies of this condition have provided scientists with insight into basic mechanisms of sleep–wake regulation. Although the exact cause of narcolepsy is unknown, causes appear to be multifactorial, for example, genetics, infection, stress, and low levels of the brain neurochemical hypocretin. Effective treatments are available and can improve this chronic condition, as well as its negative consequences. The purpose of this chapter is to describe the characteristics and consequences of narcolepsy and strategies for assessment and treatment of this sleep disorder. Implications for nursing practice are discussed.

CHARACTERISTICS OF NARCOLEPSY

Common symptoms of narcolepsy include excessive daytime sleepiness, cataplexy, hypnagogic hallucinations, sleep paralysis, and fragmented nocturnal sleep. However, the diagnostic criteria for narcolepsy vary somewhat, depending on whether the patient also has cataplexy or not (see Table 9.1).

Excessive Daytime Sleepiness

All patients with narcolepsy develop excessive daytime sleepiness that usually takes the form of brief, multiple refreshing naps (sleep attacks) throughout the day. These can occur without warning or be preceded by a period of drowsiness. Following arousal from a nap, the narcoleptic individual usually feels refreshed and often has a refractory period of 1–2 hours before the next sleep attack. Many narcoleptic patients show a "waxing and waning" pattern of alertness and attention, that is, periods of alertness alternating with profound and debilitating sleepiness. Some patients also complain of abnormal drowsiness throughout the day (Guilleminault & Fromherz, 2005).

Fragmented Sleep

Patients with narcolepsy often report shortened and fragmented nocturnal sleep. Unlike patients with other sleep disorders, poor nocturnal sleep is not the cause of excessive daytime sleepiness in patients with narcolepsy. Rather, disrupted nocturnal sleep in patients who have narcolepsy usually develops several years after the appearance of excessive daytime sleepiness; it can be the result of aging (Dauvilliers & Plazzi, 2010; Wittig & Zorick, 1983) or the presence of co-occurring sleep disorders such as periodic leg movements or sleep-disordered breathing (Guilleminault & Fromherz, 2005).

Cataplexy

Cataplexy, a sudden reversible bilateral loss of music tone triggered by strong emotions, occurs

Table 9.1 ■ *Diagnostic Criteria for Narcolepsy With and Without Cataplexy*

	Diagnostic Criteria	
Symptom or Finding	**With Cataplexy**	**Without Cataplexy**
Complaint of excessive daytime sleepiness	Occurs almost daily for at least 3 months	Occurs almost daily for at least 3 months
History of cataplexy	A definite history of cataplexy, triggered by strong emotion	No history of cataplexy or reports of doubtful or atypical cataplexy
Nocturnal polysomnogram, followed by MSLT	Recommended to confirm diagnosis; sleep onset latency during MSLT ≤ 8 minutes and two SOREMPs observed	Required to confirm diagnosis; sleep onset latency during MSLT ≤ 8 minutes and two SOREMPs observed
Hypersomnia	Is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, and medication use or substance use disorder	Is not better explained by another sleep disorder, medical or neurological disorder, mental disorder, and medication use or substance use disorder

Source: International Classification of Sleep Disorders: Diagnostic & Coding Manual (2nd ed.), by American Academy of Sleep Medicine, 2005, Westchester, IL: Author.

Abbreviations: MSLT, multiple sleep latency testing; SOREMPs, sleep onset REM periods.

in approximately 70% of patients with narcolepsy (Brooks & Mignot, 2002). Episodes of cataplexy can last anywhere from a few seconds to 30 minutes, but most episodes end within 2 minutes. Episodes of cataplexy are almost always precipitated by a sudden emotional stimulus, such as laughter, anger, or surprise (Guilleminault & Fromherz, 2005; Mattarozzi et al., 2008). Positive emotions such as joking, laughter, and elation are more likely to trigger attacks than negative emotions (e.g., anger) or undefined states (those lacking obvious valence, such as surprise) (Anic-Labat et al., 1999). The Web site of the Stanford University Center for Narcolepsy contains video clips of cataplexy in humans and animals.

The severity of cataplexy ranges from a mild sensation of muscle weakness in the neck and jaw to buckling of the knees and complete postural collapse. Speech can be impaired and respiration irregular, and there are short pauses in breathing (Guilleminault & Fromherz, 2005). Deep tendon reflexes are transiently abolished during an episode of cataplexy (Guilleminault, 1976; Overeem, Reijntjes, Huyser, Lammers, & van Dijk, 2004). Some patients experience cataplexy on an almost daily basis, while others have only one or two episodes during a lifetime; poor sleep and fatigue seem to exacerbate its occurrence (Dauvilliers, Amulf, & Mignot, 2007).

Sleep Paralysis

Approximately 40% of narcoleptic patients experience sleep paralysis, the inability to move voluntary (striated) muscles during the transition between sleep and waking (Brooks & Mignot, 2002). Patients are aware of their inability to move and able to recall the experience. Episodes last only a few minutes and can be accompanied by fear, hypnagogic hallucinations (defined below), or dream-like mentation. Sleep paralysis ends either spontaneously or immediately after the individual is touched or spoken to (Dauvilliers et al., 2007). Like cataplexy, the frequency of these episodes is highly individual. Sleep paralysis may occur daily, weekly, or once or twice in a lifetime.

Hypnagogic Hallucinations

Hypnagogic hallucinations are vivid perceptual experiences that usually occur at sleep onset and are present in 40%–80% of patients with narcolepsy-cataplexy (American Academy of Sleep Medicine, 2005). Some patients have similar hallucinations at awakening. These hallucinations are usually visual or auditory, but can also be tactile or involve the feeling of movement (levitating), and be accompanied by sleep paralysis (Brooks & Mignot, 2002; Guilleminault &

Fromherz, 2005). Most patients report that hallucinations are quite frightening and so vividly realistic that they might act upon them (Guilleminault & Fromherz, 2005).

Automatic Behavior

Many (20%–40%) patients report episodes of "automatic behavior" in which they continue an activity in a semiautomatic fashion without consciousness or memory (American Academy of Sleep Medicine, 2005; Lee-Chiong, 2008). However, automatic behavior is not considered to be a cardinal symptom of narcolepsy. Patients may continue their activities (e.g., writing a letter or continuing to work at a computer terminal), but the output will be nonsensical. Automatic behavior is believed to be caused by the intrusion of microsleep episodes (episodes of sleep last for a fraction of a second up to a few seconds) into wakefulness (Brooks & Mignot, 2002) and can be reversed by improving control of excessive daytime sleepiness.

DEVELOPMENT OF NARCOLEPSY

Narcolepsy usually begins between the ages of 10 and 25 years with the onset of excessive sleepiness and sleep attacks. Cataplexy and other rapid eye movement (REM)-related symptoms (e.g., sleep paralysis and hypnagogic hallucinations) can occur almost immediately or develop within 5–10 years or later (Bassetti & Aldrich, 1996; Sturzenegger & Bassetti, 2004). Although the severity of auxiliary symptoms may vary throughout the patient's lifetime, once excessive sleepiness develops, it is chronic and unrelenting.

Excessive daytime sleepiness is not exacerbated by the development of age-associated changes in nocturnal sleep (e.g., poorer sleep efficiency, increased awakenings, and decreases time spent in Stage 3/4 sleep) in people who have narcolepsy (Broughton & Broughton, 1994; Parkes, 1985; Stepanski, Lamphere, Badiz, Zorick, & Roth, 1984). In fact, a few patients have reported less-severe daytime sleepiness with increasing age (Billiard, Besset, & Cadihac, 1983; Broughton, 1990; Parkes, 1985), but whether this represents a partial remission or improved coping with the illness as people get older and accustomed to the symptom is unknown. Patients often learn to avoid situations that trigger cataplexy and therefore report a decrease in cataplectic events over time (American Academy of Sleep Medicine, 2005).

EPIDEMIOLOGY OF NARCOLEPSY

Although often considered a rare disease, narcolepsy is more common than Huntington's chorea, multiple sclerosis, and muscular dystrophy (National Commission on Sleep Disorders Research, 1993). Current estimates suggest that it affects 26–36 out of every 100,000 people or about 0.02%–0.5% of the U.S. population (Brooks & Mignot, 2002; Hublin et al., 1994; Longstreth et al., 2009; Silber, Krahn, & Slocumb, 2005). The prevalence of narcolepsy has been estimated to be as low as 0.002% in Israel and as high as 0.16%–0.18% in Japan (American Academy of Sleep Medicine, 2005; Brooks & Mignot, 2002).

Symptoms of narcolepsy often begin in adolescence or early adulthood. Onset of excessive daytime sleepiness occurring during middle age is more suggestive of obstructive sleep apnea than narcolepsy. (See Chapter 7, Sleep-Related Breathing Disorders.)

Males and females are affected equally, although some authorities report slightly more men than women experience narcolepsy (American Academy of Sleep Medicine, 2005). Although a recent epidemiologic study suggested that narcolepsy might be more common in both women and African Americans (Longstreth et al., 2009), these findings were based on a small sample of patients with limited ethnic diversity. Therefore, there is a need for studies involving more ethnically diverse populations before conclusions can be drawn about the prevalence of narcolepsy, particularly racial or ethnic groups in the United States.

CONSEQUENCES OF NARCOLEPSY

Narcolepsy often causes many disruptions in daily life, from poor performance in school to compromised work performance. Numerous studies over the past 30 years have documented

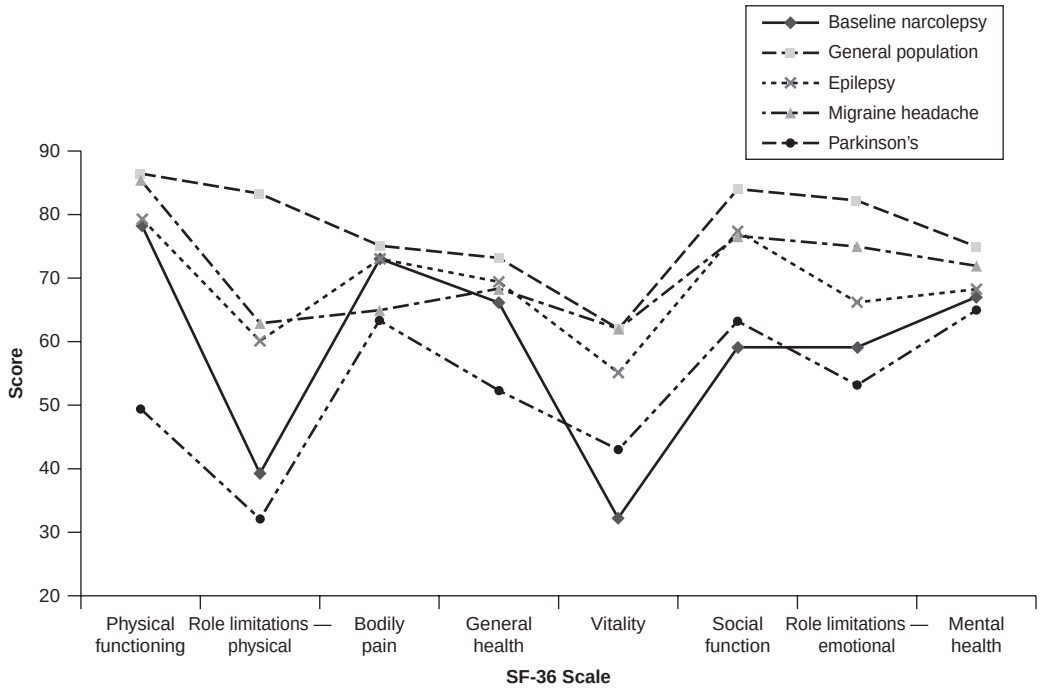

Figure 9.1 ■ Comparison of narcolepsy patients to the general and other patient populations. *Source:* "Health-Related Quality of Life Effects of Modafinil for the Treatment of Narcolepsy," by K. M. Beusterien, A. E. Rogers, J. A. Walsleben, H. A. Emsellem, J. A. Reblando, L. Wang, et al., 1999, *Sleep, 22*, pp. 757–767. Used with permission.

the adverse consequences of narcolepsy on safety, performance, and quality of life. For example, a series of studies comparing individuals with narcolepsy to matched controls (Broughton & Ghanem, 1976; Broughton et al., 1981, 1983) demonstrated that patients in Asia, Europe, and North America experienced similar problems: they had a higher risk for motor vehicle crashes and occupational, household, and smoking accidents due to excessive daytime sleepiness.

Most of the psychological and economic effects of narcolepsy can be directly attributed to sleep attacks and excessive daytime sleepiness (Broughton et al., 1983; Dodel et al., 2007; Rogers, 1984). Cataplexy and other symptoms of narcolepsy (particularly those occurring only at night) are usually not considered as disabling as the inability to stay awake when studying, working, driving, or interacting with friends and family members (Bayon, Leger, & Phillip, 2009).

Because narcolepsy usually develops during adolescence, academic performance is often compromised by difficulty in remaining awake during classes, examinations, and when trying to study (Broughton & Ghanem, 1976; Broughton et al., 1981, 1983; Rogers, 1984). During adulthood, excessive daytime sleepiness can compromise occupational performance, family and social activities, and the ability to drive safely (Broughton & Ghanem, 1976; Broughton et al., 1981, 1983; Rogers, 1984; Roy, 1976).

In addition to difficulties with functioning, patients with narcolepsy have profound difficulties with vitality, social functioning, and coping with physical and emotional problems (Beusterien et al., 1999). Their scores on several subscales of the Short Form Health Survey or SF-36 (see Figure 9.1) were as bad or worse than patients with Parkinson's disease or epilepsy, but these scores improved following treatment with modafinil (Provigil®) (Beusterien et al., 1999). Taken together, it is clear that the impact of narcolepsy on functioning and well-being is often profound.

PATHOPHYSIOLOGY OF NARCOLEPSY

There have been significant advances in understanding the pathophysiology of narcolepsy during the past 30 years, yet the exact cause remains elusive. Animal and human studies have documented alterations in neurotransmitter activity, a genetic predisposition for narcolepsy, and, most recently, a decrease in the production of hypocretin, an excitatory neuropeptide hormone (see below). Environmental factors may also play a role. However, none of these mechanisms alone completely explain all the symptoms of narcolepsy. In fact, most experts believe that the disorder is a result of a complex interplay between one or more genetic factors and environmental triggers (Brooks & Mignot, 2002).

Neurotransmitters

The dopamine system has been a focus of interest because of the beneficial effects of dopaminergic drugs on excessive daytime sleepiness. Information about the effects of these drugs has assisted in understanding the mechanisms underlying the symptoms of narcolepsy. For example, amphetamine-like stimulants are believed to reduce sleepiness via presynaptic stimulation of dopamine transmission, whereas methylphenidate blocks dopamine uptake (Nishino & Mignot, 2005; Wisor, Nishino, Sora, Uhl, & Mignot, 2001). Animal studies have suggested that there is decreased dopamine turnover and accumulation of dopamine in the presynaptic terminals of the amygdala (Miller, Faull, Bowersox, & Dement, 1990). Postmortem autoradiographic studies showed an increase in striatal dopamine D_2 receptor binding among narcoleptic patients, but this finding was not replicated in living narcoleptic patients (Rinne et al., 1995).

Numerous other neurotransmitters and peptides, such as norepinephrine, acetylcholine, serotonin, γ-aminobutyric acid (GABA), histamine, glutamate, adenosine, substance P, interleukin-1, and prostaglandins, play a role in regulating sleep and alertness and might also contribute to the pathophysiology of narcolepsy. The noradrenergic system (norepinephrine), which is involved in the regulation of REM sleep, is also implicated in the control of cataplexy. $Alpha_{1b}$ antagonists, such as prazosin (Minipress) and tamsulosin (Flomax), dramatically increase cataplexy, whereas stimulation of the $alpha_{1b}$ receptors decreases cataplexy (Mignot, Bowersox, Maddaluno, Dement, & Ciaranello, 1989; Miller et al., 1990). Yohimbine, an $alpha_2$ antagonist, also completely suppresses cataplexy (Fruhstorfer et al., 1989; Nishino et al., 1990). The effectiveness of monoamine reuptake blockers (tricyclic antidepressants) or serotonin reuptake blockers (e.g., fluoxetine) or dual serotonin and adrenergic reuptake blockers (e.g., venlafaxine) (Mignot & Nishino, 2005) supports their role in the control of cataplexy.

The efficacy of gamma-hydroxybutyrate (GHB) or sodium oxybate in treating multiple symptoms of narcolepsy, for example, cataplexy, disturbed nocturnal sleep, and excessive daytime sleepiness (Arnulf & Mignot, 2004; Mamelak, Black, Montplaisir, & Ristanovic, 2004; Mignot & Nishino, 2005), suggests that GABA, a neurotransmitter which enhances slow wave sleep, plays a role in the development of narcolepsy (Mamelak, 2009; Mamelak et al., 2004).

Hypocretin

Deficiency of hypocretin, an excitatory, neuropeptide hormone produced in the lateral and posterior hypothalamus, is thought to be one of the most important factors contributing to narcolepsy. Hypocretin plays a key role in wakefulness and is implicated in the regulation of appetite and energy expenditure. (See Chapter 1, Physiological and Behavioral Aspects of Sleep.)

The association of hypocretin with narcolepsy is supported by the finding of a mutation of the Hcrtr2 gene in narcoleptic dogs (Lin et al., 1999) and narcoleptic-like behavior in mice lacking the hypocretin gene (Chemelli et al., 1999). Levels of hypocretin in the spinal fluid of patients with narcolepsy–cataplexy are usually undetectable (Mignot et al., 2002; Nishino, Ripley, Overeem, Lammers, & Mignot, 2000; Nishino et al., 2001). Reduced, but detectable, levels of cerebrospinal fluid (CSF) hypocretin occur in patients who experience excessive daytime sleepiness without cataplexy

(Mignot et al., 2002; Nishino & Kanbayashi, 2005). Postmortem studies have also shown substantial losses of hypocretin neurons (up to 90%) in the hypothalamus of narcoleptic patients compared to age-matched controls (Peyron et al., 2000; Thannickal et al., 2000). Although researchers have proposed an autoimmune mechanism for the loss of hypocretin neurons (Silber, Black, Krahn, & Fredickson, 2007; Zeitzer, Nishino, & Mignot, 2006), this has not been documented.

Genetics

More than 85% of patients with narcolepsy are positive for the genes HLA DQB1*0602 and DQA1*0102 (Mignot et al., 2001). However, neither of these alleles alone (or together) is necessary or sufficient for the development of narcolepsy. Of the general population, 12%–25% have these alleles, yet only 0.02%–0.18% will develop narcolepsy (Mamelak, 2009). Although people with cataplexy are more likely to be positive for DQB1*0602 than patients without cataplexy (Mignot, Hayduk, Black, Grumet, & Guilleminault, 1997; Mignot et al., 2001), some patients with cataplexy are not positive for DQB1*0602. Most experts believe that DQB1*0602 and DQA1*0102 confer susceptibility to narcolepsy (Mamelak, 2009; Mignot et al., 2001); however, other environmental factors such as head trauma or sleep deprivation might be required to trigger the development of narcolepsy.

Environmental Influences

Support for a link between narcolepsy and exposure to unknown environmental factors was obtained from a large study conducted in clinical populations from Montpellier, France, Montreal, Canada, and Stanford University in California (Dauvilliers et al., 2003). Like patients in earlier, smaller studies (Dahmen & Tonn, 2003; Okun, Lin, Perlin, Hong, & Mignot, 2002), narcoleptic patients were more likely to be born in March and less likely to be born in September than those in the general population. This pattern was not affected by gender, center location, decade of birth, or even the presence or absence of HLA DQB1*0602 (Dauvilliers et al., 2003; Picchinoni, Mignot, & Harsh, 2004). Experts (Dahmen & Tonn, 2003; Dauvilliers et al., 2003; Picchinoni et al., 2004) hypothesize that the development of narcolepsy, like other central nervous system and autoimmune disorders that exhibit season-of-birth patterns (e.g., schizophrenia, Parkinson's disease, diabetes, and Crohn's disease) (Elborn, Wakefield, Zack, & Adams, 1994; Mattock, Marmot, & Stern, 1988; Mortensen et al., 1999; Ursic-Bratina et al., 2001), suggests potential exposure to seasonally varying environmental factors, such as infectious diseases. These factors might have critical etiologic importance during early development.

ASSESSMENT OF THE PATIENT WITH NARCOLEPSY

Assessment of patients for the presence of narcolepsy includes health history, physical examination, polysomnography (PSG), and multiple sleep latency testing (MSLT) (see Chapter 5, Conducting a Sleep Assessment) to evaluate the extent of excessive daytime sleepiness.

Health History

The major goal of a health history when narcolepsy is suspected is to rule out other causes of excessive daytime sleepiness, such as thyroid disease, substance use/abuse, sleep deprivation, and other sleep disorders, such as obstructive sleep apnea, and periodic limb movements. Therefore, history of these disorders should be carefully assessed. A complete medication review, including both prescription and over-the-counter medications, should be conducted to determine if any of them appears to be causing or contributing to the patient's complaint of excessive daytime sleepiness. The nurse should also ask about use of alcohol and drugs of abuse.

Patients with narcolepsy and other causes of excessive daytime sleepiness often consume large amounts of caffeine in order to stay awake and alert. Therefore, the nurse should elicit the amount of caffeine consumed, including the

approximate size of the cup, mug, pot, and/or number of ounces in a can of soda. The consumption of Red Bull and other highly caffeinated beverages, such as tea and chocolate, should also be noted. Evaluating the amount of caffeine consumed is one indicator of the degree to which patients need to compensate for their sleepiness.

It is also important to determine if there is a family history of sleep disorders. If the patient reports that other family members have narcolepsy or idiopathic hypersomnia, the nurse should determine if they received the diagnosis from a sleep specialist and if the diagnosis was made after a sleep study. Diagnoses made prior to the use of PSG may have been erroneous. A well-documented family history of narcolepsy should raise suspicions about the presence of this condition. However, while narcolepsy runs in families, multiplex families are quite rare. On the other hand, a negative family history does not rule out narcolepsy.

Sleep History

A detailed sleep history should be conducted (see Chapter 5, Conducting a Sleep Assessment). Assessment for sleep duration, and typical bed times and wake times on weekdays and weekends, reveals the amount of sleep obtained. If the patient obtains more sleep on the weekends, the examiner should ask if they are more alert throughout the day if they get additional sleep.

The nurse should obtain detailed information about the onset of symptoms, including when the symptoms occur (e.g., time of day and the circumstances). Patients with narcolepsy report that sleep attacks occur at any time during the day, whereas adults without narcolepsy or those who are sleep deprived report only afternoon drowsiness. Students often report difficulties in staying awake during boring lectures, but those with narcolepsy might report being unable able to stay awake during exams or during a clinical and student-teaching experiences.

Cataplexy is the hallmark symptom of narcolepsy and should not be confused with seizure activity, since the patient remains fully conscious throughout the episode. The nurse should query the patient about this syndrome by posing the following question: "Does anything unusual happen when you laugh, get angry, or experience some other strong emotion?" rather than by asking, "Do you fall down when laughing or experiencing some other strong emotion?" If the patient responds by describing an episode of muscle weakness or transient paralysis, the provider should determine the duration of the episode, the level of consciousness during the episode, and the frequency of such episodes. Patients should also be asked about the more subtle signs of cataplexy, such as dysarthria, jaw tremor, head or jaw dropping, dropping of objects, or buckling of the knees.

Some symptoms are not specific to narcolepsy but raise suspicions about its presence. Although hypnagogic hallucinations and sleep paralysis are frequently reported, these symptoms are not unique to narcolepsy, since approximately 3%–4% of the normal population experience them (Lee-Chiong, 2008). Sleep paralysis is also common in patients with sleep apnea, those who are sleep deprived, and normal adults (Brooks & Mignot, 2002; Lee-Chiong, 2008).

Physical Examination

After obtaining a health history, the nurse should conduct a brief physical exam. Unless the patient with suspected narcolepsy has other health problems, the physical exam should be unremarkable. If a patient has retrognathia, a long uvula and crowded airway (e.g., a Mallampati classification of three or four), and a scalloped tongue or a large neck (>17 inches in men and >16 inches in women), the nurse should suspect obstructive sleep apnea (see Chapter 7, Sleep-Related Breathing Disorders).

Diagnosis

Objective tests are useful in confirming the diagnosis of narcolepsy (American Academy of Sleep Medicine, 2005). When a clear history of cataplexy is not present, however, objective testing, including polysomnography (PSG) to rule out other sleep disorders and a multiple sleep latency test (MSLT), to evaluate the degree of

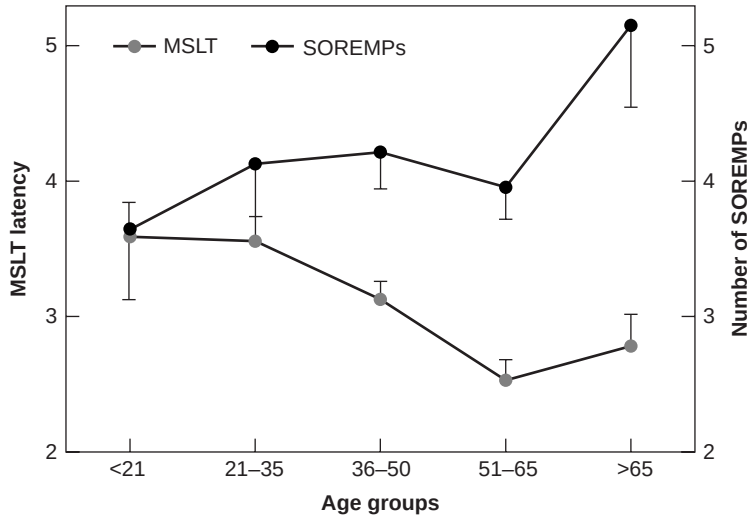

Figure 9.2 ■ Sleep onset latency and sleep onset REM periods (SOREMPs) on the multiple sleep latency (MSLT) testing in narcoleptic patients of various ages. *Source:* "Effect of Age on MSLT Results in Patients With Narcolepsycataplexy," by Y. Dauvilliers, A. Gosselin, J. Paquet, J. Touchon, M. Billiard, and J. Montplaisir, 2004, *Neurology, 62*(1), 46–50. Used with permission.

excessive daytime sleepiness and to document the presence of sleep onset REM periods (SOREMPs), is necessary. (See Chapter 5, Conducting a Sleep Assessment.) Although a definitive history of cataplexy is sufficient to make the diagnosis of narcolepsy, the American Academy of Sleep Medicine (2005) recommends that a PSG and MSLT be done to provide objective support for the diagnosis of narcolepsy.

Polysomnographic findings often include sleep fragmentation, an increase in Stage 1 sleep, short sleep latency (<10 minutes), and abnormally short REM latency (≤20 minutes) (American Academy of Sleep Medicine, 2005; Lee-Chiong, 2008). A nocturnal SOREMP (a REM period occurring in the first 20 minutes of sleep) is observed in approximately 25%–50% cases of narcolepsy with cataplexy and is a highly specific finding (American Academy of Sleep Medicine, 2005). MSLT findings that are considered diagnostic of narcolepsy include a mean sleep latency of less than 8 minutes and at least two SOREMPS (American Academy of Sleep Medicine, 2005). Sleep latencies are often much shorter than 8 minutes and can be as short as 3.1 minutes (Standards of Practice Committee of the American Academy of Sleep Medicine, 2005).

Interpretation of MSLT results in children depends in part on the age of the child. Normal prepubertal children between the ages of 6 and 11 years may be hyperalert during an MSLT, whereas sleep latencies less than 8 minutes are pathological in postpubertal children (Guilleminault & Fromherz, 2005). Sleep latencies recorded in prepubertal children with narcolepsy can be quite short, for example, less than 5 minutes, and SOREMPs might be recorded during the majority of MSLT naps (Guilleminault & Pelayo, 1998).

Adults can demonstrate progressive increases in sleep latency: Adults over 65 years take an average of 5.16 ± 3.42 minutes to fall asleep compared with 4.14 ± 2.97 minutes for patients aged 21–35 years of age (Dauvilliers et al., 2004). The number of SOREMPs also decreases with advancing age. Approximately, 15% of patients with narcolepsy and cataplexy, especially those older than 36 years (25%), have normal or borderline MSLT results (e.g., sleep latency ≤8 minutes or only one SOREMP) (Standards of Practice Committee of the American Academy of Sleep Medicine, 2005) (see Figure 9.2).

Although some authorities suggest that cataplexy is necessary for the diagnosis of narcolepsy

(Honda & Juji, 1988; Moscovitch, Partinen, Patterson, et al., 1991), the current diagnostic criteria allow for the diagnosis of narcolepsy to be made in the absence of cataplexy if certain conditions are met (see Table 9.1) (American Academy of Sleep Medicine, 2005).

Nocturnal PSG and MSLT testing should be performed only when (1) the patient has been free of drugs that influence sleep (e.g., opiates, narcotics, REM suppressants) for at least 15 days or for at least five times the duration of the half-life of the drug and the longest acting metabolites; (2) the patient's sleep schedule has been standardized for at least 7 days and documented using sleep logs or actigraphy; and (3) the patient gets at least 6 hours of sleep on the night preceding the MSLT (Standards of Practice Committee of the American Academy of Sleep Medicine, 2005). MSLT should always follow a nocturnal polysomnogram conducted in the sleep laboratory.

Although some laboratories obtain a urine drug screen at the time of the sleep study to rule out recent use of opiates, sedatives, hypnotics, and stimulants (Lee-Chiong, 2008), this is not required. Obstructive sleep apnea, periodic limb movements, and other sleep disorders can cause excessive daytime sleepiness and affect MSLT results. Therefore, these conditions must be treated before diagnosing narcolepsy.

Subjective measures of sleepiness, such as the Epworth Sleepiness Scale (Johns, 1991) and 24-hour diaries (Rogers, Caruso, & Aldrich, 1993), may be useful in assessing sleepiness, but are not sufficient for diagnosing narcolepsy. (See Chapter 5, Conducting a Sleep Assessment.) Currently, there are no objective tests to determine the presence or absence of cataplexy, nor is there a standardized measure of cataplexy severity.

TREATMENT AND FOLLOW-UP CARE

Treatment goals include the following: (1) eliminating excessive daytime sleepiness; (2) facilitating the fullest possible return to normal school, work, and social functioning; and (3) controlling other REM-related symptoms, if present and troublesome (Guilleminault & Anagnos, 2000; Morgenthaler et al., 2007). Initial treatment focuses on minimizing excessive daytime sleepiness because it is usually the most disabling symptom. All patients should be offered stimulant medications for the treatment of excessive daytime sleepiness, and nurses should provide patient education and support in using these medications, managing their side effects, and monitoring long-term adherence and efficacy. Evaluation of functional status and patient education regarding safety-related issues, for example, driving and work-related injuries, is an important role of nursing.

Medications Used to Treat Narcolepsy Symptoms

Medications used to treat patients with narcolepsy are focused on treating the symptoms. These often include stimulants, hypnotics, selective serotonin reuptake inhibitors (SSRIs), tricyclic antidepressants, and sodium oxybate or GHB.

Excessive Daytime Sleepiness

Table 9.2 lists the most common medications used to treat excessive daytime sleepiness in narcoleptic patients and their side effects. Modafinil is currently the drug of choice for initiating therapy in adults and adolescents who are newly diagnosed with narcolepsy. Unlike traditional stimulants, modafinil has little or no effect on dopaminergic activity (U.S. Modafinil in Narcolepsy Multicenter Study Group, 1998), but its exact mechanism is unknown.

Patients should begin drug therapy at the lowest dose of the safest agent (modafinil) and be titrated to the optimal dosage within 10 days. Because not all patients will respond equally to the same drug or dosage, a period of experimentation with various drugs, dosages, and dosing schedules might be required to determine the optimal therapy. Some patients might also require a second dose of a longer acting medication (e.g., modafinil or methylphenidate-SR), or supplementation with a short-acting stimulant in the afternoon (Littner et al., 2001). Although it has a longer half-life than traditional stimulants (U.S. Modafinil in Narcolepsy Multicenter Study Group, 1998), some studies suggest that

Table 9.2 ■ *Medications Used to Treat Excessive Daytime Sleepiness*

Medication	Usual Daily Dose (mg)	Common Side Effects	Other Information/Precautions
Armodafinil	150–250	Headache, nausea, and insomnia	If a rash develops, medication should be stopped immediately. Use with caution in patients with a history of cardiac problems, hypertension, or psychosis
Dextroamphetamine	5–60	Tremor, palpitations, headache, irritability, sweating, insomnia, anorexia, hypertension, and erectile dysfunction	Comes in both short-acting and long-acting formulations. The short-acting form can be quite useful when patients want to tailor their treatment to their daily activities
Methamphetamine	5–60	Same as for dextroamphetamine	More potent and more effective than dextroamphetamine
Methylphenidate	10–60	Nervousness, insomnia, dizziness, hypertension and hypotension, headache	Comes in both short-acting and long-acting formulations. The short-acting form can be quite useful when patients want to tailor their treatment to their daily activities
Modafinil	100–400	Headache and nausea	Use with caution in patients with a history of cardiac problems

twice-daily dosing, with the second dose taken at lunchtime, might be more effective than the single dose originally recommended (Billiard et al., 1994; Schwartz, Feldman, & Bogan, 2005). Some patients prefer armodafinil (Nuvigil), a longer acting isomer of modafinil (Harsh et al., 2006).

Methylphenidate is usually considered the safest treatment for children aged 6–12 years (Lee-Chiong, 2008). Since modafinil and methylphenidate are quite popular among college students, older adolescents with narcolepsy should be encouraged to keep their medications hidden or in a locked container to prevent their diversion. College students who have narcolepsy should also be warned that some student health clinics have policies that forbid the prescription of stimulant medications for any reason. Therefore, they may need to obtain their prescriptions from a private physician or through a physician in their hometown.

In the past, the stimulants amphetamines and methylphenidate were the mainstays of narcolepsy treatment (Morgenthaler et al., 2007). However, they are associated with high risk for elevated blood pressure, appetite suppression, and tolerance. Some clinicians prefer to switch patients taking traditional stimulants to modafinil, but patients may find that modafinil does not adequately control their excessive daytime sleepiness (Besset, Chetrit, Carlander, & Billiard, 1996; Guilleminault, Aftab, Karadeniz, Phillip, & Leger, 2000). Some patients might also notice the reappearance of cataplexy after switching from amphetamines to modafinil (Guilleminault & Anagnos, 2000), since amphetamines sometimes reduce the severity of REM-related symptoms.

Cateplexy

Tricyclic antidepressants such as clomipramine (Anafranil), desimpramine (Norpramin), imipramine (Tofranil), and protriptyline (Vivactil), were the earliest effective treatments for cataplexy (Houghton, Scammell, & Thorpy, 2004). These REM-suppressing drugs also reduce the severity of other REM-related symptoms (e.g., sleep paralysis hypnagogic hallucinations). Monoamine oxidase inhibitors (MOIs), such as selegiline (Eldepryl), are effective in controlling cataplexy, but their use is limited by serious

Table 9.3 ■ *Medications Used to Treat Cataplexy*

Medication	Usual Dose (mg)	Common Side Effects	Other Information/Precautions
Tricyclic antidepressants Clonipramine Desipramine Imipramine Protriptyline	10–150 10–100 5–60	Dry mouth, urinary retention, constipation, nausea, anorexia, diarrhea, weight gain, tiredness, decreased libido, erectile dysfunction, and delayed ejaculation	Use with caution in patients with coexisting cardiac disease. Contraindicated in patients with seizure disorders and glaucoma. Abrupt discontinuation can lead to severe rebound cataplexy or status cataplecticus lasting several days
Selective serotonin reuptake inhibitors (SSRIs) Fluoxetine Fluvoxamine Paroxetine Sertraline	20–60	Headache, nausea, weight gain, dry mouth, and delayed ejaculation	Use with caution in patients with diabetes mellitus, hepatic or renal impairments, or a seizure disorder
Serotonin-norepinehrine reuptake inhibitor (SNRI) Venlafaxine	37.5–300	Anorexia, dry mouth, nervousness, and elevated blood pressure	
Central nervous system (CNS) depressant Sodium oxybate	3–9 mg (divided dose, twice nightly)	Dizziness, headache, nausea, confusion, vomiting, urinary incontinence, weight loss, and occasional residual sedation	Medication has a short half-life and is only available as a liquid. Patients must be instructed to set their alarm clocks to awaken them 4 hours later to ensure that they take their second dose of medication. Because the combined use of alcohol and sodium oxybate may result in significant potentiation of CNS-depressant effects of sodium oxybate, patients should be warned not to consume any alcohol while taking the medication. Although a schedule III drug, sodium oxybate is available from one pharmacy in the United States.

side effects and potentially dangerous drug interactions (Houghton et al., 2004). SSRIs and serotonin–norepinephrine reuptake inhibitors have fewer side effects than tricyclic antidepressants and MOIs (Houghton et al., 2004; Morgenthaler et al., 2007).

SSRIs commonly used to reduce cataplexy include fluoxetine (Prozac), fluvoxamine (Luvox), paroxetine (Paxil), and sertraline (Zoloft). Venlafaxine (Effexor), a serotonin-norepinephrine reuptake inhibitor, is also effective in the control of cataplexy (Morgenthaler et al., 2007). More detailed information about these drugs, dosages, and side effects is included in Table 9.3. Cataplexy usually improves within 1–2 days of starting treatment, particularly with MOIs, SSRIs, or venlafaxine (Houghton et al., 2004; Linnoila, Simpson, & Skinner, 1980).

Sodium oxybate or GHB (Xyrem) is the only medication approved by the FDA for the treatment of cataplexy in the United States (Houghton, et al., 2004). GHB, a gamma-aminobutyric acid (GABA) precursor, increases sleep continuity and decreases the frequency of cataplexy (Scharf, Brown, Woods,

Brown, & Hirschowitz, 1980; Scrima, Hartman, Johnson, & Hiller, 1989; U.S. Xyrem Multicenter Study Group, 2002, 2003). Sodium oxybate also helps to reduce excessive daytime sleepiness. Because of its rapid onset action and short half-life, it must be taken immediately before bedtime and again 2.5–4 hours later. Due to the potential for abuse by body builders and its association with date rape, sodium oxybate is available only through a restricted distribution system (Xyrem Success Program). All prescriptions must be sent to a central mail order pharmacy that registers patients and determines whether they have read and understood the patient education booklet before they receive their first shipment. Additional patient education and tips for storing sodium oxybate are available at http://www.xyrem.com/healthcare-professionals/getting-started/directing-patients.php.

Sleep Fragmentation

Patients who are distressed by their fragmented nocturnal sleep may benefit from hypnotic drugs, such as benzodiazepines or trazodone. Short-acting hypnotics can also improve tolerance of continuous positive airway pressure (CPAP) in narcoleptic patients who suffer from sleep-related breathing disorders (Brooks & Mignot, 2002). Consolidating the patient's nighttime sleep, however, has not been shown to affect the severity of excessive daytime sleepiness in people with narcolepsy.

Medication Treatment Plan

Table 9.4 shows an example of an initial treatment plan for an adult patient as well as suggestions for modifying treatment if symptoms persist. It is especially important to institute

Table 9.4 ■ *Suggested Treatment Approaches for Adults With Narcolepsy*

	Specific Suggestions
General measures	Avoid shifts in sleep schedule Avoid heavy meals and alcohol intake Regular timing of nocturnal sleep
Initial management of sleepiness[a]	The effects of stimulant medications vary widely among patients; therefore, the dosage and timing of medications should be individualized to optimize performance. Additional doses, as needed, may be suggested for periods of anticipated sleepiness. Modafinil 200–400 mg taken when first awakening in morning (starting with 200 mg q a.m. may prevent the development of headaches, which is sometimes associated with starting this medication); Methylphenidate 10 mg when waking up, 5 mg before lunch, and 5 mg approximately 3:00 PM, or 20 mg methylphenidate SR in the morning
If persistent difficulty in managing sleepiness	Modafinil 200 mg in morning with an additional 100 or 200 mg at noon Methylphenidate SR 20 mg in the morning, with 5 mg at noon and 5 mg at 4:00 PM, or add a second dose of methylphenidate SR 20 mg at noon
If no response	Dextroamphetamine sulfate (Dexedrine Spansule) 15 mg upon awakening, 5 mg at noon and 5 mg at 3:30 or 4:00 PM Dextroamphetamine sulfate (Dexedrine Spansule) SR 15 mg in the morning and 15 mg at noon
Management of cataplexy*	Clomipramine 75–125 mg at bedtime or Imipramine 75–125 mg at bedtime or Fluoxetine 20–60 mg in the morning or Venlafaxine 150–300 mg Viloxazine 150–200 mg or Sodium oxybate 3–9 mg in divided doses at bedtime

[a] Only modafinil and sodium oxybate are FDA approved for the treatment of narcolepsy.

Source: Principles and Practice of Sleep Medicine (3rd ed., pp. 676–686), by M. H. Kryger, T. Roth, and W. C. Dement (Eds.), 2000, Philadelphia: WB Saunders Company.

close follow-up care and flexibility in dosages and medications during the early months of treatment. Although most patients (40%–50%) achieve satisfactory levels of alertness with low-to-moderate doses of stimulant medications (Lee-Chiong, 2008), some require higher doses or a combination of short- and long-acting stimulants (Morgenthaler et al., 2007).

Behavioral Strategies for Managing Narcolepsy Symptoms

Exercise routines, dietary changes, and scheduled naps are often appealing to both patients and health care providers; however, there is little evidence that behavioral approaches are effective. Scheduled nap periods might be a useful adjuvant therapy, but they are insufficient for controlling excessive daytime sleepiness associated with narcolepsy (Littner et al., 2001; Morgenthaler et al., 2007).

Patient Education

Patients and their families should be educated about the clinical features of narcolepsy, its therapy, and the necessity for lifelong treatment. In the case of students and workers, information should be shared with schools and employers only if the patient makes the request; or if the employee seeks to request work accommodation, for example, through the Americans with Disabilities Act. If a patient requests work accommodation through the Americans with Disabilities Act, they may need documentation of their diagnosis in order to take frequent short naps, or to avoid rotating and/or extended duty periods. If medications with short half-lives are prescribed (e.g., methylphenidate and dextroamphetamine), patients must arrange to take their mid-day doses of medication at work or school.

Patients should also be advised to avoid sleep deprivation and shift work (especially rotating or irregular shifts) that may exacerbate excessive daytime sleepiness. They should avoid driving or performing other potentially dangerous activities when drowsy.

Many patients find that support groups are helpful. They can be directed to support groups sponsored by the Narcolepsy Network (http://www.narcolepsynetwork.org) or the National Sleep Foundation (http://www.sleepfoundation.org). Both sites, and the Web site sponsored by Stanford University's Center for Narcolepsy (http://med.stanford.edu/school/Psychiatry/narcolepsy/), also provide accurate information about the disorder (see Table 9.5).

Follow-Up Care

Once treatment is optimized, patients should be seen every 6–12 months for follow-up care (Morgenthaler et al., 2007) and be evaluated for the development of side effects, tolerance to medications (especially important if on high doses of amphetamines), efficacy of therapy, and compliance with the prescribed treatment regime.

All patients, regardless of age, education, or socioeconomic status, particularly those who continue to report difficulties with excessive daytime sleepiness, should be questioned about adherence, since many patients reduce their dosage of medication (Rogers, Aldrich, Berrios, & Rosenberg, 1997), take their medications at different times than prescribed, or take more or less drug than prescribed (Firestone, 1982; Sleator, 1985). In general, approximately one-third of all patients are estimated to be satisfactorily adherent, another third will be partially compliant, and the remaining third will not comply at all with treatment

Table 9.5 ■ *Web Sites for Support Groups and Patient Information on Narcolepsy*

Narcolepsy Network (http://www.narcolepsynetwork.org)
National Sleep Foundation (http://www.sleepfoundation.org)
Stanford University's Center for Narcolepsy (http://med.stanford.edu/school/Psychiatry/narcolepsy/)
http://med.stanford.edu/school/Psychiatry/narcolepsy/movies/Child.avi
http://med.stanford.edu/school/Psychiatry/narcolepsy/movies/dogs.avi

CASE STUDY 9.1

S.A. is a 21-year-old college student who presents to the Student Health Clinic with a complaint of being unable to stay awake in class. He usually goes to bed around midnight and wakes up around 7:30 or 8:00 am, feeling refreshed. He reports struggling to stay awake during his 10:00 am class, as well as in afternoon classes, and recently fell asleep during a midterm exam. Once an honor student, he is now struggling to maintain a 2.5 GPA.

When questioned, he admits that he began having trouble staying awake in high school classes when he was about 16 years old, and that he falls asleep several times each day, no matter how much sleep he obtains at night. Further questioning reveals that he has been experiencing symptoms suggestive of cataplexy for the past 2 months; for example, he feels weak whenever he laughs at one of his roommate's jokes. He denies any hypnagogic hallucinations, sleep paralysis, disrupted nocturnal sleep, or snoring.

His health history is negative and there is no family history of sleep disorders. He does not smoke, use recreational substances, and consumes approximately 1–2 beers/week. Physical exam is noncontributory and oral exam is normal.

His differential diagnosis includes narcolepsy and idiopathic hypersomnia. Two weeks after his initial visit, he underwent a nocturnal PSG and a MSLT. There was one SOREMP during the nocturnal polysomnogram and two SOREMPs during the MSLT. Sleep latency on the MSLT was 3.5 minutes. Modafinil 200 mg q a.m. was prescribed and when that was not effective, his dose was increased to 400 mg q a.m. Since his symptoms of cataplexy were fairly mild and were not distressing to him, no medications were prescribed for cataplexy.

During his first follow-up visit after the sleep studies, he was given information about narcolepsy, and safety issues related to driving discussed. He was cautioned not to drive unless he was alert and if he became sleepy to stop driving and take a nap.

(Urquhart, 1993). Underdosing rather than overdosing is particularly pervasive (Kauffman, Smith-Wright, Reese, Simpson, & Jones, 1981; Urquhart, 1993). Patients also fail to fill their original prescription or refill their prescriptions in a timely fashion, take "drug holidays," or stop taking their medications after a few months, without consulting their health care providers.

If patients adhere to their prescribed medications and fail to respond to stimulant medications and/or develop excessive daytime sleepiness after a period of successful treatment, they should be assessed for other sleep disorders that could contribute to excessive daytime sleepiness (e.g., periodic limb movements of sleep and sleep related breathing disorders). Approximately, 30% of patients with narcolepsy develop sleep apnea with increasing age and/or weight (Lee-Chiong, 2008). Anticataplectic medications can trigger the development of periodic limb movements of sleep and REM sleep disorder (Guilleminault & Anagnos, 2000; Lee-Chiong, 2008). In addition to the assessment for excessive daytime sleepiness, it is important to evaluate patients with narcolepsy for the extent to which they are able to perform their normal work, school, social, and leisure-time activities. Assessment of coping and mental health is also important.

CONCLUSIONS

Narcolepsy is a serious disorder with significant adverse effects on quality of life. With diagnosis and appropriate treatment, most patients can lead productive lives. Nurses and advanced practice nurses, as members of an interdisciplinary team, play important roles in assessment, diagnosis, treatment, and follow-up care.

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10 Circadian Rhythm Disorders

Glenna Dowling and Judy Mastick

Circadian rhythm disorders (CRD) result from a complex interplay of developmental and pathophysiological circadian processes, comorbid psychiatric and medical conditions, and environmental factors. They have profound implications not only for sleep–wake patterns, but also for school and work place performance, safety, cognition, and morbidity and mortality. For the most part, effective treatments are available and involve behavioral and environmental changes, as well as adjunctive pharmacological therapy. This chapter provides an overview of the circadian timing system and disorders that result from disruption of this system.

Nurses play important roles in raising awareness and educating patients about these disorders and their treatment. At the same time, it is important for nurses to understand these disorders in order to optimally assess and intervene with patients, and to mitigate some of the effects of shift work (e.g., shift work sleep disorder) on themselves and other nurses. Throughout this chapter, we will refer to the diagnoses as described in the International Classification of Sleep Disorders (American Academy of Sleep Medicine, 2005).

THE CIRCADIAN RHYTHM OF SLEEP AND WAKE

The master time keeper or circadian pacemaker resides in the suprachiasmatic nucleus (SCN) in the hypothalamus. This timing system has an intrinsic approximately 24-hour oscillation that is synchronized with the 24-hour light–dark cycle. Entrainment is the synchronization of circadian pacemaker output with the environmental light–dark, and social and activity cycles. The primary time cues for entrainment of circadian rhythms are dusk and dawn. Other non-photic time cues, such as regular activity and meals, also affect circadian timing, but the effect is weak, compared to light exposure (Sack, 2009).

Desynchronization, lack of entrainment, is when circadian rhythms are out of sync with the 24-hour day, and occur at socially unacceptable or non-conventional times. Desynchrony can have a variety of etiologies including jet travel across time zones and shift work (Turek, 2005). Purposeful, therapeutic light exposure can be used to reset the body clock. Morning light resets the body clock to an earlier time (i.e., phase advance), and evening light exposure resets the body clock to a later time (i.e., phase delay). Examples of the use of light in this fashion include the treatment of seasonal depression and the correction of sleep disturbances in circumstances such as jet lag and shift work.

The most prominent circadian rhythm in humans is the sleep–wake cycle. The phases and timing of normal sleep are regulated by two primary processes. The homeostatic drive to sleep is controlled by the duration of wake such that the longer one is awake, the stronger the drive to sleep becomes. Similarly, deprivation of a specific sleep stage results in a greater drive to recover that stage of sleep. For example, a very short night's sleep results in proportionately more loss of rapid eye movement (REM) compared to non-REM sleep. Thus, the next sleep

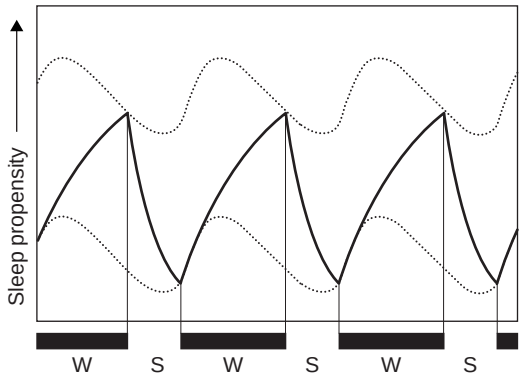

Figure 10.1 ■ *The two-process model of sleep regulation.* Sleep propensity grows during wakeful periods (W) and abates during sleeping periods (S). Homeostatic process (process S solid line) is limited to a certain range of values determined by a clock-like circadian process (process C dotted lines) that varies with time of day. *Source:* "Jet Lag and Other Sleep Disorders Relevant to the Traveler," by R. R. Auger and T. I. Morgenthaler, 2009, *Travel Medicine and Infectious Disease, 7*(2), pp. 60–68. Reprinted with permission from Elsevier Publishers.

Table 10.1 ■ *Summary of Findings from the 2008 Sleep in America Poll: Sleep, Performance and the Workplace*

- Shift workers were defined as persons who start work after 6 PM but before 6 AM.
- Seven percent of the sample (n=1,000) met the criteria for being shift workers.
- The data compared non-shift workers to shift workers.
- Results showed that shift workers were most commonly male blue collar workers who:
  1. Spent less than 6 hours in bed and slept less than 6 hours on workdays
  2. Disclosed that a doctor had informed them that they had sleep apnea
  3. Admitted to driving drowsy at least once a month in the past year
  4. Consistently worked on average more than 49 hours per week

Source: Adapted from the National Sleep Foundation, 2008

episode will contain an amount of REM that is greater than usual in order to "make up" for the loss and the resultant deficit. The circadian drive sends "alerting" and "sleep" signals based on time of day regardless of duration of sleep or wake (see Figure 10.1). (See Chapter 1, Physiological Aspects of Normal Sleep.)

SHIFT WORK SLEEP DISORDER

While people have a natural propensity to be active during the day, personal preference for morning (lark) or evening (owl) activity varies between individuals (Sack et al., 2007). Larks may be drawn to work that starts early in the day and owls may be drawn to evening or night shift work. People forced to work in jobs that are counter to their preferences may experience some of the disorders discussed in the following sections.

Characteristics

Symptoms of shift work sleep disorder (SWSD) include excessive sleepiness during work hours and insomnia during the sleep period. This may occur in individuals who work night, evening, early morning shifts, or rotate shifts, but night and early morning shifts (starting before 06:00 AM) are the most problematic for adaptation. Total 24-hour sleep time can be reduced by 1–4 hours, sleep quality is perceived as poor, and patients report not feeling refreshed upon awakening. Although SWSD is usually self-limiting and resolves when work periods move into normal daytime hours, some people can continue to experience sleep disturbances after their actual shift work has ended (see Table 10.1). (See Chapter 22, Sleep Promotion in Occupational Health Settings.)

Epidemiology

Approximately 20% of the workforce is employed in jobs that require shift work, that is, work outside the hours of 0700–1900. The prevalence of clinically significant sleep disturbances and daytime sleepiness in SWSD is 1%–5% in the general population and approximately 30% in shift workers (Drake, Roehrs, Richardson, Walsh, & Roth, 2004). Less than 3% of permanent night shift workers ever completely reentrain to sleep during the day (Folkard, 2008). There is no known gender vulnerability

(Presser, 1987), but the ability to cope with shift work decreases as people enter the fifth decade of life. Thus, people who have coped successfully with shift work in their younger years may begin to experience symptoms as they age (Monk, 2005). Treatments for SWSD also tend to be more effective in younger adults than those in middle age or older (Campbell, 1995). Night shift workers in isolated situations such as oil rigs where there are no societal/family pressures to assume a "normal" day–night schedule on days off (Barnes, Deacon, Forbes, & Arendt, 1998; Midwinter & Arendt, 1991; Ross, Arendt, Horne, & Haston, 1995) are able to adapt successfully to working at night. However, these people often encounter difficulty when they return home to the typical 24-hour day–night sleep pattern.

Related Factors

Workers whose shifts begin in the early morning hours may have difficulty falling asleep at night and awakening in the morning. Shift workers who work in the early morning hours or into the evening may experience daytime sleepiness and take naps.

Consequences

Fatigue-related impairments in physical and cognitive performance result from the cumulative sleep deficit and decreased circadian alerting output at night. These impairments result in work performance deficits and safety risks. Many major accidents (e.g., Exxon Valdez oil spill, NASA Space Shuttle Challenger explosion) have been linked to fatigue-related human error (National Transportation Safety Board, 1990; Report of the Presidential Commission on the Space Shuttle Challenger Accident, 1986). Night shift workers are also at risk for accidents during their commute home and exposure to daylight provides input to the circadian system that is in sync with the 24-hour light–dark cycle but out of sync with the shift workers internal circadian time. Daytime sleepiness and irritability are common symptoms associated with night shift workers' cumulative sleep deficit; and the drive to sleep during the daytime hours often conflicts with demands for social and family activities. The desire to change back to regular sleep times during days off and vacation may lead to chronic sleep disturbances in some people.

Other complications of shift work include depression (Monk, 2005), gastrointestinal and cardiovascular disorders, increased risk for metabolic disturbances, cancer and drug and alcohol dependency (Conlon, Lightfoot, & Kreiger, 2007; Lavie & Lavie, 2007; Morikawa et al., 2007; Pietroiusti et al., 2006).

Pathophysiology

Symptoms of SWSD are due to misalignment of the circadian rhythm with the work schedule. Workers' internal preference for morning or evening activity influences their ability to adjust to shift work. Morning types report more difficulty getting enough sleep during the day after working a night shift. When they do sleep, the sleep may be fragmented and of poor quality. The balance between sleep–wake regulation and clock resetting varies greatly between individuals. Those with a very strong circadian clock and need for stable hours may be at risk for poor adjustment to shift work. Comorbid conditions and other sleep disorders also contribute to difficulty adjusting to shift work.

Assessment Methods

Polysomnography (PSG) may be useful during the "shifted" sleep period to determine etiology and rule out comorbid conditions, such as sleep apnea and periodic limb movement disorder. The Multiple Sleep Latency Test (MSLT) provides 4–5 nap opportunities throughout the usual wake period time during which sleep onset latency is measured. A mean sleep onset latency of less than five minutes is considered pathological sleepiness. The Epworth Sleepiness Scale, administered during the work shift, is also useful to determine the extent of subjective daytime sleepiness. Scores of 10 or greater are considered pathological. Actigraph (movement) data collected in conjunction with a sleep

diary over a one-week period can provide useful data from which estimates of total sleep time (TST), sleep efficiency, and sleep fragmentation can be inferred. (See Chapter 5, Conducting a Sleep Assessment.)

Diagnosis

A history of shift work accompanied by complaints of insomnia, poor sleep quality, and daytime sleepiness is generally adequate to diagnose SWSD. Symptoms present for at least one month with sleep diary or actigraphy confirmation of sleep–wake misalignment and the absence of an alternative explanation for symptoms (e.g., substance abuse, medication, or comorbid medical conditions) are diagnostic.

Treatment and Follow-Up Care

The goal of treatment of SWSD is to lessen the impact of shift work on the body clock, thereby improving night work tolerance. Behavioral treatment strategies include good sleep hygiene, minimizing exposure to natural light by wearing sunglasses when commuting home in the morning, and optimizing the sleep environment by using black out curtains to reduce light in the bedroom and wearing ear plugs to reduce noise. Two hour naps taken shortly before the night shift begins increase vigilance, reaction time, and alertness. Twenty-minute naps taken during the night shift also improve alertness (Garbarino et al., 2004; Morgenthaler et al., 2007; Schweitzer, Randazzo, Stone, Erman, & Walsh, 2006).

Alterations in work schedules may also be beneficial. Permanent night workers can benefit from a partial reentrainment schedule designed to delay the circadian "sleepiest" time out of the night shift work time and into the early portion of the daytime sleep period during work days and near the end of late nighttime sleep periods on days off. This sort of compromised phase position when maintained throughout work and days off facilitate alertness during night shifts and afternoon and evening alertness during days off (Smith, Fogg, & Eastman, 2009).

Pharmacologic intervention can also be helpful. Melatonin in doses of 3 mg or less taken prior to initiation of daytime sleep increases sleep time and quality (Bjorvatn et al., 2007; Iskra-Golec et al., 2001; Morgenthaler et al., 2007; Sharkey & Eastman, 2002; Yoon & Song, 2002). Hypnotic medications can also promote daytime sleep. However, the balance of risk and benefit need to be carefully weighed on an individual basis. Stimulants including caffeine (Morgenthaler et al., 2007) and modafinil 200 mg taken 1 hour before the night shift (Rosekind, 2005) and the longer acting armodafinil 150 mg taken 30 minutes before the shift improve wakefulness and attention during the shift (without disrupting daytime sleep) (Czeisler et al., 2005; Czeisler, Walsh, Wesnes, Arora, & Roth, 2009). (See Table 10.2 for caffeine content of various foods and beverages.)

Education about the personal health and safety risks associated with sleep and circadian disruption should include the basics of sleep need, the effects of sleep loss and cumulative sleep debt, circadian impacts on alertness, and vulnerability to problems with performance.

Table 10.2 ■ *Caffeine Content of Popular Ingestibles*

Product	Serving Size	Caffeine Content (mg)
Coffee	8 oz	110
Starbucks	Grande 16 oz	330
Starbucks Americano	16 oz	225
Starbucks Mocha	16 oz	175
Espresso	2 shots	150
Tea, black or green	16 oz	60–100
Coca-Cola	12 oz	35
Root Beer	12 oz	25
Red Bull	250 ml	80
Jolt	23.5 oz	280
Arizona Green Tea Energy	16 oz	170
Monster	16 oz	150
Chocolate milk	8 oz	5
Jolt gum	1 piece	60
Excedrin, extra strength	2 tablets	130
No Doz, reg strength	1 tablet	100

Source: Compiled from company information.

Assessment of signs and symptoms and an individualized treatment plan is recommended (Rosekind, 2005). (See Chapter 5, Conducting a Sleep Assessment and Chapter 22, Sleep Promotion in Occupational Health Settings.)

Employers can decrease the negative impacts of shift work by decreasing the number of shift changes, changing forward rather that backward (days to evenings to nights), giving workers regular rest periods, providing bright light exposure starting early in the night shift and ending approximately 2 hours before the end of shift, maintaining a cool ambient temperature, and offering the option of exercise breaks using bright light to imitate sunlight (American Academy of Sleep Medicine, n.d.). (See Chapter 22, Sleep in Occupational Health Settings.)

SWSD has particular relevance to nurses because they are frequently required to do shift work and often fall victim to the disorder itself (Admi, Tzischinsky, Epstein, Herer, & Lavie, 2008). Awareness and self-care regarding the potential negative effects of shift work is critical for nurses' well-being. Addressing performance deficits in nurses and other shift workers may also improve patient safety and reduce risks of accidents.

A preventative approach to SWSD includes routine screening, assessment for SWSD, and related risks in all persons who do shift work. Patient and family teaching regarding the effects of shift work and beneficial strategies is an important nursing role. For nurses who do not have experience with SWSD, consultation with a clinician who is experienced in these methods may be helpful.

JET LAG DISORDER

Characteristics

Jet lag is a temporary disorder that results from travel across two or more time zones. Upon arrival at the destination, the body's internal clock is no longer synchronized with the 24-hour light–dark cycle. Patients report fatigue, impaired daytime function, daytime sleepiness and decreased alertness, and disturbed nighttime sleep. Jet lag can also precipitate gastric complaints including loss of appetite, indigestion, and feeling bloated after eating because the ability to metabolize and store food are compromised. Jet lag in the morning is strongly associated with poor sleep during the night before and is associated with decreased ability to concentrate, lack of motivation, and increased irritability during the daytime. In the evening, jet lag is associated with not feeling tired and ready for the next sleep episode (Waterhouse, Nevill, Edwards, Godfrey, & Reilly, 2003; Waterhouse et al., 2005). Symptoms tend to be more pronounced with increasing number of time zones traveled, and eastward, compared to westward travel.

"Phase tolerance," the ability to sleep at an abnormal circadian phase, varies across individuals and age groups. In a simulation experiment, middle-aged subjects had greater impairments in alertness and more fragmented sleep than their younger counterparts (Auger & Morgenthaler, 2009; Moline et al., 1992; Monk, Buysse, Carrier, & Kupfer, 2000). Older adults tend to have more prolonged and more pronounced symptoms. Although there is little empirical evidence, larks (morning types) tend to do better with eastward travel, because they can adapt more readily to advancing (moving ahead) their bedtimes and rise times because of their predisposition to early awakening. Owls (evening types) tend to do better with westward travel (Reilly, Waterhouse, & Edwards, 2009).

Related Factors

People often experience increased stress and activity in preparation for travel. Preparatory activities can extend into the regular sleep period and causing a sleep debt before departure. This can contribute to the negative effects of jet lag.

Consequences

The consequences of jet lag are nocturnal or early awakenings, delayed sleep onset, fatigue, sleep loss, lethargy, and impaired cognitive and physical performance (Coste & Lagarde, 2009).

Pathophysiology

Symptoms of jet leg are due to the desynchrony of the endogenous circadian time with the local time zone and related sleep loss. Transmeridian travel entails sitting for extended periods of time in a typically small and uncomfortable seat. Low aircraft cabin pressure and poor air quality, consumption of alcoholic or caffeinated beverages in flight, and the physical and psychological stress associated with disruption of normal routines all contribute to the severity of insomnia and impaired function and alertness in the destination time zone (Nicholson, 2009). Approximately, 1 day per time zone crossed is needed for the body clock to adjust to the new destination time.

Assessment Methods

PSG or other objective methods of assessment are not typically needed to diagnose the presence and severity of jet lag. If PSG or actigraphy are obtained, results will indicate an abnormal sleep–wake pattern and a mismatch of the pattern to the local time zone. Self-report is the primary method used to evaluate jet lag. Reentrainment to the new time zone occurs at about 1 hour (one time zone) per day. Thus, travel across four time zones requires about four days to reentrain.

Diagnosis

The diagnosis of jet lag is made based on the following criteria: (1) Jet travel across at least two time zones followed in 1–2 days by complaints of insomnia and excessive daytime sleepiness and impaired function, fatigue, and gastrointestinal irregularity; (2) symptoms cannot be explained by another disorder or medication or substance use. Persistent symptoms may predispose the individual to chronic psychophysiological insomnia.

Treatment and Follow-Up Care

Jet lag treatment options include planning the sleep schedule, timed light exposure, hypnotics, stimulants, and melatonin administration (Morgenthaler et al., 2007). Behavioral management strategies to reduce the amount of psychological and physical stress and sleep debt before the flight are useful. For trips of short duration, jet lag can be minimized or prevented by keeping bedtime and wake-up time on the home time schedule at the travel destination. For a longer stay, adjusting sleep and wake-up times before leaving on the trip to minimize the difference between the home and destination location times may facilitate entrainment to the new time zone (see Table 10.3) (Sack, 2009).

Modifying the sleep schedule before departure includes waking up earlier and going to bed earlier when travel is eastward. When travel is in westward direction, the individual should go to bed later, wake up later and sleep on the flight during the future nighttime in the destination time zone. Short acting non-benzodiazapine hypnotic agents (see Chapter 6, Insomnia) may be useful to promote in-flight sleep.

After the Transmeridian flight, sleep promoting agents may be useful for sustaining nighttime sleep without adversely affecting performance. Creating an optimal environment at the destination by minimizing noise with ear plugs and light with eye shades can facilitate adequate sleep. Appropriately timed bright light exposure and avoidance of light exposure can facilitate entrainment to the new time zone. For example, morning exposure facilitates resynchronizing the biological clock after eastward travel, whereas evening exposure facilitates phase delay after westward travel (Coste & Lagarde, 2009). People with a normal sleep routine who are not extreme larks or owls and need to phase advance (start early in the day, as in eastward travel) should obtain an hour of light exposure starting at 0600–0800. Conversely, an hour of light exposure starting at 1400–1500 (Paul et al., 2009) will facilitate phase delay (as is needed with westward travel).

Melatonin administration, when dosed and timed correctly, can work in synergy with light exposure to expedite the resynchronizing of the circadian clock to the destination light–dark cycle. Melatonin is not FDA approved as

Table 10.3 ■ *Recommendations for Minimizing Jet Lag and Travel Fatigue*

Strategy	Traveling Westward	Traveling Eastward
Before travel		
Begin to reset the body clock	If possible, shift the timing of sleep to 1–2 hour later for a few days before the trip; seek exposure to bright light in the evening	If possible, shift the timing of sleep to 1–2 hour earlier for a few days before the trip; seek exposure to bright light in the morning
Try to get an adequate amount of sleep	Do not leave packing and other travel preparations to the last minute; if possible, schedule a flight at a time that will not cut short the sleep time before travel	
In flight		
Try to optimize comfort	Travel in business class or first class, if financially feasible	
Drink judiciously	Drink a lot of water to remain hydrated; minimize consumption of caffeine if you expect to sleep; do not drink alcohol if you intend to take a sleeping pill during the flight	
Use a sleeping medication, if necessary	Consider a short-acting sleeping pill (e.g., zaleplon [Sonata, King Pharmaceuticals] at a dose of 5–10 mg) to promote sleep during the flight; a longer-acting sleeping pill (e.g., zolpidem [Ambien, Sanofi-Aventis] or eszopiclone [Lunesta, Sepracor]) could result in grogginess on arrival; a sleeping pill should not be taken if there is a risk of deep vein thrombosis, and it should not be combined with alcohol	
Take measures to avoid deep vein thrombosis	Because sitting immobile for a long time can increase the risk of a blood clot, change positions frequently and walk around when possible; if you are prone to blood clots, consult a physician, since a more specific preventive measure may be needed (e.g., using antiembolism stockings)	
On arrival		
Be prepared for changes in sleep pattern	Expect to have trouble staying asleep until you have become adapted to local time	Expect to have trouble falling asleep until you have become adapted to local time
Take appropriate naps	If you are sleep-deprived because of an overnight flight, take a nap after arrival at your destination; on subsequent days, take daytime naps if you are sleepy, but keep them as short as possible (20–30 minutes) in order not to undermine nighttime sleep	

(Continued)

Table 10.3 ■ *Recommendations for Minimizing Jet Lag and Travel Fatigue (Continued)*

Strategy	Traveling Westward	Traveling Eastward
Use sleeping medication, if necessary	Consider taking a sleeping medication (e.g., zolpidem [Ambien] or eszopiclone [Lunesta]) at bedtime for a few nights until you have adjusted to local time	
Take melatonin	To promote shifting of the body clock to a later time, take 0.5 mg (a short-acting dose) during the second half of the night until you have become adapted to local time	To promote shifting of the body clock to an earlier time, take 0.5–3 mg at local bedtime nightly until you have become adapted to local time
Seek appropriately timed exposure to light	Seek exposure to bright light in the evening	Seek exposure to bright light in the morning
After crossing more than eight time zones, avoid light at times when it may inhibit adaptation[a]	For the first 2 days after arrival, avoid bright light for 2–3 hours before dusk; starting on the third day, seek exposure to bright light in the evening	For the first 2 days after arrival, avoid bright light for the first 2–3 hour after dawn; starting on the third day, seek exposure to bright light in the morning
Drink caffeinated drinks judiciously	Caffeine will increase daytime alertness, but avoid it after midday since it may undermine nighttime sleep	

[a]This strategy is based on the theory that after a person crosses eight or more time zones, the circadian system may initially misinterpret "dawn" as "dusk" (or vice versa).

a medication in the Unites States but is sold as a dietary supplement. (See Chapter 6, Insomnia and Chapter 14 Complementary and Alternative Medicine and Sleep.)

Short naps of 20-minute duration improve cognitive performance and do not cause sleep inertia (grogginess and impaired motor performance upon awakening). After a longer nap, sleep inertia can be mitigated by having a cup of coffee prior to the nap (Coste & Lagarde, 2009). Caffeine, an adenosine antagonist, is the most popular compound used by travelers to enhance wakefulness in the new time zone. A dose equivalent to two cups of coffee produces a temporary improvement in daytime alertness after a night without sleep (Walsh, Muehlbach, & Schweitzer, 1995; Wright, Badia, Myers, & Plenzler, 1997). The effects of caffeine are long acting and can adversely affect nighttime sleep if used to close to bedtime. The best advice is to drink coffee at the destination breakfast time and avoid caffeine in the evening (Reilly et al., 2009). Dopamimetic compounds (e.g., modafanil) can also be useful for the short-term treatment of jet lag (Nicholson, 2009) to enhance alertness during the desired wakeful period. (See Chapter 9, Narcolepsy.)

ADVANCED SLEEP PHASE DISORDER

Characteristics

Advanced sleep phase disorder (ASPS) is an involuntary advance in the timing of the major sleep period in relation to the desired (conventional) sleep time and wake time such that habitual sleep and wake times are several hours earlier than "normal." People with this disorder experience partial sleep loss, late afternoon sleepiness, and an inability to stay awake until the desired bedtime, coupled with an inability to remain asleep until the desired and socially acceptable time for awakening.

Epidemiology and Related Factors

While the exact prevalence of ASPS is unknown, it is estimated to be 1%–7% in middle-aged and older adults (Bloom et al., 2009). The incidence increases with advancing age, and both sexes are affected at the same rate (Ando, Kripke, & Ancoli-Israel, 2002).

When patients are allowed to choose their preferred schedule for bed and rise times, TST and sleep quality are normal for their age. (See Chapter 2, Developmental Aspects of Normal Sleep.) These patients are entrained to the 24-hour clock, but sleep and wake times occur earlier than the social norms. The difficulty arises when patients, family, or society (e.g., employers) perceive this mismatch to be problematic. If daily demands require a later bedtime, there may be a deficit in TST. People with ASPD may use medications or alcohol to combat the early morning awakening or evening sleepiness. These substances may worsen the phase disorder.

Pathophysiology

The pathogenesis of ASPD is thought to involve both behavioral and genetic factors. The sleep homeostatic process and the circadian process are not in synchrony.

Assessment Methods

Actigraph monitoring and sleep diaries show early bed and early rise times, typically between 0200 and 0500 with a stable rhythm. It may be helpful to assess preference for morning or evening activity, since people with ASPD are almost always larks, whose preference is for early wake times and early bedtimes. Self-report is usually adequate. PSG is generally not standard practice for assessment of ASPD, but if conducted during the patient's preferred sleep period, sleep architecture is usually normal for age. PSG may be useful in screening for primary sleep disorders including sleep apnea and periodic limb movements, because they may be associated with ASPD and increase with aging (Reid & Zee, 2005).

Diagnosis

The differential diagnosis for early awakening includes ruling out other causes of primary or secondary insomnia such as depression. (See Chapter 12, Sleep and Psychiatric Disorders.) ASPS is confirmed when the patient's symptoms cannot be attributed to other psychiatric or medical conditions or medication use.

Treatment and Follow-Up Care

Treatment may or may not be initiated depending on the patient's perception of the degree of disruption (see Figure 10.2). Behavioral approaches and bright light therapy can postpone evening sleepiness and delay bedtime and sleep onset. Light exposure between 1500 and 2100 and planned sleep schedules can be useful (Morgenthaler et al., 2007; (Morgenthaler et al., 2008). However, patients often have difficulty complying with bright light treatment and easily revert back to the phase advanced timing of their sleep. (See Chapter 12, Sleep and Psychiatric Disorders.) As with other CRD, recognition of the signs and symptoms of the particular disorder allow for a fuller assessment of the symptoms, accurate diagnosis and appropriate intervention. Nurses who work with older adults, who are particularly vulnerable to ASPS, should incorporate assessment for this condition and patient teaching into their plans of care.

DELAYED SLEEP PHASE DISORDER

Characteristics

Delayed sleep phase disorder (DSPD) is characterized by routine bed and rise times that are more than 2 hours later than conventional, socially acceptable times. Falling asleep at "normal" bedtimes is difficult but, once asleep, sleep is normal. Patients with DSPD typically have reduced exposure to light in the morning and exposure to light in the late evening.

Epidemiology

DSPD is the most commonly reported primary CRD with a prevalence of 7%–16% (Yamadera, Takahashi, & Okawa, 1996). It is most common in adolescents and young adults with a mean age of onset at 20 years. (See Chapter 2, Developmental Aspects of Sleep.) An estimated 10% of sleep clinic patients are diagnosed with

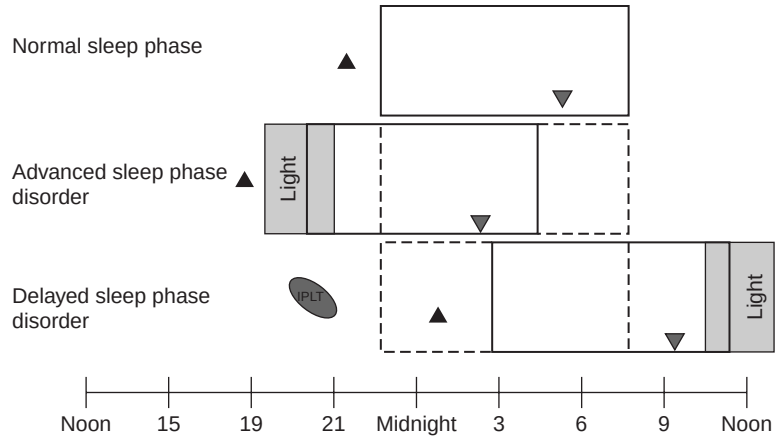

Figure 10.2 ■ *Schematic representation of treatment strategies for advanced sleep phase disorder (ASPD) and delayed sleep phase disorder (DSPD).* In this example, the primary sleep period is indicated by the solid lines and the desired sleep phase following treatment is indicated by the dashed lines. The estimated core body temperature (CBT) rhythm minimum and the dim-light melatonin onset, both common markers of circadian phase, are indicated by triangles. The CBT occurs 2–3 hours prior to habitual wake time (gray triangles) and dim-light melatonin onset occurs 2 hours prior to habitual sleep onset (black triangles). When light is administered prior to the CBT, it results in a phase delay of the circadian clock (moves it later), and when light is given after the CBT, it results in a phase advance of the circadian clock (moves it earlier). To phase delay the sleep period of an individual with ASPD, bright light should be administered during the first half of the evening; in this example, light is given from 1900 to 2100 (indicated by the gray box). To phase advance the sleep period of an individual with DSPD, bright light should be administered in the morning after the estimated CBT; in this example, light is administered between 1100 and 1300 (indicated by the gray box). The current examples try to minimize the amount of sleep lost to maximize the phase shifting effects of the light. Melatonin (MLT) given in the evening is also used to phase advance circadian rhythms in DSPD. Melatonin administered relative to the dim-light melatonin onset has shown the most consistent phase advances in DSPD, and administration 5 hours prior to dim-light melatonin onset or 7 hours prior to habitual sleep onset gives consistent phase advances. In this example, melatonin would be administered at 2000. *Source*: "Circadian Rhythm Disorders," by K. J. Reid and P. C. Zee, 2009, *Seminars in Neurology, 29*(4), pp. 393–405. Reprinted with permission from the publishers.

DSPD. Inability to adjust to travel across time zones, shift work, atypical social, or work schedules may precipitate DSPD.

Related Factors

DSPD is associated with depression, schizophrenia, and avoidant personality disorders. Its onset may follow an environmental, psychological, or medical stressor (Alvarez, Dahlitz, Vignau, & Parkes, 1992; Regestein & Monk, 1995).

Consequences

DSPD is often associated with mood and personality disorders and impairment in social, occupational, and other areas of functioning (Alvarez et al., 1992; Regestein & Monk, 1995). Awakening in the early morning is difficult, and patients tend to have difficulty with clear thought processes at that time. Sleepiness is greatest in the morning and lessens as the circadian drive for wakefulness peaks in the late afternoon. Morning performance on the job or at school is impaired and daytime irritability is common. Patients with DSPD also have decreased ability to compensate for sleep debt (Uchiyama et al., 1999; Uchiyama et al., 2000). DSPD is a common problem in adolescents who are usually expected to awaken early in the morning for school. (See Chapter 2, Developmental Aspects of Normal Sleep.)

The inability to go to sleep at normal socially acceptable times may lead to nighttime use of sedatives or alcohol and morning use of caffeine and other stimulants to treat sleepiness, as well as marital and financial problems from poor job per-

formance (Alvarez et al., 1992; Thorpy, Korman, Spielman, & Glovinsky, 1988). DSPD can be a chronic condition, usually lasting at least 3 months and often many years (Reid & Zee, 2005).

Pathophysiology

The exact cause of DSPD is not known, but researchers have proposed that behavioral and physiological factors may contribute. An abnormal interaction between the circadian time keeping system and the homeostatic sleep–wake regulation process may be responsible for the disorder and many physiologic markers of circadian phase are delayed even when early sleep and wake times are enforced (Carskadon, Labyak, Acebo, & Seifer, 1999; Czeisler, Richardson, Zimmerman, Moore-Ede, & Weitzman, 1981). During puberty, a biologically based circadian delay of about 2 hours occurs in conjunction with an increased need for sleep (Carskadon et al., 1980; Carskadon, Vieira, & Acebo, 1993). (See Chapter 2, Developmental Aspects of Normal Sleep.) The normal nighttime suppression of melatonin by bright light may also be altered. Positive family history may be present, and there may be an autosomal dominant trait. Polymorphisms of circadian clock gene *hPer3*, human leukocyte antigen (HLA), and *Clock* are associated with DSPD.

Assessment Methods

Most patients with DSPD are "evening" types or owls. Actigraphic recordings and/or sleep logs reveal delayed sleep onset and offset relative to usually socially acceptable times. Sleep onset is typically between 0100 and 0600, with wake-up times in the late morning or afternoon. Weekday demands often force individuals into an earlier than desired wake time, but the preferred delayed bed and wake times reemerge on weekends (Reid & Zee, 2005). PSG is normal if conducted during the preferred sleep interval. However, the timing of other circadian inputs (e.g., dim-light melatonin onset (DLMO) and temperature nadir [trough]) will be later than normal (phase delayed).

Diagnosis

Patients usually report that the major sleep period is later than conventionally accepted (between 0100 and 0600) and is accompanied by a delayed wake time. Enforced early rise times lead to a severely shortened sleep period with a resulting sleep deficit, sleep inertia, with grogginess or confusion upon awakening. When self-selected times for bed and rise are allowed, the sleep period is normal and set to the 24-hour clock. Adolescents are particularly at risk for this disorder.

Treatment and Follow-Up Care

DPSD is self-perpetuating because late wake-up times delay exposure to light in the morning and prevent advancement of the circadian clock. Treatment is designed to reset the circadian clock to coincide with desired bed and rise times and the light–dark cycle; however, patients have a natural propensity to delay, and there is a high incidence of relapse despite initial successful therapy. This suggests that physiological as well as behavioral factors may be responsible for DPSD. Treatment should be customized based on the severity of symptoms, schedules and obligations, ability to comply with treatment, and social pressures (Regestein & Monk, 1995).

Therapy with bright light in the morning 0700–0900, coupled with light reduction in the evening, can be helpful (Regestein & Pavlova, 1995; Rosenthal et al., 1990). Compliance with morning bright light is difficult because patients often have difficulty awakening in order to initiate the light intervention. Low-dose melatonin (e.g., 3 mg) taken at 2200 advances the sleep period. The combination of morning bright light and evening administration of melatonin may be particularly helpful (American Academy of Sleep Medicine (AASM) practice parameters) (Morgenthaler et al., 2007). Use of caffeine or other stimulants in the afternoon can contribute to delayed sleep onset and should be avoided. Patients often use hypnotics, sedatives, or alcohol to initiate earlier sleep, and these should be avoided as the risk of dependence is high.

Table 10.4 ■ *Circadian Rhythm Disorders: Web-Based Resources*

Web Sites	Description
http://www.sleepeducation.com	Self-help ideas, quiz, sleep diary, sleep problems throughout the life span, sleep hygiene
http://www.sleepfoundation.org	National Sleep Foundation
Sleep facts and topics including jet lag and shift work in easy-to-understand language that include etiology and treatment options	

Nurses play an important role in patient education to raise awareness among patients, especially adolescents and their families, of DPSD and its negative consequences and assisting them with behavioral strategies to reduce its negative effects. Education regarding the proper use of sedatives and stimulants is essential. As discussed in Chapters 2 and 17, nurses also have important roles in schools and adolescent health care settings to educate families and advocate for school schedules that are consistent with adolescents' circadian rhythms. Web-based resources are provided in Table 10.4.

IRREGULAR SLEEP–WAKE RHYTHM

Characteristics

This condition is characterized by a lack of a clearly defined sleep–wake cycle. Sleep and wake periods are disorganized and occur throughout the 24-hour period. Symptoms include excessive sleepiness periods during the day and insomnia during the night. TST may be normal for a 24-hour period, but sleep is obtained during periods of napping rather than in one consolidated nighttime sleep episode. Napping at all times of the day is common. Circadian rhythm amplitude is often lower than normal.

Epidemiology, Related Factors, and Consequences

The prevalence of irregular sleep–wake rhythm in the general population is unknown, but it is thought to be rare, and the onset can occur at any age (Bittencourt, Santos-Silva, De Mello, Andersen, & Tufik, 2010). Neurological dysfunction, dementia, brain injury, and damage to central processes can lead to changes in circadian rhythms. Lack of exposure to external environmental synchronizers such as light, activity, social schedules, mealtimes, and poor sleep hygiene contribute to a disorganized, irregular sleep–wake rhythm (Reid & Zee, 2005). Little is known about the course and complications of low amplitude, disorganized rest-activity rhythms.

Pathophysiology

A combination of circadian regulation dysfunction (anatomic or functional abnormalities of the circadian clock) and reduction of environmental cues most likely contribute to irregular sleep–wake rhythms.

Assessment Methods

Self-report is helpful in eliciting the presence of irregular sleep–wake rhythms. Sleep logs and actigraphy elicit periods of irregular sleep (at least three) and wake bouts throughout the 24-hour period with low overall amplitude. Actigraphy should be collected for two weeks to adequately characterize the irregular rhythm. Although not indicated for clinical purposes, PSG and 24-hour core body temperature monitoring indicate a loss of circadian rhythmicity. Core body temperature is regulated by the hypothalamus and provides an excellent index of circadian rhythm function.

Diagnosis

Voluntary maintenance of irregular sleep schedules, poor sleep hygiene, and medical or injury-related etiologies must be distinguished from irregular rhythm disorder (Reid & Zee, 2009). Accurate diagnosis is difficult because

the criteria for this condition overlap with the diagnostic category "disorder due to medical condition" (Bjorvatn & Pallesen, 2008). Careful analysis of actigraphy or diary will demonstrate multiple periods of sleep and wake, with three or more sleep episodes across the 24-hour cycle and evidence of no major sleep period.

Treatment and Follow-Up Care

The treatment goal for patients with irregular sleep–wake rhythms is to increase the amplitude of the circadian rhythm and its alignment with the external environment so that there is one major sleep period and one wake time during a 24-hour period. The 2007 AASM practice parameters (Morgenthaler et al., 2007) recommend a combination of increased social and physical activity to stimulate the brain and provide time cues. Also, exposure to timed bright light during the day to reduce napping, increase alertness, and provide input to the biological clock. Good sleep hygiene and a bedtime ritual can be helpful in creating a more organized sleep schedule. Cognitive–behavioral therapy may also have beneficial effects. (See Chapter 6, Insomnia.) Melatonin is not recommended as the means to improve sleep consolidation in older adults (Morgenthaler et al., 2007).

As with interventions related to any of the disorders noted in this chapter, it is important to recognize that sleep-related changes take time. Incorporating a reasonable time frame for behavioral change is a critical key to clinical success with the treatment plan.

CIRCADIAN RHYTHM SLEEP DISORDER DUE TO MEDICAL CONDITION

Many medical and neurological conditions, such as dementia, movement disorders, blindness, and hepatic encephalopathy can result in circadian rhythm sleep disorders (American Academy of Sleep Medicine, 2005). Abnormalities in patients with dementia include altered phase, decreased amplitude, or lack of rhythm. These alterations may lead to behaviors such as sundowning and nocturnal wandering. (See Chapter 21, Sleep in Adult Long-Term Care and Chapter 11, Sleep in Medical Disorders.) Patients with Parkinson's disease may have diurnal fluctuations of their symptoms related to shifts in circadian functions. Patients with cirrhosis complain of insomnia, excessive sleepiness, and delayed sleep phase. Blind persons can have free-running rhythms that are slightly longer than the 24-hour day and lead to progressively later bed and rise times so that over time, bed and rise times become out of sync with societal norms. Because of the wide variety in conditions that can lead to circadian rhythm abnormalities, specific data on prevalence are not available.

Conditions that result in decreased exposure to light and lack of other inputs to the circadian system (e.g., meals, activity, and social interaction) can increase the severity of the consequences of medical conditions on sleep. This is especially true in acute care hospitals and long-term care facilities where there may be low light levels or light levels that are inconsistent with normal day–night patterns and frequent patient care activities that do not allow adequate time for sleep (in the case of the acute care hospital) or limited opportunities for social interaction (e.g., long-term care setting). (See Chapter 21.) Poor sleep quality and resultant decrements in physical performance and/or cognitive function may, in turn, exacerbate the underlying condition.

Assessment

PSG may be useful to diagnose alterations in sleep architecture. Sleep diaries and actigraphy for at least seven days are useful to assess amplitude and altered or fragmented sleep–wake pattern, or phase.

Treatment and Follow-Up Care

Nursing care for patients who have CRD secondary to medical disorders focuses on preventive or therapeutic measures to maximize circadian rhythmicity, for example, including bright daytime light exposure, maintaining a regular schedule (e.g., regular meal times, rise time, and bedtime) with as much physical activity as

tolerated. These strategies are useful for patients living in the community, as well as patients in acute care hospitals and long-term care settings. (See Chapter 20, Sleep in Adult Acute and Critical Care Settings and Chapter 21, Sleep in Adult Long-Term Care.) Education of patients and families, especially caregivers of patients with disabling chronic conditions, is a critical nursing role. Nocturnal awakenings, agitation, and wandering often result from dementia and its associated circadian abnormalities and lead to increased caregiver burden, as well increased risk for admission to long-term care facilities. Therefore, improving the circadian rhythm of sleep and wake may ameliorate or delay the occurrence of these negative consequences. Additional sleep hygiene measure are also useful and include avoiding alcoholic beverages within several hours of bedtime, avoiding caffeine and nicotine in the afternoon and evening hours, and avoiding the regular use of sedatives/hypnotics, with only occasional use after 3–4 nights of poor sleep.

CONCLUSIONS

There are a variety of physiological conditions that result from alterations in circadian rhythms. The etiology ranges from internal and genetic to psychological to psychosocial and environmental. Treatments are likewise variable and include pharmacologic, (e.g., hypnotics and melatonin), environmental (e.g., controlling exposure to light and dark), behavioral (e.g., napping), occupational (e.g., managing shift rotation schedules), and public health interventions (e.g., advocacy for later school start times for adolescents and improved workplace scheduling).

Nurses' roles in working with people who experience these disorders are multifaceted and focus on anticipatory guidance, assessment, patient and family teaching, and assisting patients to adopt behavioral strategies and environmental changes that support the regularization of circadian rhythms. It is important for nurses to have an integrative perspective that recognizes the important contributions of circadian physiology and environmental factors to sleep disorders and incorporate this perspective in assessment and intervention with the wide variety of specific evidence-based strategies available. Clinical supervision is a critical element in developing skill and expertise with the clinical management of these disorders and should be used to broaden the nurse's comfort and competence in a new area of practice for the novice (see Table 10.5).

Table 10.5 ■ *Implications for Nursing Practice*

- Jet lag, shift work, and other less common circadian disorders put people at risk for sleep-related problems
- A thorough sleep history should be conducted including bed and rise times, napping, insomnia, and sleepiness
- Assess impact of sleep-related problems on physiological, cognitive, behavioral, emotional, and social parameters. Refer or intervene as appropriate
- Educate regarding risks of accident and injury to self and others when active during times of sleepiness
- Educate about risks of substance abuse when using sedatives to help with sleep and/or stimulants to combat sleepiness
- Teach sleep hygiene principles
- Educate regarding risks of long-term CRD, including diabetes, obesity, cardiovascular disease, and cancer

In addition, advanced practice nurses

- Educate and initiate light and/or melatonin therapies
- Assess for mood disorders and initiate/refer for cognitive–behavioral therapy

CASE STUDY 10.1

Three nurses working off-hours shifts include a rotating day/night shift nurse (4 weekdays 7:00 AM–3:30 PM; 2 weeknights, 11:30 PM–7:30 AM) who is the mother of two children aged 6 and 3 years; a permanent 12-hour night shift worker who helps in her husband's store on her days off; and a permanent 8-hour night shift worker. What would be different?

CASE STUDY 10.2

Clinical Nurse Specialist Jake, who lives in New York State, will be presenting a poster on the second day of a 3-day conference in Lyon, France (6 hours ahead of New York). Jake has only 5 days off, and his goal is to be as awake and alert as possible for his poster presentation. Jake's normal rise time is 5:30 AM and bedtime is 10:00 PM. Two days prior to departure, he "advances" himself by an hour, rising at 4:30 AM and at going to bed at 9:00 PM. The following day, he awakens at 3:30 AM. His flight leaves at 6:00 PM. As soon as possible when on the plane, Jake takes a zaleplon 10 mg and sleeps for as long as possible. Upon arrival in France, he stays awake and walks outdoors to maximize his bright light exposure. At bedtime, he uses a zolpidem 10 mg to help himself get to sleep and stay asleep in the new time zone. On his return trip, he stays awake as best as he can. Upon his return to New York, Jake does not schedule late evening engagements; he knows he will be tired. He stays up as late as possible.

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11 Sleep in Medical Disorders

Kathy P. Parker

Prior to the twentieth century, many believed that sleep was a simple and passive phenomenon, similar in many aspects to death. Nonetheless, sleep was also often valued for its health promoting effects (Thorpy, 1991). As early as the fourth century BC, Hippocrates (480–360) wrote:

> "With regard to sleep—as is usual in health, the patient should wake during the day and sleep during the night. If this rule be anywise altered it is so far worse ..... but the worst of all is to get no sleep either day or night; for it follows from this symptom that the insomnolency is connected with sorrow and pain, or that he is about to become delirious" (Adams, 1868).

As the modern field of sleep science developed, numerous studies have indeed demonstrated that sleep deprivation and/or disruption are associated with a number of medical disorders and can adversely affect important clinical outcomes such as quality of life, functional health status, and mortality rates (Ferrie et al., 2007; Johansson, Arestedt, et al., 2010; Marin, Carrizo, Vicente, & Agusti, 2005; Parish, 2009; Redeker, Muench, et al., 2010; Young, 2009).

In general, individuals can be at risk for developing sleep problems due to a number of demographic, psychological, and behavioral factors. However, medical comorbidities add additional factors that often appear to be primarily related to the condition and/or its treatment (Parish, 2009). In fact, a number of different medical disorders, ranging from a common cold to cancer, can alter an individual's sleep patterns (see Table 11.1).

Individuals with medical disorders may experience a variety of sleep-related symptoms, yet be unaware or unwilling to discuss these issues with a health care provider. However, with proper diagnosis and treatment, many of these problems can be addressed with improvement in clinical outcomes. This chapter provides a brief overview of information related to some of the most prevalent medical disorders associated with sleep problems including the prevalence, consequences, pathophysiology, and treatment. In addition, a summary of information related to assessment, diagnosis, and follow-up care is also provided.

SLEEP IN CARDIOVASCULAR DISEASE

As of 2006, more than 81 million Americans were estimated to have cardiovascular disease, the major cause of death in the United States (AHA, 2010). Recent epidemiologic studies, such as the Sleep Heart Health Study and the Wisconsin Sleep Cohort Study, have linked sleep apnea with over 50% of those with these disorders (Gottlieb et al., 2010; Newman et al., 2001; Redline et al., 2010; Shahar et al., 2001; Young, 2009). However, although many studies confirm a likely causal relationship between obstructive sleep apnea (OSA) and hypertension and a strong relationship between central sleep apnea (CSA) and heart failure, similar evidence for other cardiovascular disorders such as atrial fibrillation, stroke, and myocardial infarction (MI) remains limited (AHA, 2010) (Gottlieb et al., 2010).

Table 11.1 ■ *Sleep Problems in Patients With Common Medical Conditions*

Pulmonary disease
COPD
Poor sleep efficiency
Increased sleep latency
Decreased total sleep time
Increased wake after sleep onset
Decreased rapid-eye-movement (REM) sleep
Hypoxemia
Overlap syndrome with OSA
Asthma
Nocturnal asthma
Disrupted sleep
Restrictive lung disease/kyphoscoliosis
Hypoxemia
Fragmented sleep
Increased arousals
Increased Non-REM (NREM) sleep
Decreased stage N3 sleep, Stage R
GI Disorders
Arousals from acid reflux
Renal disorders
Difficulty initiating and maintaining sleep
Restless legssyndrome
Daytime somnolence
Sleep apnea
Endocrine disorders
Hypothyroidism
Obstructive sleep apnea (OSA)
Acromegaly
Diabetes
Difficulty initiating and maintaining sleep
Short and long sleep associated with diabetes
Associated with OSA
Infectious diseases
Viral and bacterial infections
Hypersomnolence
Disrupted sleep
HIV infection associated with poor sleep
Fibromyalgia syndrome
Reduction in total sleep time
Reduced sleep efficiency
Increase in NREM sleep
Decrease in REM sleep
Menopause-related sleep disorders
Insomnia
Sleep apnea
Restless legssyndrome
Sleep in cancer patients
Excessive fatigue
Leg restlessness
Insomnia
Excessive sleepiness

Source: Adapted from "Sleep-Related Problems in Common Medical Conditions," by J. Parish, 2009, *Chest, 135,* pp. 563–572.

Impaired cardiac function can produce symptoms such as chest pain and dyspnea that interfere with sleep and cause complaints of reduced total sleep, increased nighttime wakefulness, and symptoms of restless legs (Quan, 2009). The psychological impact of heart disease also has an impact on sleep and affects comfort, social relationships, and ability to work. Anxiety and depression are common after an MI with reports of insomnia lasting for months to years (Johansson, Karlson, Grankvist, & Brink, 2010).

Symptoms of insomnia are common even in patients with stable heart failure and adversely affect daytime symptoms and functional performance (Redeker, Jeon, et al., 2010; Redeker, Muench, et al., 2010). Insomnia is also common in patients with other forms of cardiovascular disease. Pharmacological and/or behavioral treatment for insomnia may improve sleep as well as daytime function in cardiovascular disease. (See Chapter 6, Insomnia.) However, the efficacy of these treatments in patients with cardiovascular disease is understudied.

Other symptoms of sleep problems commonly encountered (often associated with OSA and/or CSA) include difficulties with sleep onset and maintenance snoring, witnessed apneas or gasping, obesity, sleepiness, morning headache, and daytime sleepiness (Somers et al., 2008a). Unfortunately, short sleep durations are also associated with an

increased incidence of cardiovascular mortality (Ferrie et al., 2007).

OSA (see Chapter 8, Sleep-Related Movement Disorders and Parasomnias), characterized by partial or complete collapse of the airway, often induces severe intermittent hypoxemia and CO_2 retention during sleep. These changes result in disrupted autonomic and hemodynamic responses and initiate a range of pathophysiological mechanisms, which may act to promote cardiac and vascular disease. Heightened sympathetic drive, decreased cardiovascular variability, and the release of vasoactive and trophic substances (such as endothelin) may elicit vasoconstriction. Hypoxemia also appears to be an important trigger for systematic inflammation manifested by increased production of interleukin-6, C-reactive protein, adhesion molecules, and serum amyloid A as well as oxidative stress. Collectively, these abnormities result in endothelial dysfunction, increased insulin resistance, and platelet activation and thrombosis (Somers et al., 2008a). Treatment for this disorder may be behavioral (lateral positioning), weight loss (if indicated), or use of a dental appliance. Continuous Positive Airway Pressure (CPAP) is typically successful in treating OSA but compliance may be problematic. Additional long-term clinical trials designed to determine the efficacy of CPAP in improving clinical outcomes in patients with OSA and cardiovascular disease are needed (Somers et al., 2008b).

CSA (see Chapter 7, Sleep-Related Breathing Disorders) is a form of sleep-disordered breathing characterized by intermittent lack of respiratory effort. Cheyne-Stokes breathing is a form of CSA characterized by waxing and waning patterns of respiration. CSA has been documented in 40%–50% of heart failure patients with left ventricular ejection fraction below 40% (Quaranta, D'Alonzo, & Krachman, 1997) and can lead to fragmented sleep with frequent arousals and nocturnal oxygen desaturations resulting in poor sleep efficiency (Redeker, Muench, et al., 2010). These conditions also increase both arrhythmia risk and mortality (Javaheri et al., 1998; Lanfranchi et al., 1999). The pathophysiologcial mechanisms underlying CSA may include an increase in circulation time, increased sympathetic drive, and inflammation. However, the exact cause is difficult to identify because of other comorbidities often present and medications taken by this group of patients. A number of treatment strategies have been tested but, although some success has been documented, none have proven to be ideal in regard to efficacy, tolerance, or survival. These include nocturnal supplemental oxygen and theophylline. CPAP may improve some cases of CSA but a recent multicenter, randomized clinical trial of CPAP in patients with heart failure, failed to demonstrate improved clinical outcomes after a 2-year period (Bradley et al., 2005).

Unfortunately, sleep problems in patients with cardiovascular disease often go unrecognized and untreated. Clinician factors for this problem include lack of awareness of classic symptoms, lack of mechanistic research, and the need for multicenter random clinical trials. Enhanced education and additional research is needed. In addition, patients themselves may have an overall lack of awareness, reluctance to undergo diagnostic testing, and resistance to using CPAP (Malhotra & Loscalzo, 2009). Nursing can potentially play an important role in patient education and support as well as outcomes research (Redeker, 2002; Redeker, Muench, et al., 2010). A comprehensive scientific statement on nursing interventions designed to promote physical activities and dietary lifestyle changes for cardiovascular risk factor reduction was recently published (Artinian et al., 2010). Many of the interventions, strategies, and recommendations cited may positively affect sleep problems in this population warranting additional research in this area.

SLEEP AND DIABETES/OBESITY

The prevalence of type 2 diabetes and obesity is rapidly increasing in the United States (Mokdad et al., 2001). When a variety of variables are controlled, both of these disorders are associated with an increase in mortality risk and economic burden (Ettaro, Songer, Zhang, & Engelgau, 2004). Although the causes for these phenomena are not completely understood, changes in lifestyle that have occurred over

the past 50 years may play an important role. Of note is sleep curtailment. In fact, an analysis of a large sample conducted by the National Sleep Foundation observed that an increasing number of adult Americans slept 6 hours or less in 2004 when compared to 1985 (NSF, 2005). In addition, a number of epidemiologic studies, some controlling for covariates such as obesity, hypertension, and exercise, have demonstrated a consistent association between sleep problems and the risk of type 2 diabetes (Ayas et al., 2003; Knutson, Spiegel, Penev, & Van Cauter, 2007; Taub & Redeker, 2008).

Lack of sleep exerts deleterious effects on a variety of systems with detectable changes in metabolic, endocrine, and immune pathways. Short-term, acute, laboratory, and cross-sectional studies indicate that disturbed or reduced sleep (<5–7 hours) predisposes individuals to type 2 diabetes (Ayas et al., 2003; Beihl, Liese, & Haffner, 2009; Knutson et al., 2007; Vgonzas et al., 2009; Yaggi, Arauja, & McKinlay, 2006). Paradoxically, long sleep duration (>8–9 hours) has also been associated with the development of this disorder (Ayas, Mancini, & Fleetham, 2006; Beihl et al., 2009). Although a number of studies have suggested that these relationships were stronger in men than women (Mallon, Broman, & Hetta, 2005; Meisinger, Heier, & Loewel, 2005), a recent study demonstrated the reverse relationship (Tuomilehto et al., 2008).

Causal mechanisms underlying the relationship between sleep duration and both diabetes and obesity may, in part, be related to reciprocal changes in circulating levels of leptin and ghrelin leading to an increase in appetite and weight gain (Chaput, Despres, Bouchard, & Tremblay, 2007; Spiegel, Tasali, Penev, & Van Cauter, 2004). Leptin, an appetite-inhibiting hormone secreted by adipose tissue, promotes satiety. Ghrelin is a peptide released primarily from the stomach that increases appetite and food intake. Under normal conditions, the 24-hour profile of human plasma leptin levels shows a marked nocturnal rise, which is partly dependent on meal intake. The 24-hour profile of ghrelin levels also shows a nocturnal rise but levels spontaneously decrease in the second half of the sleep period. Sleep deprivation reverses these relationships leading to an inappropriate increase in appetite, especially in the evening. Other factors include reduced energy expenditure related to fatigue, increased insulin resistance, increased cortisol, and low-grade inflammation (see Figure 11.1) (Spiegel, Knutson, Leproult, Tasali, & Van Cauter, 2005; Van Cauter et al., 2007; Van Cauter,

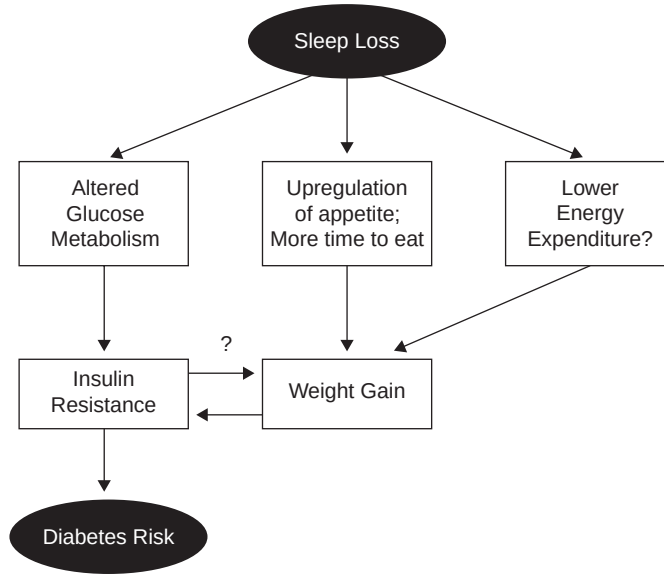

Figure 11.1 ■ Schematic of the potential pathways leading from sleep loss to diabetes risk.

Spiegel, Tasali, & Leproult, 2008; Williams, Hu, Patel, & Mantzoros, 2007).

OSA is also more prevalent in diabetic patients when compared with those without the disorder (Elmasry et al., 2001). The frequent episodes of apneas, arousals, and hypoxemia typical of OSA appear to contribute to the development of diabetes because of increased insulin resistance, hyperinsulinemia, metabolic syndrome, and heightened sympathetic activity all of which may contribute to its development. Obesity seems to be a common risk factor for both of these disorders and may explain their relationship (Taub & Redeker, 2008).

Conversely, diabetes is often associated with a variety of symptoms, which may trigger the development of sleep problems. These include anxiety, depression, painful neuropathy, nocturia, and restless legs. The symptoms may interact in a vicious cycle, which leads to adverse effects on glucose control (Cuellar & Ratcliffe, 2008; Taub & Redeker, 2008). Research regarding the mechanisms by which symptoms in general interact with sleep and lead to poor clinical outcomes is greatly needed (Parker, Kimble, Dunbar, & Clark, 2005).

In summary, although numerous confounding factors may have affected study results, a consistent relationship between sleep duration and the expression of type 2 diabetes has been observed. It has also been shown that a decrease in subjective sleep predicts poorer control in patient with type 2 diabetes (Knutson, Ryden, Mander, & Van Cauter, 2006) and that weight loss improves clinical outcomes in patients with OSA and diabetes (Foster et al., 2009). Given that obesity and diabetes are also risk factors for cardiovascular disease, sleep duration and quality should be carefully assessed, particularly in patients experiencing problems with glycemic control.

SLEEP AND CANCER

Sleep problems are common in approximately 30%–57% of cancer patients (Barton-Burke, 2006; Janz et al., 2007; Palesh et al., 2010; Savard & Morin, 2001a; Vena, Parker, Cunningham, Clark, & McMillan, 2004). Subjective reports reveal a pattern of difficulty getting to sleep, early morning awakenings, prolonged nocturnal waking periods, and unrefreshing sleep (Anderson et al., 2003; Parker et al., 2008). Daytime sleepiness is prevalent. Sleep problems may also trigger or enhance other symptoms such as pain, depression, fatigue, anxiety, night sweats, and restless legs (Flynn et al., 2010; Lee, 2001; Lee, Portillo, & Miramontes, 2001; Miaskowski & Lee, 1999b; Parker et al., 2005). Unfortunately, the results of a study conducted in 1999 suggest that 85% of cancer patients did not communicate these problems to their physicians—often because they thought, nothing could be done (Engstrom, Strohl, Rose, Lewandowski, & Stefanek, 1999). Nonetheless, over 40% of all prescriptions written for this population are hypnotics (Derogatis et al., 1979; Stiefel, Kornblith, & Holland, 1990), an observation that highlights the magnitude of the problem.

Studies using actigraphy, an instrument which estimates sleep parameters from activity levels (see Chapter 1, Physiological and Behavioral Aspects of Sleep and Chapter 5, Conducting a Sleep Assessment), have provided important insights into the nature of these complaints. The results from one study demonstrated that women with breast cancer had disturbed sleep and those with delayed circadian rhythms had more daytime dysfunction and fatigue (Ancoli-Israel et al., 2006). Berger et al. found that fatigue that was associated with a greater number of nighttime awakenings was greater during chemotherapy and negatively correlated with activity (Berger, 1998; Berger & Farr, 1999). Polysomnographic evaluations of nocturnal sleep reveal altered sleep architecture (Friedman et al., 2001; Parker et al., 2008; Silberfarb, Hauri, Oxman, & Lash, 1985; Silberfarb, Hauri, Oxman, & Schnurr, 1993).

Treatments for cancer may disrupt sleep because of their emotional impact, physiologic effects, or side effects (Savard & Morin, 2001b). For example, sleep disturbances and daytime sleepiness have been reported in patients undergoing chemotherapy (Berger & Higginbotham, 2000; Broeckel, Jacobsen, Horton, Balducci, & Lyman, 1998; Palesh et al., 2010; Redeker, Lev, & Ruggiero, 2000) and in response to hormonal

therapy (i.e., tamoxifen) (Broeckel et al., 1998; Couzi, Helzlsouer, & Fetting, 1995b; Mourits et al., 2001; Stein, Jacobsen, Hann, Greenberg, & Lyman, 2000). Problems with nocturnal sleep disturbances and/or daytime sleepiness have also been reported in patients undergoing radiotherapy (Beszterczey & Lipowski, 1977; Faithfull & Brada, 1998; Miaskowski & Lee, 1999a).

Although the mechanisms by which chemotherapy and radiotherapy may interfere with sleep remain to be fully elucidated, some suggest that changes in cytokine expression may play a prominent role (Belka, Budach, Kortman, & Bamberg, 2001; Greenberg, Gray, Mannix, Eisenthal, & Carey, 1993). Use of cytokines, especially interferon, interleukin-2, and tumor necrosis factor, is associated with a variety of side effects including daytime sleepiness, disturbed sleep, and depression (Capuron, Ravaud, & Dantzer, 2000; Valentine, Meyers, Kling, Richelson, & Hauser, 1998). Estrogen deficiency resulting from treatment can cause menopausal symptoms that can interfere with sleep. Hot flashes and night sweats occur in at least one half of women treated with tamoxifen (Fisher, Costantino, & Redmond, 1989; Love, Cameron, & Connell, 1991) and often occur in men with prostate cancer treated with androgen deprivation therapy (Erlichman & Loprinzi, 1997). Couzi et al. noted a linear association between severity of hot flashes and sleep disturbances in women treated for breast cancer (Couzi, Helzlsouer, & Fetting, 1995a).

Reducing the severity and impact of symptoms has long been an important priority in the management of individuals with cancer. However, specific guidelines for symptom management are lacking. Recently, an interdisciplinary workgroup, Assessing the Symptoms of Cancer using Patient-Reported Outcomes (ASCPRO), has been formed to generate evidence-based recommendations for patient-reported outcomes assessment focusing on nonpain symptoms, including fatigue and sleep disturbance (Cleeland & Sloan, 2010). An excellent update on the state of the sleep science and summary of interventions and randomized clinical trials was also recently published (Berger, 2009). Promising nonpharmacologic interventions addressed in this later report include cognitive behavioral therapy and complementary/alternative treatments (see Chapter 6, Insomnia and Chapter 14, Complementary and Alternative Medicine (CAM) and Sleep.) Other potentially effective interventions that have been tested included patient education and behavioral strategies delivered face to face or by telephone (Barsevick et al., 2010), mindfulness-based stress reduction (Kvillemo & Branstrom, 2011), and home-based exercise during and after chemotherapy with or without radiotherapy (Dodd et al., 2010).

SLEEP AND HIV/AIDS

Human immunodeficiency virus (HIV) infection is often accompanied by sleep disruption (Vance & Burrage, 2005). One study (Rubinstein & Selwyn, 1998) demonstrated that 73% of 115 HIV-positive patients had sleep disturbance, an observation consistent with the results of several additional studies (Cohen, Ferrans, Vizgirda, Kunkle, & Cloninger, 1996; Hudson, Lee, & Portillo, 2003). Sleep disturbances appear to develop relatively soon after the initial infection, continue across the disease course, and increase with advancing disease (Moeller et al., 1991; Savard et al., 1999). Sleep problems have also been reported in children with HIV (Franck et al., 1999).

Difficulties with initiating and maintaining nocturnal sleep and daytime sleepiness are among the most common complaints (Phillips, 1999; Rubinstein & Selwyn, 1998). One study reported that HIV-infected men had more nocturnal awake time when compared with healthy controls (Norman et al., 1992). Similar findings have been reported by other researchers (Rubinstein & Selwyn, 1998; Wiegand, Moller, Schreiber, Krieg, & Holsboer, 1991). HIV-infected patients seem to sleep and nap more, have more early morning awakenings, and be less alert in the morning (Darko, McCutchan, Kripke, Gillin, & Golshan, 1992). Insomnia (difficulties was sleep onset and reduced total sleep time) or excessive sleepiness (total sleep time >10 hours) (Cohen, Ferrans, Vizgirda, Kunkle, & Clonginer, 1996) are also problematic.

Polysomnographic measures of sleep appear to be affected by the disease. One study using polysomnography (PSG) revealed that among asymptomatic HIV-infected males, subjects experienced an increase in deep sleep (slow-wave sleep) and nocturnal arousals (Norman et al., 1992; Norman, Chediak, Kiel, & Cohn, 1990; Norman et al., 1988). Other researchers reported a high level of daytime sleepiness in HIV-positive subjects, possibly related to the presence of significant OSA secondary to adenotonsillar hypertrophy (Epstein et al., 1995). Moderate to severe sleep-related breathing disorders were present in 57% of a sample of 99 men ($n = 58$ on highly active antiretroviral therapy [HAART] and 41 not on antiretroviral therapy), an observation noted only in the HARRT group and one which was significantly correlated with an increase in body mass index and both waist and neck circumference (Brown et al., 2010). In a sample of 100 women, a study using actigraphy demonstrated that those with high fatigue experienced more problems with falling asleep, more nocturnal awakenings, poor daytime function, and worse depression when compared with low-fatigue (Lee et al., 2001).

Numerous physiological factors regulating sleep and circadian rhythms may be affected by HIV. Central to these are changes in the hypothalamic-pituitary-adrenal axis circadian rhythm. HIV is associated with decreased production of growth hormone, increased production of corticotrophin-releasing factor and adrenocorticotropin hormone (ACTH), and elevation of cytokines (interleukin-1-β, interleukin-6, and tumor-necrosis factor-α) (Kumar, Kumar, Walfrop, Antoni, & Eisdorfer, 2003; Phillips, 1999). Degeneration of central dopaminergic systems may also play a role in the development of sleep-wake cycle abnormalities (Berger & Arendt, 2000; Koutsilieri, Scheller, ter Meulen, & Riederer, 2005).

The impact that sleep problems have on the clinical outcomes of HIV/AIDS patients remains to be described. However, one study of 57 HIV-positive men and women observed that higher levels of distress were significantly related to lower T-cytotoxic/suppressor (CD3+ CD8+) cells and that sleep disturbances mediated this relationship (Cruess et al., 2003). White et al. observed a correlation between CD4 T cell counts and increased SWS during the last half of the night (White et al., 1995). Poor sleep also appears to be related to impaired functional status (Nokes & Kendrew, 2001). These results suggest that interventions designed to enhance sleep may improve immune function in the patients. Research in this area is greatly needed.

SLEEP AND RENAL DISEASE

End-stage renal disease (ESRD) is a significant health problem in the United States. By the end of 2007, the prevalence rate of patients on dialysis in this country rose to 1,665 per million population, or over 600,000 people. While this rate is almost 19% greater than that noted in 2000, the annual rate of growth has remained between 2.0 and 2.3 since 2003. However, there remains a disproportionately high prevalence of this disorder in African and Native Americans (US Renal Data System [USRDS], 2010).

Sleep complaints and primary sleep disorders such as OSA and CSA (Beecroft, Pierratos, & Hanly, 2009), restless legs syndrome (RLS) (Araujo et al., 2010), and periodic limb movement disorder (PLMD) (Loewen, Siemens, & Hanly, 2009) are very common in these groups (Gusbeth-Tatomir, Boisteanu, Seica, Buga, & Covic, 2007) and occur in an average of 43% of the population (Murtagh, Addington-Hall, & Higginson, 2007). Daytime sleepiness is common and dialysis patients reported napping for periods averaging 1.1 ± 1.3 hours per day (Parker, Bliwise, Bailey, & Rye, 2003). Sleep problems are among the most disturbing symptoms experienced by this group (Eichel, 1986; Kumar et al., 2010; Molzahn, Northcott, & Dossetor, 1997; Parker, Kutner, Bliwise, Bailey, & Rye, 2003) and are also associated with an increase in mortality (Benz, Pressman, Hovick, & Peterson, 2000; Unruh et al., 2004).

Numerous demographic, clinical, and laboratory correlates of sleep complaints in the dialysis population have been identified. These include increased age (Holley, Nespor, & Rault, 1991, 1992; Kutner, Bliwise, Brogan, & Zhang, 2001; Walker, Fine, & Kryger, 1995),

male gender (Kutner et al., 2001; Walker et al., 1995), and white race (Kutner et al., 2001). Positive relationships between subjective sleep complaints and caffeine intake, pruritus, bone pain, cigarette use, and premature discontinuation of dialysis have also been reported (Holley et al., 1992; Walker et al., 1995). Although no consistent relationships between subjective sleep complaints and BUN (blood urea nitrogen), creatinine, or Kt/V (a measure of dialysis adequacy) have been detected (Holley et al., 1992; Puntriano, 1999; Walker et al., 1995), anemia is associated with complaints of poor sleep with improvement noted after treatment with recombinant erythropoietin (Evans, Rader, & Manninen, 1990).

The mechanisms underlying the increased prevalence of OSA and CSA in dialysis patients remain to be fully elucidated, but numerous theories have been proposed. Hypocapnia from metabolic acidosis and acidemia may predispose the patients to an unstable breathing pattern (Fraser & Arieff, 1988; Kimmel, Miller, & Mendelson, 1989; Mendelson, Wadhwa, Greenberg, Gujavarty, & Bergofsky, 1990). In addition, accumulation of uremic toxins may affect the central nervous system and result in a reduction of airway muscle tone during sleep, a discoordination of diaphragm, an upper airway muscle activity, or an instability of respiratory control (Fein, Niederman, Imbriano, & Rosen, 1987). Anemia, hormone abnormalities, inflammation, cytokine production during hemodialysis (HD) (Fein et al., 1987; Fletcher, 1993; Kimmel, 1989; Koehnlein et al., 2009; Santiago, Edelman, & Fishman, 1975; Zoccali, 2000), and the mechanical effects of peritoneal fluid on diaphragmatic action may all contribute to ventilatory control instability (Fletcher, 1993; Hanly, 2008).

The pathophysiologic mechanisms involved in RLS and PLMD are unknown (Montplaisir, Nicolas, Godbout, & Walters, 2000; Winkelmann & Trenkwalder, 2001). (See Chapter 8, Sleep-Related Movement Disorders and Parasomnias.) Anemia, iron and vitamin deficiencies, disturbances in peripheral and central nervous system functioning, and musculoskeletal abnormalities have all been proposed as contributing factors (ASDA, 1997; Montplaisir et al., 2000; Winkelmann & Trenkwalder, 2001). Given the response of the conditions to dopamine (DA) agonists and opiates, it is likely that neurotransmitter disturbances play an important role (Montplaisir et al., 2000; Winkelmann & Trenkwalder, 2001). Within this context, several factors intrinsic to the uremic state may predispose dialysis patients to RLS and PLMD.

Treatment for sleep apnea has been successfully accomplished in dialysis patients with CPAP (Pressman, Benz, Schleifer, & Peterson, 1993). However, the long-term impact has not been assessed and improvements in cognition and other aspects of functional status in dialysis patients remain to be described (Hanly, 2008). In addition, studies regarding CPAP compliance rates are needed, particularly in view of the numerous limitations already imposed on patients by virtue of their disease and treatment. Several research groups have reported that sleep apnea is cured in a limited number of patients (Auckley, Schmidt-Nowara, & Brown, 1999; Beecroft, Zaltzman, Prasad, Meliton, & Hanly, 2007; Langevin, Fouque, Leger, & Robert, 1993).

The general management of RLS and PLM include reduction of lifestyle risk factors (caffeine, alcohol, and nicotine), elimination of medications which exacerbate these conditions (tricyclics antidepressants and serotonin reuptake inhibitors), and medical conditions (anemia and iron deficiency). The beneficial effects of treatment with DA in dialysis patients with RLS/PLMD (see below) indeed suggest that dopamine pathways are involved. Pharmacological interventions for RLS and PLMD include DA, dopamine precursors, benzodiazepines, and opiates. Neuroleptics, such as gabapentin and carbamazapine, are also effective in some patients. (See Chapter 8, Sleep-Related Movement Disorders and Parasomnias.) One recent study demonstrated a significant improvement in RLS following renal transplant (Azar, Hatefi, & Talebi, 2007).

In summary, sleep disturbances are very prevalent in dialysis patients and appear to have important adverse affects on their overall health, well-being, and survival. Therefore, the effective assessment and management of these

sleep disturbances has the potential to significantly enhance patient outcomes. In addition, research designed to identify the mechanisms underlying these sleep problems would expand both the clinical and basic sciences. Thus, the dialysis population presents clinicians and researchers alike with an extraordinary opportunity for interdisciplinary collaboration.

SLEEP AND CHRONIC PAIN

Chronic pain affects over 76 million Americans and is associated with significant human and economic costs (APF, 2010). Pain is especially problematic for numerous individuals with comorbid conditions (see Table 11.1). Unfortunately, there are a number of barriers in our society and culture that place access to pain management out of reach of many individuals. Educating the medical community, public, implementing standards of care, and ensuring adequate reimbursement are crucial to initiate change.

Between 30% and 60% of patients in pain complain of sleep difficulties (Ashworth, Davidson, & Espie, 2010; Dorrepaal, Aaronson, & van Dam, 1989; Ripamonti et al., 2000), problems which are significantly associated with the intensity and severity of pain (McMillan, Tofthagen, & Morgan, 2008). Adequate control of pain often results in a reduction in the occurrence and severity of insomnia (Meuser et al., 2001). Treatment with opioids, a group of medications commonly used to treat pain, adversely affects sleep (Mystakidou et al., 2010); high doses disrupt virtually all stages of sleep, decrease or abolish REM, and increase nighttime awakenings (Kay, Eisenstein, & Jasinski, 1969; Knill, Moote, Skinner, & Rose, 1990). In addition, opioids may disrupt the circadian rhythm by altering episodes of sleep and waking (Byku & Gannon, 2000a, 2000b; Byku, Legutko, & Gannon, 2000; Meijer et al., 2000). Nonsteroidal anti-inflammatory drugs may also decrease deep sleep and increase nocturnal awakenings (Horne, Percival, & Traynor, 1980; Landis, Levine, & Robinson, 1989).

Sleep disruption itself appears to increase the perception of pain and may even trigger its development (Moldofsky & Scarisbrick, 1976; Shaver et al., 1997). It has long been noted that waking each morning feeling unrefreshed, fatigued, and tired are extremely common symptoms experienced by patients with fibromyalgia, a syndrome characterized by somatic pain (Wolfe et al., 1990). Moldofsky and his colleagues were the first to describe a particular sleep EEG abnormality (alpha-delta sleep) associated with this condition (Moldofsky & Scarisbrick, 1976). When alpha-delta sleep was experimentally induced in a group of normal volunteers by selective SWS interruption and deprivation, they developed muscular fatigue and tenderness within a few days (Moldofsky & Scarisbrick, 1976). Restricting sleep to an average of approximately 5 hours a night for seven consecutive nights also resulted in an increase in somatic complaints including headaches, gastrointestinal problems, and sore joints in a group of normal subjects (Dinges et al., 1997). Similarly, in a study of 12 healthy women, Lentz et al. (Lentz, Landis, Rothermel, & Shaver, 1999) found that disrupting SWS, without reducing total sleep time or sleep efficiency, for several consecutive nights was associated with decreased pain threshold and increased discomfort.

Several pain and sleep experts have suggested that these two symptoms are often interrelated and that pain may interact with sleep disturbances predisposing individuals to even greater morbidity (Lee, 2003; Miaskowski & Lee, 1999b). The relationship between pain and impaired sleep is complex and little is known about treating the two problems when both are present together. Successful management of chronic/persistent pain should be individually tailored to each patient, taking into account his or her pain intensity and duration, disease state (Zorba Paster, 2010).

ASSESSMENT, DIAGNOSIS, AND FOLLOW-UP CARE

Thorough assessment of sleep is an important component of nursing care of individuals who have chronic medical conditions, especially because these individuals may be at higher risk

for sleep disorders than others in the general population. (See Chapter 1, Physiological and Behavioral Aspects of Sleep and Chapter 5, Conducting a Sleep Assessment.) As reviewed in this chapter, it is evident that sleep disturbances may contribute to the development of chronic medical conditions; medical conditions and associated symptoms and medications may lead to sleep disturbances; and in some cases, sleep disorders and medical conditions may share underlying pathophysiology. Sleep disorders may also exacerbate the pathophysiological and functional consequences of chronic conditions. These observations underscore the importance of a careful evaluation of the characteristics of sleep disorders, factors that may contribute to sleep disorders (e.g., medications, other symptoms), and the potential contributions of sleep disorders to outcomes (e.g., quality of life, function, pathophysiology).

Assessment of sleep in patients with medical conditions should be organized as discussed in Chapter 5 of this book, with particular focus on the potential contributions of sleep disorders to pathophysiological (e.g., metabolic control in diabetes; blood pressure in patients with hypertension) and functional/quality of life outcomes because these patients are at high risk for these consequences. Review of medications is particularly important. (See Chapter 5, Conducting a Sleep Assessment.)

The general treatment plan should include education about sleep and on promoting adequate, restful, and restorative sleep for patients. This can be accomplished by preventing or reducing the factors that are disturbing the patient's sleep or have potential to do so and providing bedtime routines, comfort measures, and a setting conducive to sleep. Although further research is needed to support the most efficacy treatments, optimal treatment of the underlying medical disorder and symptom management often helps reduced sleep problems.

As reviewed in this chapter, it is evident that subgroups of patients with medical conditions are at high risk for specific "primary" sleep disorders (e.g., RLS, sleep disordered breathing, periodic limb movements during sleep) that require treatment by a sleep specialist. Therefore, these patients should be referred to sleep disorders centers. Important components of nursing care upon returns to the referring clinical setting are follow-up to assure that patients are willing and able to adhere with treatment (e.g., CPAP); ongoing evaluation of treatment effects; and assisting patients with troubleshooting problems that may arise. (See Chapter 7, Sleep-Related Breathing Disorders and Chapter 8, Sleep-Related Movement Disorders and Parasomnias.) Behavioral treatment for insomnia is promising for some group of patients who have insomnia comorbid with medical conditions (e.g., pain, heart disease, cancer). Depending on the severity of the problem, insomnia can be managed in the general medical setting or may require a sleep specialist. (See Chapter 6, Insomnia.)

Chronic medical conditions are often the cause or a secondary diagnosis associated with admission to an acute or intensive care setting. Given the close association of these conditions with sleep disorders, it is critical that nurses are aware of the relationships between medical conditions and sleep, as they often have an impact on sleep during acute care hospitalization. (See Chapter 20, Sleep in Adult Acute and Critical Care Settings.)

SUMMARY

Individuals with a variety of medical conditions are at high risk for sleep problems. Despite the widely held belief that sleep promotes well-being, care practices are rarely designed to encourage optimal sleep and promote maximal daytime alertness. Assessment and treatment of sleep disturbance and sleep disorders should be an integral component of disease management for these individuals, with referral for specialized sleep treatment as necessary. Further research is needed to better characterize the nature of sleep disorders in individuals with medical conditions; factors that contribute to sleep disorders in these patients; consequences of sleep disorders; and most importantly, the efficacy and effectiveness of sleep-promoting treatments. These treatments may improve sleep and important pathophysiological and functional outcomes.

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Sleep and Psychiatric Disorders

12

Geoffry Phillips McEnany

I am a shepherd of those sheep
That climb a wall by night,
One after one, until I sleep,
Or the black pane goes white.
Because of which I cannot see
A flock upon a hill,
But doubts come tittering up to me
That should by day be still.
And childish griefs I have outgrown
Into my eyes are thrust,
Till my dull tears go dropping down
Like lead into the dust.
—Nuit Blanche, Edna St. Vincent Millay

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Sleep is of paramount importance to the care of people who live with psychiatric disorders, but the relationships between sleep and psychiatric disorders is complex. Sleep dysregulation is almost universal in psychiatric illness, whether in the form of insomnia (more common), hypersomnia, or disturbed circadian patterning of sleep-wake. Sleep disturbance and psychiatric disorders may both reflect underlying abnormalities in the central nervous system, and sleep symptoms are a component of many psychiatric diagnoses. Sleep disorders may contribute to or result from psychiatric disorders but may also result from medication treatment.

Scientific interest in the relationship between sleep and psychiatric illness spans more than 30 years. Benca Obermeyer, Thisted, and Gillin (1992) ground-breaking historical meta-analysis of 177 studies representing 7,151 participants demonstrated the virtually universal nature of sleep disturbance and its association with psychiatric disorders. The objective findings revealed widespread reductions in sleep efficiency, total sleep time, and non-REM sleep. Although no single sleep alteration is specific for any particular psychiatric disorder, the patterns of sleep were most disturbed among patients with mood disorders. The investigators did not address the corollary of the lived experience associated with these objective changes in sleep architecture. However, Benca (2008) acknowledges that poor sleep efficiency and reduced sleep time result in sleep deprivation, namely daytime sleepiness, reduced energy, and possible cognitive cloudiness. These findings broadened the scientific perspective on sleep as a critical component in conceptualizing psychiatric disease, and interest in this area of research and practice remains strong (Krahn, 2005; Stores, 2007). For example, in a recent critical review on sleep and psychiatric disorders, Sateia (2009) documents high rates of comorbidity between sleep (especially insomnia) and various psychiatric disorders, especially mood, and anxiety disorders.

Untreated sleep-related symptoms increase morbidity, decrease quality of life, and are associated with, impressive costs to the health care system. While there are no economic data on the overall impact of insomnia in the United States alone, Canadian data may provide a close approximation. Daley, Morin, LeBlanc, Gregoire, and Savard (2009) estimated the total annual cost of insomnia in the province of Quebec to be 6.5 billion Canadian dollars, which represents approximately 1% of the province's $228.5 billion in gross domestic product for 2002. In 2008 in the United States alone, 56,287,000 prescriptions were dispensed for sleep medications, a 7% rise since 2007. The implications of these data are compelling for contemporary nursing practice across all specialty areas of practice. Because nurses frequently care for patients who have insomnia, the specific approach to treatment has a powerful economic impact. Implementation of effective treatment strategies requires a detailed understanding of insomnia and the rationale behind effective treatment, as well as the impact of the 24-hour day. de Niet, Tiemens, Lendemeijer, and Hutschemaekers (2008), a group of nurse researchers, acknowledge that sleep disturbances are often misconstrued by providers as a night time concern and erroneously compartmentalize sleep to the darker hours of the 24-hour day. Such a myopic perspective will perpetuate inadequate assessment of and intervention for insomnia, contributing to the economic burden associated with the condition.

This chapter will familiarize the reader with interactions between sleep and sleep disorders and psychiatric disorders. Implications for nurses who work with patients who have psychiatric disorders are discussed. Given the high prevalence of sleep disorders, their associations with medical, as well as psychiatric disorders, and their presence in patients who present for care across a wide range of clinical, community-based and hospital settings, the implications are broad. Patients with psychiatric disorders are treated across many practice settings. Therefore, the content of this chapter holds relevance for all nurses.

The discussion addresses some of the most clinically salient and well-documented linkages between sleep and psychiatric illness and includes common mood disorders, anxiety disorders, schizophrenia, alcoholism, and attention deficit hyperactivity disorder (ADHD). The discussion of each diagnostic category addresses pharmacologic and behavioral interventions related to treatment of the particular disorder as it relates to sleep. Although this chapter focuses primarily on care of adults, childhood issues are addressed in the content related to ADHD. To clarify the discussion, important information is provided in a series of tables throughout the chapter: information on the diagnostic features of the disorders; changes seen in the polysomnographic changes in sleep across these illnesses; and the impact of pharmacologic treatment on sleep.

SLEEP AND MOOD DISORDERS

Depressive illness has a number of manifestations that are reflected in the breadth of diagnoses. While major depression is the most disabling, other types of depression include dysthymic disorder, depressive disorder related to a medical illness, depression related to substance abuse, and depression not otherwise specified. While each of these types of depression share a common set of core symptoms, they are distinguished by their severity, course, and etiology. Course specifiers are used to identify specific manifestations of a depressive illness and include conditions such as seasonal variance or postpartum depression (American Psychiatric Association [APA], 2000c). The symptom clusters of depression are in Table 12.1.

Unipolar Depressive Disorders

Major depression is the leading cause of disability in the age group between 15 and 44 years of age. It affects approximately 14.8 million American adults, or about 6.7% of the U.S. population aged 18 and older in a given year. Major depression is more common in women than men (Source: http://www.nimh.nih.gov/health/publications) and is distinguished by acute episodes of illness. However, dysthymia, a less severe disorder,

Table 12.1 ■ *Major Symptom Clusters of the Psychiatric Disorders*

Mood Disorders

- Depression: Depressed mood, decreased interest in usual activities, low energy, thoughts of death or suicide, sleep disturbances manifest as either insomnia or hypersomnia, diminished ability to take pleasure in usual activities, changes in appetite and libido, guilt, and diminished cognitive capacities.
- Mania/hypomania: Irritability, high energy, reduced need for sleep, grandiosity, racing thoughts, pleasure-seeking activities, and others.

Anxiety Disorders

- Panic: Perception narrows, accompanied by a sense of impending doom and the experience of severe anxiety, severe fearful anticipation (often a fear of dying or experiencing a life-threatening event such as a heart attack), increased heart rate, sweating, tension, paresthesias, trembling, and others. Symptoms may be accompanied by agoraphobia, best characterized by an intense fear of going places or being in situations with crowds.
- PTSD: Symptoms are preceded by a traumatic or life-threatening event followed by emotional numbing, intrusive recollection of the traumatic event (flashbacks, nightmares), avoidance of stimuli associated with the trauma, hyperarousal (exaggerated startle, difficulty falling asleep, irritability, difficulty concentrating, angry outbursts).
- OCD: Obsessive thoughts such as fear of contamination coupled with compulsive behaviors such as washing or cleaning, intense anxiety associated with the obsessive/compulsive patterns.
- GAD: Worry, apprehension, irritability, difficulty with concentration and fearful expectation, restlessness, fatigue, difficulty with sleep, and muscle tension.

Schizophrenia

- Symptom clusters bifurcate along two lines: negative and positive symptoms. Negative symptoms: affective flattening, alogia (inability or unwillingness to speak due to pathology), anhedonia, and social disengagement. Positive symptoms: overt hallucinations, delusional thinking, agitated behavior evidenced in pacing or assuming unusual postures.
- Schizoaffective disorder, a variant, includes all the core symptoms of schizophrenia coupled with either depression or bipolar symptom clusters.

Alcoholism

- Characterized by abuse (frequent drinking that causes impairment in social/occupational functioning, often with legal consequences); dependence (unable to reduce or stop using alcohol with signs and symptoms of physiologic withdrawal after stopping).

ADHD

- Inattentive subtype: Inattention to details; careless mistakes; trouble keeping attention on tasks or play activities; does not seem to listen when spoken to directly; does not follow instructions and fails to finish tasks; trouble organizing activities; avoids activities that require sustained attention; loses things needed for tasks; easily distracted; forgetful.
- Hyperactive subtype: Fidgets with hands or feet or squirms in seat/difficulty remaining seated; impulsive
- Combined subtype: Criteria from both categories are present simultaneously.

Source: *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., Text Rev.) by American Psychiatric Association, 2000, Washington, DC: Author.

Abbreviations: GAD, generalized anxiety disorder; OCD, obsessive compulsive disorder; PTSD, posttraumatic stress disorder.

encompasses a trajectory of symptoms lasting 2 or more years (APA, 2000c). Depression related to medical illness or substance abuse shares the core symptoms of major depression but is secondary to a medical disorder (Dobkin et al., 2010) or addiction (Soares, 2010).

Depression rarely occurs in a single episode. The risk of recurrence increases with the number of episodes of depression (Suija, Aluoja, Kalda, & Maaroos, 2011). The chronic nature of this condition, if untreated, is underscored by the fact that the risk for another episode increases by 90% after three episodes.

Characteristics of Disturbed Sleep in Unipolar Depressive Disorders

Disturbed sleep is ubiquitous across the depressive disorders. Conversely approximately 20% of patients with insomnia meet diagnostic criteria for depression (Tsuno, Besset, & Ritchie, 2005). Early research (Kupfer, Frank, Grochocinski, Gregor, & McEeachran, 1988) focused on common changes in the sleep architecture of those diagnosed with major depression. Table 12.2 details the sleep changes that are common to depression. There are some consistent trends across the sleep patterns

Table 12.2 ■ *Common Features of Sleep Disturbances in Psychiatric Disorders*

Depression *(Common to both unipolar and bipolar depression)*

- **REM abnormalities** ([1] Early REM latency, defined as the time of the first REM period after sleep onset. Normally the first REM period occurs between 60 and 90 minutes, but in depression 20–30 minute onset is not uncommon. [2] Changes in REM architecture including an increase in the amount of rapid eye movement and an abnormally high percentage of REM in the earlier part of the sleep period.)
- **Reduction in slow-wave sleep [SWS]** (Defined as non-REM Stages 3 and 4, considered to be deep, restorative sleep.)
- **Changes in sleep continuity** (Difficulty getting to sleep, staying asleep, early morning awakening, poor sleep efficiency, and overall reduced sleep time.)

Mania

- Shortened sleep duration
- Sleep architecture variables are similar to those seen in major depression.

Panic

- Nocturnal panic may be associated with respiratory events but specific sleep architecture changes are not evident.
- Nocturnal panic is an NREM event and occurs in the transition from Stage 2 to Stage 3 sleep.

PTSD

- High degree of arousal in sleep
- Poor sleep efficiency
- Nightmares preceded by REM sleep

OCD

- Reduced sleep continuity and overall sleep time
- Reduced Stage 2 and Stage 4 sleep
- Some evidence of sleep onset REM

GAD

- Long sleep latency
- Poor sleep efficiency
- Early morning awakenings
- Reduced total sleep time

Schizophrenia

- Prolonged sleep latency
- Decreased sleep efficiency
- Decreased total sleep time
- Shortened REM latency

Alcoholism

- Prolonged sleep latency
- Frequent awakenings
- Reduced slow-wave sleep
- Increased REM pressure

ADHD

- Prolonged sleep latency in adults
- In children: fragmented sleep, motor restlessness, presence of parasomnias

Sources: *ACCP 2008 Sleep Medicine Board Review* (pp. 71–87), by R. Benca, 2008, Chicago: AACP Sleep Institute. "Sleep and Psychiatric Disorders: A Meta Analysis," by R. M. Benca, W. H. Obermeyer, R. A. Thisted, and C. Gillin, 1992, *Archives of General Psychiatry, 49*(8), pp. 651–668. "Impact of Alcoholism on Sleep Architecture and EEG Power Spectra in Men and Women," by I. M. Colrain, S. Turlington, and F. C. Bake, 2009, *Sleep, 32*(10), pp. 1341–1352. "Polysomnographic Characteristics of Young Manic Patients. Comparison With Unipolar Depressed Patients and Normal Control Subjects," by J. I. Hudson, J. F. Lipinski, P. E. Keck, Jr., H. G. Aizley, S. E. Lukas, and A. J. Rothschild, 1992, *Archives of General Psychiatry, 49*(5), pp. 378–383. "Sleep Onset REM Periods in Obsessive Compulsive Disorder," by M. Kluge, P. Schussler, M. Dresler, A. Yassouridis, and A. Steiger, 2007, *Psychiatry Research, 152*, pp. 29–35; Kraske and Tsao, 2004. "Sleep Events Among Veterans With Combat-related Posttraumatic Stress Disorder," by T. A. Mellman, R. Kulick-Bell, L. E. Ashlock, and B. Nolan, 1995, *American Journal of Psychiatry, 152*(101), p. 110. "Sleep Disorders in Children With Attention-Deficit/Hyperactivity Disorder (ADHD) Recorded Overnight by Video-Polysomnography," by R. Silvestri, A. Gagliano, I. Arico, T. Calarese, C. Cedro, O. Bruni, et al., 2009, *Sleep Medicine, 10*, pp. 1132–1138. "Association Between Attention-Deficit/Hyperactivity Disorder and Sleep Impairment in Adulthood: Evidence From a Large Controlled Study," by C. B. Surman, J. J. Adamson, C. Petty, J. Biederman, D. C. Kenealy, M. Levine, et al., 2009, *Journal of Clinical Psychiatry, 70*(11), pp. 1523–1529.

Abbreviations: ADHD, attention deficit hyperactivity disorder; GAD, generalized anxiety disorder; OCD, obsessive compulsive disorder; PTSD, posttraumatic stress disorder.

of those with psychiatric illness. Commonly, sleep efficiency is poor and associated with significant arousal. There is often a reduction in the deep, restorative stages of sleep (NREM Stages N3), and changes in REM architecture are common. The person who is depressed experiences sleep deprivation, including sleepiness, low energy, and blunted cognitive functions. However, most patients are not aware of the changes in sleep architecture other than their lived experience. This is very different than the symptom experience associated with other conditions such as angina where the symptoms and the source are readily identified by the patient and clinician alike.

Sleep architecture changes documented in depression vary according to a number of related variables such as severity of illness, age, comorbidities, and gender. Common confounders in sleep and mood disorders include gender (Lee, 1988), pregnancy and postpartum status (Da Costa, Dritsa, Verreault, Balaa, & Kudzman, 2010), health disparities (see Chapter 15, Racial/Ethnic Health Disparities and Sleep Disorders), culture (Mindell, 2010), age (Robinson & Waters, 2008), presence of medical comorbidity (Redeker, 2008), pain (Turk & Cohen, 2010), and other comorbid sleep disorders (Chasens, Twerski, Yang, & Umlauf, 2010). These variables need to be considered in any assessment of sleep, given their powerful influence on the lived experience and the underlying sleep physiology.

Sleep disturbance, especially short sleep duration, is a risk factor for suicidal behavior (Gangswisch et al., 2010; Goodwin & Marusic, 2008). This association is strongly influenced by the presence of a mood disorder, panic attacks or a substance abuse disorder (Goodwin & Marusic, 2008). However, developmental stage also played a role in sleep-disturbed adolescents with depression. Data from Mack (2009) indicate that adolescent suicide completers have higher rates of overall sleep disturbance, insomnia, and hypersomnia, compared with controls within both the last week before the suicide and within the current episode of illness. Given these findings, clinical assessment of sleep patterns may provide an impressive benchmark for mortality risk in this age group, and merits close clinical attention.

Across studies, disturbed sleep and suicidal ideation/behaviors are closely associated, and nightmares seem to increase the risk (Bernet & Joiner, 2007). Although data are limited to a case-report, the finding that treatment of obstructive sleep apnea (OSA) led to rapid resolution of severe suicidal ideation (Krahn, Miller, & Bergstrom, 2008) suggests the commonality of risk for suicide across different sleep disorders. Although further research is needed into the physiological mechanisms for these relationships, these findings suggest that identification and treatment of sleep disorders may play a powerful role in reducing the risk of suicide.

Pathophysiological Explanations for the Relationships Between Depression and Sleep

There are several possible pathophysiological explanations for the relationships between psychiatric disorders and sleep disorders. For example, both sleep disorders and depression are associated with dysregulation in the monoaminergic (dopamine, norepinephrine, serotonin) and cholinergic systems in the central nervous system, particularly an increase in cholinergic activity as compared to monoaminergic activity. Neurochemicals from these classes share a strong relationship in both mood and sleep. (See Chapter 1, Physiological and Behavioral Aspects of Sleep.) Alternative hypotheses include disturbances in the homeostatic drive (manifested in changes in slow-wave sleep), physiologic hyperarousal associated with sympathetic activation, and altered circadian rhythms as seen in phase shifts or shifts in REM architecture. (See Chapter 10, Circadian Rhythm Disorders.) The question of whether the documented changes in sleep are state-dependent or markers of vulnerability for depression remains incompletely answered and is the focus of ongoing research.

Sleep-Related Considerations in Treatment of Unipolar Depression

Many of the medications used to treat depressive disorders target monoaminergic systems. Therefore, they may also address associated sleep changes (Carskadon & Dement, 2000). While all of the antidepressants are equally effective in the treatment of depression, they are

Table 12.3 ■ *Medications Used in the Treatment of Specific Psychiatric Disorders*

Antidepressants (Depression, Anxiety Disorders: PTSD, Panic, OCD, GAD)

- Selective serotonin reuptake inhibitors (fluoxetine, sertraline, paroxetine, citalopram, escitalopram, fluvoxamine, and others)
- Serotonin/norepinephrine reuptake inhibitors (venlafaxine, desvenlafaxine, duloxetine, and others)
- Tricyclic antidepressants (amitriptyline, amoxapine, clomipramine, desipramine, doxepin, imipramine, nortriptyline, protriptyline, trimipramine, and others)
- Atypical antidepressants (burpropion, mirtazapine, trazodone)
- Monoamine oxidase inhibitors (tranylcypromine, phenelzine, selegiline, and others). This class of antidepressants is not included in the discussion within this chapter due to the extremely rare use of these drugs in clinical practice.

Mood Stabilizers (Bipolar Spectrum Disorders)

- Lithium carbonate
- Valproate
- Lamotrigine
- Gabapentin
- Carbemazepine
- Topiramate
- Others

Anxiolytics (Anxiety Disorders: PTSD, Panic, OCD, GAD, Off-Label Hypnotics)

- Benzodiazepines (lorazepam, diazepam, clonazepam, alprazolam, others)
- Azaspirones (buspirone)
- Antihistamines (diphenhydramine, hydroxyzine, others)
- Beta blockers (propranolol, metoprolol, others)

Antipsychotics (Schizophrenia, Bipolar Disorder, Select Off-Label Hypnotics)

- Conventional or first generation (haloperidol, chlorpromazine, fluphenazine, loxapine, perphenazine, and others)
- Second generation (clozapine, quetiapine, rispiradone, olanzapine, and others)

Stimulants/Nonstimulants (ADHD, Augmenting Agents in Depression)

- Amphetamines (methylphenidate, dextroamphetamine, dexmethylphenidate, and others)
- Nonstimulants (atomoxetine)

Hypnotics (Sleep Pattern Disturbance Across the Disorders)

- Nonbenzodiazepines (zolpidem, zopiclone, eszopiclone)
- Benzodiazepines (temazepam, triazolam, flourazepam, and others)
- Melatonin receptor agonist (ramelteon)
- Benzodiazepines are routinely avoided in alcoholism due to issues of cross dependence; Off-label use of medications such as trazodone is common.

Source: *Essential Psychopharmacology: The Prescriber's Guide* by S. M. Stahl, 2009, Cambridge, UK: Cambridge University Press.
Abbreviations: ADHD, attention deficit hyperactivity disorder; GAD, generalized anxiety disorder; OCD, obsessive compulsive disorder; PTSD, posttraumatic stress disorder.

not uniformly sleep-friendly (see Tables 12.3 and 12.4). Consequently, FDA-approved (e.g., benzodiazepine receptor agonists) (see Chapter 6, Insomnia) or off-label hypnotic medications are often co-prescribed. The most commonly used off-label examples are trazodone and tricyclic antidepressants (TCA). Potent second generation antipsychotic medications (e.g., quetiapine) are also used because of their sedative effects (Philip, Mello, Carpenter, Tyrka, & Price, 2008), but use of these drugs presents high risk for metabolic dysregulation.

Psychotropic medications used to treat sleep disturbance share a common sleep-promoting mechanism: antagonism of serotonin receptor subtype 2A (5-HT2A), an important neurophysiologic regulator of sleep. Of the antidepressants with negative effects on sleep architecture, SSRIs are the most common culprits. SSRIs stimulate 5-HT2A, leading to arousal and often fitful sleep.

Table 12.4 ■ *Effects of Psychotropic Medications and Alcohol on Sleep Architecture*

Antidepressants

- Selective serotonin reuptake inhibitors: REM suppression and prolonged REM latency; increased awakenings after sleep onset; increased sleep latency; reduced sleep efficiency
- Serotonin norepinephrine reuptake inhibitors: REM suppression and prolonged REM latency; decreased sleep efficiency; increased wake after sleep onset
- Tricyclics: Potent REM suppression with prolongation of REM latency; promotion of sleep initiation; improved sleep efficiency
- Bupropion: No consistent effect on REM sleep; few alterations in sleep architecture have been reported
- Serotonin two receptor antagonists/reuptake inhibitors (nefazodone, trazodone): Improved sleep efficiency; increased Stages 3 and 4 NREM sleep
- Mirtazapine: Increased total sleep time; decreased sleep latency; decreased Stage 1 NREM sleep

Mood Stabilizers

- Lithium carbonate: Sedation; prolonged REM latency; suppressed REM sleep time; increases in Stages 3 and 4 NREM
- Valproate: Sedation; increased total sleep time; decreased number of awakenings after sleep onset
- Carbamazepine: Sedation; decreased sleep latency; increased total sleep time
- No consistent data available on the other anticonvulsants

Anxiolytics

- Benzodiazepines: Reduced sleep latency; no consistent effect on REM; suppression of Stages 3 and 4 NREM
- Buspirone: REM suppression

Antipsychotics

- Conventional: Decreased REM latency; increased REM density; decreased Stages 3 and 4 NREM
- Second generation: Improved sleep continuity; increased REM latency; increased Stage 3 NREM sleep

Stimulants

- Amphetamines: REM suppression; reduced sleep efficiency; increased wakefulness after sleep onset
- Modafinil: No significant change in sleep documented

Hypnotics

- Benzodiazepines: Reduced sleep latency; no consistent effect on REM; suppression of Stages 3 and 4 NREM; decreased wake after sleep onset
- Nonbenzodiazepines: No effects on Stages 3 and 4 NREM; reduced sleep latency; decreased wake after sleep onset
- Ramelteon: No significant effects on sleep architecture

Alcohol

- With ingestion, shortened sleep latency; enhancement of NREM initially after sleep onset; reduced REM in the first hours of sleep
- With metabolism of alcohol, shallow and disrupted sleep; increased REM; rebound wakefulness in the second half of the night
- Disturbances in sleep often continue long after detoxification has occurred

Sources: *ACCP 2008 Sleep Medicine Board Review* (pp. 71–87), by R. Benca, 2008, Chicago: AACP Sleep Institute. "Effects of Psychiatric Medications on Sleep and Sleep Disorders," by N. A. DeMartinis and A. Winokur, 2007, *CNS & Neurological Disorders—Drug Targets, 6*, 17–29. "The Effects of Antidepressants on Sleep: A Review," by J. T. Gursky and L. E. Krahn, 2000, *Harvard Review of Psychiatry, 8*, pp. 298–306. "Antidepressants and Sleep: A Review," by J. M. Holshoe, 2009, *Perspectives in Psychiatric Care, 43*(3), 191–197. "Sleep Disturbances and Depression: A Challenge for Antidepressants," by R. W. Lam, 2006, *International Clinical Psychopharmacology, 21*(S1), S25–S29. *Principles and Practice of Sleep Medicine* (pp. 441–462), by P. K. Schweitzer, 2000, Philadelphia: WB Saunders. Wilson, Bailey, Rich, et al., 2005.

Through a different mechanism, SSRIs may also unmask restless legs and periodic limb movements during sleep (Picchietti & Winkelman, 2005) and contribute to arousal and poor sleep continuity. (See Chapter 8, Movement Disorders.) Iatrogenic sleep disturbance is not limited to the SSRIs (see Table 12.4) but may also result from other types of antidepressants such as the serotonin norepinephrine reuptake inhibitors (e.g., duloxetine and others). Stimulants that are used to treat ADHD or as augmenting agents to antidepressants also create disturbances in sleep quality. However, antidepressants, including SSRIs, suppress REM sleep and reverse early

REM onset. This often leads to abatement of depressive symptoms.

Agomelatine, the newest antidepressant agent, currently available only in Europe, is a melatonergic MT1/MT2 receptor agonist with serotonin 5-HT2C receptor antagonist activity. It is the first antidepressant that specifically targets the melatonin receptors that are critical to sleep and may regularize circadian rhythmicity. Clinical trials demonstrated the efficacy and tolerability of agomelatine compared with placebo and trials of venlafaxine and sertraline (Kennedy & Rizvi, 2010). Gorwood (2010) suggests that the antidepressant activity of agomelatine results from the resynchronization of disturbed circadian rhythms in many depressed patients. The author notes that treatment with agomelatine has significantly improved the amplitude of the circadian rest-activity/sleep-wake cycle and decreased depression and anxiety symptoms when compared with sertraline, a common SSRI antidepressant. Further clinical trials are underway in the United States.

Nonpharmacologic Interventions in Unipolar Depression

Pflug and Tolle (1971) documented the extraordinary finding that a night of total sleep deprivation in those with major depression led to remission of symptoms, and the depression recurred with recovery sleep. This work was the impetus for a significant body of research that explored the use of other forms of sleep deprivation as a treatment for depression. Examples included partial sleep deprivation (Brunner, Dijk, Tobler, & Borbely, 1990) in which depressed individuals were deprived of sleep either at the beginning or end of the usual sleep period and rapid eye movement sleep deprivation in which patients were awakened every time they entered REM sleep. Although these interventions were effective treating depression (Beersma, Dijk, Blok et al., 1990), they were not feasible. Although they are not currently used in clinical practice, they have contributed to the understanding of the shared pathophysiology of sleep and depression.

Light therapy, first described by Rosenthal and colleagues in 1984, involves the application of bright light to entrain circadian rhythm and reduce melatonin levels and is effective in the treatment of seasonal depression (Sher, 2003) and shows promise as a treatment for nonseasonal depression (McEnany & Lee, 2005; Tuunainen, Kripke, & Endo, 2004). (See Chapter 1, Physiological and Behavioral Aspects of Sleep and Chapter 10, Circadian Rhythm Disorders.) Table 12.5 addresses some of the fundamental

Table 12.5 ■ *Information on Light Therapy*

- Light therapy involves exposure to intense bright light for specified amounts of time and at specific times during the day; for example, seasonal depression is treated with morning light and phase advanced sleep (see Chapter 10, Circadian Rhythm Disorders) is treated with evening light. Light taken before bedtime may cause insomnia.
- The light quality that is used approximates the spectrum of daylight though without the ultraviolet bands in the spectrum.
- Duration of exposure is individualized to produce desired effects. In seasonal depression, it is not uncommon to see exposure time increase as the days shorten.
- Light intensity is determined by proximity to the light source itself. Light is measured in *lux* and 10,000 lux (similar to late morning light) is commonly used. Sitting 2 feet from the source may provide 10,000 lux but sitting 4 feet from the source will deliver 5,000 lux.
- Various light equipment is available and includes standard light boxes, light visors, and dawn/dusk simulators, which gradually produce or reduce light, similar to what would be seen with environmental light.
- Light works directly by suppressing melatonin (produced by the pineal in darkness). In doing so, light has a powerful impact on the master clock for the body, located in the suprachiasmatic nucleus in front of the hypothalamus and above the optic chiasm.
- Side effects of light therapy include eye strain, headache, irritability, and may precipitate hypomania in bipolar illness.
- Light therapy should be used in consultation with an experienced clinician and should be used cautiously in people with preexisting eye diseases such as macular degeneration, retinitis pigmentosa, or diabetic retinopathy.
- Light is commonly used adjunctively with medications.

Source: Society for Light Treatment and Biological Rhythms (http://www.sltbr.org/sadfaq.htm#b2).

Figure 12.1 ■ Dawn simulator alarm clock.
Photograph provided courtesy of BioBrite, Inc., Bethesda, MD.

Figure 12.2 ■ Light therapy visor.
Photograph provided courtesy of BioBrite, Inc., Bethesda, MD.

Figure 12.3 ■ Light box.
Photograph provided courtesy of BioBrite, Inc., Bethesda, MD.

information on the use of light therapy in depression (see Figures 12.1–12.3).

A related area of research is the finding of vitamin D deficiency in patients with seasonal depression, but it is not yet clear if the relationship exists in both seasonal and nonseasonal depression. Humble (2010) discusses the role of vitamin D and its relationship to mental health. The active vitamin D hormone is calcitriol that impacts a number of the neurochemicals associated with the regulation of mood. An emerging body of evidence supports the role of vitamin D in mood modulation in light of vitamin D deficiency. Vitamin D deficiency seems to be directly related to depressive disorders, though the mechanism is not clear (Shipowick, Moore, Corbett, & Bindler, 2009). Significant improvement in depression with exogenous supplementation of vitamin D has been documented (Berk et al., 2007). Research is ongoing into the use of vitamin D supplementation (Leard-Hansson & Guttmacher, 2008) and light therapy as treatments for seasonal depression and sleep conditions is underway.

Bipolar Spectrum Disorders

Bipolar spectrum disorders represent a continuum of serious and life-threatening diseases characterized by variability of symptom presentation, ranging from severe and incapacitating depression to disorganized and frenetic mania. Bipolar illness affects 5.7 million Americans or 2.6% of the U.S. population over 18 years of age. The mean age of onset is 25 years (Source: http://www.nimh.nih.gov/health/publications/). Current DSM IV-TR diagnostic nomenclature (APA, 2000) classifies bipolar spectrum disorders as follows: bipolar 1, bipolar 2, cyclothymic disorder, and bipolar disorder not otherwise specified (APA, 2000). Bipolar 1 is the most severe and disabling form; cyclothymic disorder is the least severe. Bipolar spectrum disorders are associated with disability, incomplete inter-episode recovery, and decline in cognition (Giglio, Magalhaes, Kapczinski, Walz, & Kapczinski, 2010).

Characteristics of Disturbed Sleep in Bipolar Disorders

Long recognized as a result of circadian and homeostatic influences, sleep dysregulation is a hallmark symptom of the onset of a new episode of bipolar illness, an index of symptom control over time, and an important marker of symptom recurrence. Sleep disturbance during acute illness and between episodes is a critical feature of the bipolar spectrum (Dijk & Lockley, 2002; Richardson, 2005).

Sleep in Mania Changes in sleep are hallmarks of the onset of an episode of mania and are evident in a sharp decrease in the need for sleep and severely shortened sleep duration.

Sleep in Bipolar Depression While the similarities in sleep architecture among those with unipolar depression and bipolar depression are striking, hypersomnolence, and early morning awakenings may be more characteristic of bipolar depression (Plante & Winkelman, 2008).

Pharmacologic Interventions in Bipolar Disorder

The mainstays of treatment for bipolar disorder are mood stabilizers and conventional and second-generation antipsychotic agents. Due to the potential for mania, antidepressants are contraindicated in the treatment of bipolar 1 disorder, but are used in the treatment of cyclothymic and bipolar 2 disorders (El-Mallakh, Elmaadawi, Loganathan, Lohano, & Gao, 2010).

Nonpharmacologic Interventions in Bipolar Disorder

Psychotherapy and group therapy are the primary and standard interventions for bipolar disorders (APA, 2007a). Education for both the patient and family focused on risk reduction and relapse prevention is critical.

Recurrent depression, cyclical mood variation, or suboptimal treatment of either unipolar or bipolar depression often lead to chronic insomnia. Episodes of acute illness precipitate acute changes in sleep, followed by patterns of maladaptive behavior that perpetuate the sleep disturbance. By the time a person with a mood disorder receives appropriate assessment and intervention for sleep disturbances, the insomnia is often chronic. For this reason, cognitive behavioral therapy for insomnia (CBT-I) is an efficacious treatment for insomnia in bipolar illness, with moderate to large effects (Cohen's *d,* range 0.35–2.2) (Smith, Huang, & Manber, 2005).

Assessment and Treatment of Sleep in People with Mood Disorders

Because of the close interrelationship between sleep disorders and mood disorders, sleep assessment is an important component of nursing care. Symptoms of insomnia are the most prevalent in psychiatric disorders. Principles of sleep assessment are described in Chapter 5 and focused assessment for insomnia is discussed in Chapter 6. Assessment in patients with psychiatric disorders should emphasize the role of psychiatric symptoms, as well as the effects of psychotropic medications, including SSRIs. Since sleep complaints are an important component of exacerbation of bipolar disorders, nurses and patients can use these cues (e.g., decreased sleep duration, perceived decreased sleep need, or conversely increased sleep) to determine the need for changes or accelerated behavioral or pharmacological treatment. Assessment should also include standard approaches to assessment for the presence of sleep-disordered breathing, parasomnias, and movement disorders (see Chapter 7, Sleep-Related Breathing Disorders, and Chapter 8, Sleep-Related Movement Disorders and Parasomnias.)

Stabilization of sleep is essential during acute illness and is often accomplished with the use of sedating medications. Benzodiazepine receptor agonists are used across the mood disorders. In bipolar illness, sedating antipsychotic medications such as olanzapine are used because they not only facilitate sleep, but are effective in abating the symptoms of both mania and bipolar depression with psychotic disorders. Sedating antidepressants, such as trazodone, are frequently used for patients with unipolar depression. Trazadone improves sleep, while providing an

augmenting influence to the primary antidepressant treatment. Trazodone is rarely used as a first line treatment for depression because of the intolerable sedation that is associated with its use. Sleep hygiene and stimulus control are very helpful once acute symptoms stabilize. (See Chapter 6, Insomnia.) As the condition stabilizes, the treatment plan may expand to include CBT-I. An important component of CBT-I is sleep restriction therapy. However, this should be used cautiously and only by experienced clinicians because sleep restriction, a key component of CBT-I, may be a trigger for mania. Complementary and alternative therapies (see Chapter 15, Racial/Ethnic Health Disparities and Sleep, and Chapter 6, Insomnia) may be useful, but evidence of their efficacy is limited.

Patient teaching should focus on adherence to a sleep-specific regimen and may be enhanced through explanation of the shared relationship between the psychiatric disorders and sleep; sleep can be used as a barometer for monitoring symptoms. Simple explanations about the role of homeostatic and circadian regulation (see Chapter 1, Physiological and Behavioral Aspects of Sleep) may assist the patient and family member to understand the synergy between sleep and their psychiatric disorder, their need for sleep, the importance of sleep patterning, and the principles of sleep hygiene. Regularizing sleep schedules is particularly important for patients with bipolar disorder. Teaching pattern recognition provides a foundation for risk reduction and relapse prevention. Routinizing the use of sleep-wake diaries and other pragmatic tools will help to objectify the experience of sleep and wakefulness while more fully engaging patients in their care.

SLEEP AND ANXIETY DISORDERS

The most common forms of anxiety disorders are panic disorder, posttraumatic stress disorder (PTSD), obsessive compulsive disorder, and generalized anxiety disorder (GAD). However, anxiety may be a prominent feature of other disorders such as major depressive disorder or adjustment disorders that do not meet specific criteria for separate anxiety diagnoses. Anxiety can be a normal response to perceived or real threats to safety and is ubiquitous in the lived experience of humans. However, pathological anxiety presents with severe symptoms that impair social and occupational functioning. Anxiety has been documented to be a predictor (Vahtera et al., 2007) and a result of insomnia (Neckelmann, Mykletun, & Dahl, 2007) in vulnerable individuals.

Panic Disorder

Panic disorder is characterized by recurrent and precipitous physiologically and psychologically based acute symptoms. While activation of the sympathetic nervous system and serotonin explain some of the biology of panic disorder, investigators are now exploring the role of hypocretins (chemicals that are critical to wakefulness and arousal) and psychiatric disorders including panic (Bonnavion & Lecea, 2010). This exciting research may explain much of the association between psychiatric disorders and sleep and will have direct implications for practice.

Panic attacks occur in the daytime, but also during sleep and awaken the individual in the midst of an attack (Craske, 2005). Although panic attacks have been well studied, there has been little focus on nocturnal panic attacks that may represent a distinct subtype associated with agoraphobia or respiratory events occurring OSA hypopnea syndrome (OSAS) (Nardi, 2009; Freire et al., 2007). For example, sleep-disordered breathing may present exclusively as nocturnal panic (Freire et al., 2007). Clearly, nocturnal panic and the directionality of its relationship with sleep disorders require further investigation. Specifically, there is a need to address the question: Is there a subtype of panic disorder that is characterized by nocturnal episodes of panic in the absence of sleep-disordered breathing or is there a shared relationship that has not yet been fully explained?

Pharmacological Interventions in Panic Disorder

Standard practice includes the use of benzodiazepines to prevent or abort an attack. These are used while concurrently with a medication such as an SSRI, while awaiting a therapeutic response from the SSRI that often takes 3–4 weeks.

Nonpharmacologic Interventions in Panic Disorder

Cognitive behavioral therapy (including CBT-I) or panic-focused psychodynamic therapy are the mainstays of treatment for panic disorders (APA, 2007d). Nursing intervention involves helping the person recognize that panic is a symptom of the psychiatric illness rather than the harbinger of a catastrophic event, such as a myocardial infarction (a common perception among patients with panic attacks). Risk reduction strategies including recognition of triggers for panic are incorporated into a plan for relapse prevention. Facilitating skill acquisition in relaxation techniques, self soothing, and stress reduction are all central to the plan of care. While these skills are imperative in the treatment of panic itself, they serve an important function in sleep enhancement. Perceptual distortions related to sleep, for example, *I am not going to be able to sleep tonight* require interventions that cast doubt on the veracity of the perception, similar to the myocardial infarction example noted above. CBT-I becomes a critical dimension in the acquisition of these skills.

Posttraumatic Stress Disorder

PTSD is one of the most severe anxiety disorders, and its course is often chronic. The overall impact on functional capacity and clinical outcome is complex and influenced by a number of factors including premorbid functioning, substance abuse, and other circumstances. Persons who have a*cute stress disorder* (ASD) have similar symptoms within days of the exposure to trauma, and the symptoms resolve with appropriate treatment in a short period of time. Symptoms of PTSD may not appear for 3 months or more, and its course and treatment are often protracted due to the severity of the illness, comorbid substance abuse, and associated disability.

As documented in the work of Mellman, Kulick-Bell, Ashlock and colleagues (1995), PTSD is associated with changes in sleep architecture (see Table 12.2). More recently, researchers questioned the temporal relationships between the timing of the exposure of the trauma to the onset of symptoms and suggested that this variability points to different neurobiological mechanisms of the disorder and its impact on sleep (Babson & Feldner, 2010). One question that these differences raise is whether or not there are possible subtypes of the disorder based on neurobiological expression related to the symptom cluster.

Pharmacologic Interventions in PTSD

Pharmacologic treatment of persons with PTSD is significantly broader than for other anxiety disorders and usually includes antidepressant, anxiolytic, sedative hypnotic (both off label and indicated) as well as antipsychotics, if needed (APA, 2004a). However, treatment of PTSD is often complicated by substance abuse, a maladaptive attempt at symptom relief.

Nonpharmacologic Interventions in PTSD

Cognitive–behavioral therapy is the mainstay of treatment, given the severity of perceptual distortions driving patterns of thoughts, feelings, and behavior. Group therapy, psychological debriefing, eye movement desensitization and reprocessing (EMDR), and other treatments may also be useful (APA, 2004a) for treatment of the core symptoms of PTSD, but the fundamental principles of CBT used to treat PTSD are very similar to CBT-I, and offer the clinician an opportunity to incorporate CBT-I into the standard treatment for the patient.

Obsessive Compulsive Disorder

Obsessive compulsive disorder is characterized by pathological patterns of obsessive thinking and behavioral responses (compulsions) whose function is the alleviation of the anxiety generated by the obsessive thoughts. Approximately, 3.3 million American adults aged 18–54 years have OCD. This represents approximately 2.3% of the population (Source: http://understanding_ocd.tripod.com/ocd_facts_statistics.html).

There are relatively few studies of the associations of sleep and OCD and many inconsistencies

in the findings of extant studies. This may be due to the fact that patients often have comorbid conditions, such as depression (Fineberg, Fourie, Gale, & Sivakumaran, 2005). Therefore, some of the sleep disturbance experienced by people with OCD may be related to comorbid depression. A significant challenge with the sleep of those diagnosed with OCD is that sleep itself may become the focus of obsessive thoughts, leading to heightened anxiety—clearly an impediment to sleep.

Pharmacologic Interventions in OCD

As with the other anxiety diagnoses, the mainstay of treatment includes antidepressant medications that target the biological foundation of the symptom cluster. Other medications such as the benzodiazepines or less often, the second generation antipsychotics (SGA), are used as adjunctive approaches to target symptoms that have been inadequately treated with the antidepressants alone.

Nonpharmacologic Interventions in OCD

Similar to other anxiety disorders, the mainstay of treatment is cognitive–behavioral therapy, followed by psychodynamic therapy (APA, 2007d). Interestingly, the incorporation of CBT-I into routine psychiatric care across many of the anxiety disorders has not been studied. Anecdotally, psychiatric clinicians who are using this approach generally find it to be successful. Clearly there is an indication for research in this area.

Generalized Anxiety Disorder

GAD is characterized by a long-standing pattern of anxiety and worry that includes physiological and psychological symptom clusters. These result in impairment in social or occupational functioning over the course of at least 6 months. Approximately 6.8 million American adults, or about 3.1% of people aged 18 and over, have GAD in a given year. Although the disorder can begin at any age, the median age of onset is 31 years (Source: http://www.nimh.nih.gov/health/publications/the-numbers-count-mental-disorders-in-america/index.shtml#GAD).

Several studies documented that patients with GAD have sleep onset and sleep maintenance insomnia (Monti & Monti, 2000) that are often associated with worries about sleep quality and duration. (See Chapter 6, Insomnia.) The associations with insomnia may be explained by high levels of physiologic and psychological arousal.

Pharmacologic Interventions in GAD

While the American Psychiatric Association has not published a Practice Guideline specific to GAD, the interventions are similar to those used in other anxiety disorders, namely cognitive–behavioral therapy, antidepressants, and nonbenzodiazepine medications such as buspirone. Given the chronic course of GAD and the effectiveness of the other medications available, benzodiazepines are often avoided.

Nonpharmacologic Interventions in GAD

Cognitive–behavioral and psychodynamic therapies are commonly used to treat GAD. Long-term treatment is usually indicated, because of the chronic nature of GAD. Given that sleep disturbance is one of the diagnostic criteria for GAD, sleep-focused intervention is critical. As with the other anxiety disorders, the use of CBT-I is appropriate but has not yet been applied to practice in the treatment of people with GAD.

Assessment and Treatment of Sleep in Patients with Anxiety Disorders

Assessment of people who have anxiety disorders is very similar to the approach for those with mood disorders, discussed earlier. Among those with anxiety disorders, close evaluation of iatrogenic sleep pattern disturbances is important, given the common use of medications that are known to potentially be sleep disruptive, for example, the SSRIs.

A primary dimension of treatment focuses on addressing perceptual distortions (cognitive therapy) related to sleep because patients are often anxious about sleep in general; the best predictor of a bad night of sleep is expectation of a bad night of sleep. Sleep hygiene and stimulus control are essential, but relaxation is the most useful in addressing the underlying physiological and psychological arousal. These interventions are components of CBT-I. (See Chapter 6, Insomnia.) CBT is endorsed by the American Psychiatric Association as a primary treatment across the anxiety disorders (APA, 2004, 2007), but none of the psychiatric practice parameters for anxiety disorders discuss the use of CBT-I to address insomnia in people across these diagnoses. It is possible that improving insomnia among patients with anxiety may also contribute to improved outcomes.

The care of people with PTSD presents a set of special circumstances. In addition to addressing insomnia, it is important to address the nature of the trauma and its relevance to sleep. In some traumatic circumstances, sleep may present a period of vulnerability for additional trauma. There may not be adequate assurance of safety in the place available for sleep or the trauma may have occurred in the course of a sleep episode. The symptoms of PTSD in sleep (e.g., nightmares) are experienced in ways that closely approximate the trauma itself (Caldwell & Redeker, 2005). Prevention of retraumatizing events in sleep is crucial. Assuring a sleep environment that is perceived to be safe is the foundation of risk reduction. This intervention may be difficult, especially if the trauma occurred during sleep or in the general place where the person sleeps. The use of hypnotic medications (see Table 12.3) is necessary to reduce physiological arousal. Because of the nature of PTSD and the severity of symptoms common to this disorder, hypnotic medication may be used throughout the entire treatment period. Sleep-related interventions for people with PTSD accompany other interventions focused on the consequences of the trauma, such as group or individual therapy, desensitization, and medications.

Pharmacologic and cognitive approaches to sleep are important in people with panic disorder, especially those with nocturnal panic attacks. The use of benzodiazepines in the acute phase of GAD treatment and stress reduction and relaxation that are overall components of treatment are also beneficial for sleep.

Because of the long-term nature of GAD and the challenging perceptual distortions in OCD, cognitive approaches to insomnia are important. Of course, clinical outcomes of treatment will be enhanced with control of characteristic baseline symptom clusters (see Table 12.1). Sleep-specific plans of care that build on these symptoms are likely to achieve greater success. This trend is evidenced in improved clinical outcomes. Because sleep symptoms are part of the diagnostic features of these disorders, abatement of the sleep-related symptom demonstrates healing. Complementary measures such as yoga have shown promise in facilitating symptom control in conditions such as OCD may also improve sleep in these individuals (Shannahoff-Khalsa, 2004) and should be routinely considered in the plan of care.

PSYCHOTIC DISORDERS AND SLEEP

Schizophrenia

Schizophrenia is only one of a number of psychotic disorders listed in the DSM IV-TR (APA, 2000), but it is considered to be the most debilitating and severe. Approximately, 2.4 million American adults, or about 1.1% of the population aged 18 and older in a given year, have schizophrenia. (Source: http://www.nimh.nih.gov/health/publications/the-numbers-count-mental-disorders-in-america/index), Schizophrenia affects men and women with equal frequency. The disease often appears in late adolescence or early adulthood and often corresponds with increasing demands for social and academic or work-related performance.

Schizophrenia is considered the prototypic *thought disorder* because there are significant impairments in perception and thinking that are the foundation for the symptom cluster of the disease. One of the challenges in under-

standing sleep patterns in persons diagnosed with schizophrenia is the recognition that it is not a single disease. There are subtypes such as schizoaffective disorder and others (APA, 2000). The efficacy of various medications with diverse mechanisms of action has facilitated a better understanding of the pathophysiology of the disease itself.

As in other psychiatric illnesses, dysregulation of regular sleep patterns is a forerunner of relapse. This suggests that regularization of circadian rhythms and entrainment to regular daily schedules (e.g., meals, social activities) may improve symptoms and prevent relapse. Circadian rhythmicity is closely aligned with neurochemical function as reflected in the sleep-wake cycle. An interesting example of this relationship is seen in the work of Mendoza, Clesse, Pevet, and colleagues (2010), which demonstrates entrainment of the circadian clock with food. Neurochemical indices studied in this work included dopamine and orexins. While this work utilizes a mammalian animal model, application of the findings for humans seems compelling. In an acute psychotic state, sleep is likely to be short and vary as the individual struggles with the frightening experiences of hallucinations or beliefs that may include paranoid ideation. During more stable periods, sleep may have a more regular pattern and normal duration. However, in the absence of regular scheduling of activities, sleep hygiene practices may deteriorate and perpetuate sleep disturbance.

Pharmacologic Interventions in Schizophrenia

Evidence-based approaches to assessment and treatment of people who have schizophrenia are available (APA, 2004b). The first generation antipsychotic medications used to treat symptoms of schizophrenia worked by antagonizing (blocking) the dopamine subtype 2 (D2) receptor in parts of the brain that regulate cognition, emotion, movement, and prolactin regulation. Pharmacologic antagonism of D2 receptors significantly reduces or eliminates positive symptoms but may worsen negative symptoms of the disease, and these drugs contribute to sleep-related movement disorders. (See Chapter 8, Movement Disorders.) These drugs (e.g., haloperidol) may also contribute to disturbances in circadian rhythmicity of sleep and wakefulness (Staedt, Hauser, Gudlowski and Stoppe (2010).

SGAs improve both positive and negative symptoms. However, excessive daytime sleepiness (EDS) is often an adverse effect (Hawley et al., 2010). Iatrogenic effects, including significant weight gain, type 2 diabetes, hyperlipidemia, and metabolic syndrome are well documented (Edward, Rasmussen, Munro, 2010; Smith et al., 2010). The SGAs that exert antagonism of the D2 receptor may yield restless legs because of the impact of this mechanism of action in the dopaminergic nigrostriatal track.

The weight gain resulting from SGA medications is likely to increase the risk for OSA (Cohrs, 2008). (See Chapter 7, Sleep-Related Breathing Disorders.) The sedative effects of these drugs may also contribute to the development of OSA by reducing the tone of the upper motor airway musculature (Fleischman, Ananthamoorthy, Greenberg, Harvey, & Merlino, 2008; Rishi et al., 2010). Increases in central sleep apnea have been noted with use of aripiprazole (Kohen & Sarcevic, 2009), and SGAs may also lead to restless legs syndrome (RLS) and periodic limb movement disorder (PLMD) (Ancoli-Israel et al., 1999) through antagonism of D2 receptors in the nigrostriatal track, a brain area responsible for many dimensions of movement. Although these findings need to be replicated, they underscore the need for vigilance in assessing and treating the sleep-related effects of SGAs.

Nonpharmacologic Interventions in Schizophrenia

Because of differences in patient response associated with positive and negative symptoms of schizophrenia (see Table 12.1), approaches to treatment may vary. In an acute phase, cognitive approaches are inappropriate. The primary focus for sleep is facilitating sleep hygiene and structuring effective self-care measures to promote symptom control. In practice, this means having to intervene in the basic areas

of self-care that the patient is unable to accomplish for her/himself. At times, this may include something as basic as assisting with feeding. With sleep, it involves very structured approaches to assure adequate sleep, balanced with a schedule of activities during the day.

Assessment and Treatment of Sleep Disorders in People with Psychotic Disorders

Assessing and treating disturbances in sleep and wakefulness in those diagnosed with schizophrenia is complex. Structured assessment of sleep patterns focuses on sleep duration, fragmentation, and the patterning of sleep over the course of the 24-hour day. Responses to medications and the core symptoms of the disease (positive or negative presentations) and their associations with sleep should be addressed. As with depressive disorders, changes in patterns of sleep and wake may accompany exacerbation or improvement in the condition and signal the need for changes in treatment. Because of the metabolic effects of SGAS on weight gain, OSA, and the potential for movement disorders, signs, and symptoms of these disorders should be carefully monitored. (See Chapter 5, Conducting a Sleep Assessment; Chapter 7, Sleep-Related Breathing Disorders; and Chapter 8, Sleep-Related Movement Disorders and Parasomnias.) Patients with signs and symptoms suggestive of these disorders should be referred for specialized sleep disorders evaluation and possible treatment.

For those with positive symptom presentations, perceptual distortions, hallucinations, and delusions may make it difficult to intervene with sleep and wakefulness. Medications are the first line of treatment (Wolff, Talley, & Smith, 2008) for acute symptom control, coupled with behavioral measures to restore self-care capacity in the areas of eating, hygiene, adequate rest and activity and socialization. For patients with negative symptom presentations, avoidance of social interaction, lack of communication, and significant cognitive impairment challenges the sleep-related plan of care. In acute states of illness, patients have difficulty meeting basic needs and determining measures to meet the needs. Consequently, the patient frequently ignores self-care needs and nursing intervention becomes essential to meet these needs. In these circumstances, the approach to sleep needs to be basic and structured to assure satisfactory sleep hygiene and wakefulness during the day. This involves assuring that the patient remains awake during the day, is exposed to daytime environments, and is helped both behaviorally and with medications to sleep at appropriate times. People with this form of the disease often lose connection with the common social and environmental cues of daytime and night, complicating the clinical picture further. Exposure to natural sunlight during the day, structuring routines to incorporate activities that facilitate wakefulness, for example, walking, engagement with basic social interaction, listening to music, eating meals at regular times and facilitating bedtime routines, and sleep at night all are important measures to realign an appropriate day/night routine.

SUBSTANCE ABUSE AND SLEEP

Virtually all psychoactive substances have an impact on sleep because of the role that these drugs play in altering neurochemicals associated with sleep and wakefulness. Many of the drugs of abuse are either central nervous system depressants or stimulants and as such have a potentially powerful impact on sleep and wake. Interestingly, after withdrawing from the substance, sleep often continues to be disturbed, and this reality is multifaceted. Some of the effects are purely neurochemical while use of the substance may behaviorally shape sleep and wake. For example, someone who drinks a significant amount of alcohol or smokes marijuana before bed every night easily falls asleep due to the impact of the drug on sleep induction. But in the absence of the substance, the expectation for easy sleep onset may be present without the neurochemical impact of the drug. Sleep onset difficulties may then become a source of anxiety and sleep-onset insomnia.

Given the large number of substance-related diagnoses, only alcoholism will be reviewed here. However, resources related to substance abuse and sleep are readily available from the National Center on Sleep Disorders Research, the National Institute on Alcohol Abuse and Alcoholism, and the National Institute on Drug Abuse and the links to these sites are located in Table 12.6, *Web-Based Resources*.

Alcoholism

As many as 50% of adults 18 years of age and over are regular drinkers, 14% are infrequent drinkers, 6% were former regular drinkers, 9% were former infrequent drinkers, and 21% were lifetime abstainers (Centers for Disease Control and Prevention, 2009); 23.6 million persons aged 12 or older needed treatment for an illicit drug or alcohol abuse problem (9.6% of the persons aged 12 or older). Of these, only 2.5 million—10.8% of those who needed treatment—received it at a specialty facility (National Institute on Drug Abuse, 2008). The lifetime prevalence of alcoholism in the United States is estimated at 14% (APA, 2000). Alcoholism is a chronic and disabling condition.

Substance abuse, including alcoholism, is common and may represent an attempt at symptom management in people diagnosed with schizophrenia, bipolar disorder, ADHD, GAD, obsessive–compulsive disorder, posttraumatic stress disorder, panic disorder, among others (Source: http://www.justice.gov/ndic/pubs7/7343/index.htm#What). People with insomnia who may or may not have comorbid psychiatric disorders often self-medicate to improve sleep, and this may lead to alcohol addiction.

The effects of alcohol on sleep are deceptive. While alcohol enhances gamma amino butyric acid (GABA), a major neurochemical involved with sleep (see Chapter 1, Physiological and Behavioral Aspects of Sleep) and reduces sleep latency, its negative effects are seen later in the night. Alcohol rapidly metabolizes and as it does, it causes fragmentation in sleep, reducing sleep efficiency. The summative effect is negative and becomes a perpetuating factor in continuing the use of alcohol for sleep. Sleep disturbance is also a universal predictor for relapse, thus complicating treatment and potentially compromising the quality of clinical outcomes for the affected individual (Brower & Perron, 2009).

Like the relationships of other psychiatric disorders to sleep disorders, the relationships between substance abuse and sleep disorders are complex and bidirectional. Of particular note,

Table 12.6 ■ *Web-Based Resources Related to Sleep and Psychiatric Disorders*

- National Institute of Drug Abuse, http://www.drugabuse.gov/NIDAHome.html
- National Sleep Disorders Research Plan, http://www.nhlbi.nih.gov/health/prof/sleep/res_plan/section5/section5g.html
- National Institute on Alcohol Abuse and Alcoholism, http://www.niaaa.nih.gov/
- American Psychiatric Association, http://www.psych.org
- Depression and Bipolar Support Alliance, http://www.dbsalliance.org
- American Anxiety Disorders Association, http://www.adaa.org/
- National Attention Deficit Disorders Association, http://www.add.org
- National Alliance on Mental Illness, http://www.nami.org/
- Society for Light Treatment and Biological Rhythms, www.sltbr.org/
- BioBrite (Light therapy equipment), http://www.biobrite.com
- Verilux (Light therapy equipment), http://www.verilux.com/
- Mayo Clinic Light Therapy Resource, http://www.mayoclinic.com/health/light-therapy/MY00195

Disclaimer: The light therapy sources cited here are intended solely for the purpose of showing the breadth of products available on the market. In no way are they endorsements of the company or their products. The author has no disclosures to make related to financial relationships with either of these companies.

Wong, Brower, and Zucker (2009) reported that sleep problems in children aged 3–6 years predicted early onset of substance use in boys. The implications of such findings are staggering.

Alcohol ingestion may also contribute to OSA through relaxation of the musculature responsible for patency of the airway during sleep (Remmers, 1984), although there have been conflicting findings about these effects. (See Chapter 7, Sleep-Related Breathing Disorders.) Krystal, Thakur, and Roth (2008) note the strong support in the literature for risk reduction in those diagnosed with alcoholism. The risk of relapse is greatly reduced when sleep disturbance is reduced.

Pharmacologic Interventions in Alcoholism

Given the common experience of sleep pattern disturbance during the recovery period of a person with alcoholism, the use of medications aimed to facilitate sleep is common. Naltrexone and acamprosate are used to facilitate withdrawal and may reverse some of the abnormal sleep architecture associated with withdrawal (Staner et al., 2006).

Nonpharmacologic Interventions in Alcoholism

Behavioral measures to enhance sleep are critical and include the components of CBT-I including sleep hygiene, relaxation, and stimulus control. Recent literature (Arnedt, Conroy, Rutt, Aloia, & Brower, 2007) report the positive effects of CBT-I in alcoholics in recovery but note that further studies need to be conducted to examine the impact of CBT-I on relapse rates. Reinforcing the critical role of a program of recovery (Alcoholics Anonymous or others) will endorse the importance of utilizing available resources in health maintenance in recovery.

Assessment and Treatment of Sleep Disorders in Patients with Alcoholism

Similar to other psychiatric illnesses, treatment goals vary according to the stage of recovery. During early sobriety, baseline assessment data are used to assist the patient to understand the relationship between alcoholism and sleep; the perpetuating influence of alcohol on insomnia over the course of active illness; and the need for a well-informed and structured approach to sleep. Given the high comorbidity of alcoholism and mood disorders, assessment methods are similar (Arnedt et al., 2007). During the acute phase of recovery, the focus of treatment is exclusively on safe withdrawal from alcohol and thereafter, the focus of treatment shifts to include standard approaches with CBT-I.

During recovery, behavioral measures to enhance sleep are critical and include components of CBT-I including sleep hygiene, relaxation, and stimulus control. Sleep restriction therapy and formal cognitive therapy aimed at resolution of the sleep disturbances needs to be done under the direction of an experienced clinician (Arnedt et al., 2007). Patients should be encouraged to obtain adequate sleep and avoid getting overly tired. Maintaining a regular schedule, including meals and social interaction, is helpful. Patient and family teaching related to the vulnerabilities associated with untreated insomnia and relapse are important to risk reduction and relapse prevention during recovery.

While the discussion here has focused on alcohol addiction, the principles for assessment and treatment for sleep disturbances in addiction are similar across the various categories of substance abuse. Brower and Perron (2010) have developed a conceptual framework that can be applied to understanding sleep pattern disturbances across the various classes of addictions. They cluster sleep disturbances associated with alcohol, amphetamines, cannabis, cocaine, nicotine, opioids, and sedative-hypnotics additions as a cluster of universal withdrawal symptoms or universal symptoms of protracted abstinence, and these clusters are verified with polysomnography. Both clusters are coupled with negative affect and craving, and these are subjective measures that permeate the objective measures in both acute and protracted recovery. Their recommendations for practice include judicious use of medication

for sleep disturbances across the addictions in addition to behavioral measures, including CBT-I.

ATTENTION DEFICIT HYPERACTIVITY DISORDER AND SLEEP

ADHD is a disorder of childhood that frequently has residual symptoms into adulthood. Most children and their parents report that the children have disturbed, fragmentary nocturnal sleep (Silvestria et al., 2009). Adults diagnosed with ADHD have significantly later bedtimes, longer sleep latency, and are more likely to experience difficulty going to bed, going to sleep, sleeping restfully, or waking in the morning (Surman et al., (2009). They also report more EDS than controls (Sobanski, Schredl, Kettler, & Alm, 2008). Sleep pattern disturbances in people with ADHD may be related to RLS, periodic limb movements in sleep, sleep-onset delay, increased nocturnal motor activity, sleep-disordered breathing, and deficits in alertness and sleep alterations that may be the result of comorbid psychiatric disorders or ADHD medications (Konofal, Lecendreux, & Cortese, 2010).

A significant challenge in dealing with the sleep disturbances in persons with ADHD is the common presence of psychiatric comorbidity, including bipolar disorder, substance abuse disorders, anxiety disorders, and depressive disorders (Klassen, Katzman, & Chokka, 2010)—all of which are associated with sleep abnormalities. The treatment for one comorbid condition may have a negative on another comorbid condition and sleep. For example, treatment of a bipolar illness with an antiseizure medication may lead to cognitive cloudiness that may exacerbate daytime sleepiness.

A distinguishing feature of sleep in bipolar illness versus ADHD is that the person with ADHD may have more difficulties focusing on following a standard bedtime routine to allow for adequate sleep, whereas the sleep of the person diagnosed with bipolar illness may be characterized by difficulties with sleep initiation and continuity and subsequent daytime impairment. While both groups are likely to experience consequences of sleep pattern disturbance, the specific causes of the sleep-related difficulties differ.

Pharmacologic Interventions in ADHD

A major challenge in treating ADHD is that the medications used are potent stimulants—all of which have a negative impact on sleep. Although extended or once-daily formulations may enhance adherence and treatment effectiveness across the day, they may also lead to iatrogenic sleep disturbance. One alternative is atomoxetine, a nonstimulant formulation.

Nonpharmacologic Interventions in ADHD

Standard treatment for ADHD includes addressing comorbid conditions to abate symptoms. Interventions include behavioral approaches to enhance organizational skills and cognitive function.

Assessment and Treatment of Sleep Disorders in Persons With ADHD

The work of Konofal et al. (2010) offers some outstanding points for nurses in the ongoing assessment of those diagnosed with ADHD whose vulnerability is high due to the possible iatrogenic effects of the medication used to treat the disorder. A program of ongoing monitoring and collaboration with the prescriber will reduce treatment liabilities and enhance the quality of clinical outcomes. Standard sleep assessment becomes the foundation of working with both the patient and parent(s) with a plan for sleep. Education needs to include discussion of the impact of the stimulants on sleep and interventions related to sleep hygiene and stimulus control. Cognitive–behavioral approaches to insomnia may be merited, depending on the perceptions of the patient and parents related to sleep. Online resources for children are available (sleepeducation.com and sleepfoundation.org) and can facilitate both engagement in treatment and learning with usually familiar online approaches to learning. The equivalent learning opportunities for adults receiving treatment apply here.

CONCLUSIONS

O sleep, O gentle sleep, Nature's soft nurse,
How have I frightened thee,
That thou no more wilt weight
my eyelids down
And steep my senses in forgetfulness?
—Wm. Shakespeare, King Henry IV

This chapter has addressed critical information pertinent to sleep and its relationship to psychiatric disease. It is clear that sleep disturbance goes hand in hand with psychiatric disorders. Although many questions remain unanswered about the direction of these relationships and the underlying mechanisms, sleep may lead to psychiatric disorders, psychiatric disorders may lead to sleep disorders, and both may be manifestations of underlying pathophysiological processes. It is increasingly evident that medications used to treat psychiatric disorders may also contribute to problems with sleep, and sleep patterns may also be important markers of recovery and exacerbation over the trajectory of these disabling chronic conditions.

Psychiatric disorders, as well as sleep disorders, are often comorbid with medical conditions; and both may contribute to disabling chronic conditions such as heart failure and diabetes. Although these relationships are complex, assessment, prevention, anticipatory guidance, and treatment of sleep disorders are critical components of nursing care of patients with psychiatric disorders in every health care setting. A well-informed nursing perspective on sleep and its relationship to psychiatric illness will inform assessment strategies, enhance the precision and effectiveness of intervention plans, and ideally reduce risk of morbidity in those diagnosed with psychiatric illnesses.

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13 Pediatric Sleep Disorders

Kristen Hedger Archbold

Approximately 40%–45% of children experience sleep disorders at some point between infancy and adolescence (Meltzer, Johnson, Crosette, Ramos, & Mindell, 2010). The most common are sleep-onset insomnia, parasomnias, sleep disordered breathing, and movement disorders (Archbold, Pituch, Panahi, & Chervin, 2002; Meltzer et al., 2010). Unfortunately, sleep disorders are often underdiagnosed in children of all ages (Meltzer et al., 2010). The purpose of this chapter is to provide an overview of common sleep disorders that affect children and specific information on sleep assessment in pediatric patients. An underlying theme is that children often exhibit symptoms of sleep disorders in ways that differ from the presentation of similar disorders in adults.

INSOMNIA

Insomnia occurs in 20% of children on a regular basis and up to 54% of children at least once in their development (Archbold et al., 2002; Meltzer et al., 2010). The forms of insomnia during childhood include behavioral insomnia of childhood, adjustment insomnia (associated with psychological responses to psychological or physical stressors), psychophysiological insomnia (associated with insomnia around bedtime), paradoxical insomnia (misperception of sleeplessness when sleep is normal), and idiopathic insomnia (no identifiable cause). (Lipton, Becker, & Kothare, 2008). Mindell et al. (2006) developed a developmentally appropriate definition of pediatric insomnia: "Repeated difficulty with sleep initiation, duration, consolidation, or quality that occurs despite age-appropriate time and opportunity for sleep and results in daytime functional impairment for the child and/or family (p. e1225). It is important to note that children's insomnia is often reported by a parent or guardian". Behavioral insomnias of childhood are the most prevalent form in young children and will be the focus in this chapter.

Behavioral insomnias of childhood are usually associated with dysfunctional behavior patterns and environmental influences (Moore, Allison, & Rosen, 2006). Insomnia is evident in children as young as 6 months of age and occurs over the course of development (Halbower & Marcus, 2003). Table 13.1 lists the subtypes and prevalence of the behavioral insomnias of childhood (Meltzer et al., 2010; Mindell et al., 2006; Rosen, Owens, Mindell, Xue, & Krichner, 2005).

Limit-setting insomnia is characterized by inconsistency and/or absence of limit setting from parents/caregivers around bedtime. Up to 30% of children experience this problem during the first 2 years of life (Mindell, Emslie, et al., 2006). Parents report that children do not have a consistent bedtime, may not have consistent bedtime preparation rituals or routines, and/or may be resistant to bedtime. The child may stall or attempt to avoid going to sleep altogether (Owens, Rosen, Mindell, & Kirchner, 2010).

Children with behavioral limit-setting insomnia can also experience sleep-onset association disorder. In both instances, parents become weary of their own state of sleep deprivation and seek help from nurses to intervene on the

Table 13.1 ■ *Behavioral Insomnias of Childhood*

Behavioral Insomnia Type	Main Symptoms	Rate and Prevalence
Limit-Setting Disorder[a]	Inconsistent bedtimes Lack of bedtime preparation routine Refusal of child to go to sleep or to remain in bedroom at night	10%–30%
Sleep-Onset Association Disorder[a]	Need for stimulus or object to be present in order to initiate sleep Night-time waking that requires parental intervention for child to return to sleep	10%–30%

Sources: "Prevalence of Diagnosed Sleep Disorders in Pediatric Primary Care Practices," by L. J. Meltzer, C. Johnson, J. Crosette, M. Ramos, and J. A. Mindell, 2010, *Pediatrics, 125*(6), pp. E1410–E1418. "Behavioral Treatment of Bedtime Problems and Night Wakings in Infants and Young Children—An American Academy of Sleep Medicine Review," by J. A. Mindell, B. Kuhn, D. S. Lewin, L. J. Meltzer, and A. Sadeh, 2006, *Sleep, 29*(10), pp. 1263–1276.

[a]Most often these two disorders occur simultaneously in some combination of symptoms.

child's behalf (Owens et al., 2010). Children with sleep-onset association disorder associate sleep with an object or behavior that becomes essential for falling asleep. Examples include inability of the child to fall asleep without the parent in the room until the child falls asleep or the need for the parent's hand on the child's arm while the child falls asleep. For breastfeeding infants, the mother's breast becomes a cue associated with sleep onset, and the infant of 6 months or older cannot fall asleep unless he or she is nursing (Moore, Meltzer, & Mindell, 2008). At the age of 6 months, the child is physiologically able to sleep continuously through the night for 6–8 hours without the need to be fed or have a diaper change and therefore, should not require intervention from the parent.

Both types of limit-setting insomnias are successfully treated with behavioral change interventions. Techniques range from extinction (i.e., ignoring protestations from the child after bedtime and before scheduled wake time) to scheduled check-in periods during which the parent gradually decreases nocturnal interactions with the child as the treatment progresses (Mindell, Meltzer, Carskadon, & Chervin, 2009). A complete review of behavioral insomnia treatment strategies is available (Mindell & Owens, 2010). For these techniques to be successful, both parents and caregivers must be fully invested in the intervention, and the nurse must support them in enduring the (frequently short-term) protestations from their child that may result at the beginning of intervention.

As children grow, other forms of insomnia become more common. Sleep hygiene issues may play an important role in school-aged children and adolescents. (See Chapter 2, Developmental Aspects of Normal Sleep, and Chapter 6, Insomnia.) Stress, anxiety, and the demands of school and other activities may contribute to insomnia symptoms. Psychophysiological insomnia in which the individual experiences heightened arousal and excessive arousal about sleep can also occur in adolescents (Lipton et al., 2008). Insomnia can be an antecedent or a consequence of significant mental health conditions (e.g., anxiety or depression), so reports of insomnia symptoms should be a trigger for investigation for the presence of these disorders. Insomnia is especially common in children with neuropsychiatric disorders, such as pervasive developmental disorders and children with other psychiatric or medical conditions.

Behavioral approaches are particularly important to the treatment of insomnia in children. Cognitive behavioral approaches (see Chapter 6, Insomnia) seem to be helpful in older children, but there is a need for systematic research in this area. Although pharmacotherapeutic agents (i.e., benzodiazepines, alpha-2 agonists, antihistamines, and chloral hydrate) are frequently prescribed (Lipton et al., 2008; Pelayo & Dubik, 2008), data on their efficacy and safety in children are lacking (Mindell et al., 2006), and the Food and Drug Administration (FDA) has not approved any hypnotic agents (with the exception of chloral hydrate, a drug not recommended by pediatric experts) for use

in children. Although these agents are intended for sedation, some children experience excitability with their use.

Diphenhydramine, an over-the-counter H_1 nonspecific antihistamine, is often used as a hypnotic in children with sleep-onset difficulties (Owens et al., 2010). However, results of its efficacy in improving sleep onset and prevention of night-awakenings in young children are mixed, and, paradoxically, it may lead to excitation in some children. Nurses should inform parents of these concerns (Rosen et al., 2005).

DELAYED SLEEP PHASE DISORDER

Delayed sleep phase disorder (DSPS), prevalent in 16% or more of adolescents (Hagenauer, Perryman, Lee, & Carskadon, 2009), develops as the circadian sleep-onset pattern becomes later in the evening compared to early childhood (Tarokh & Carskadon, 2010). Although this change in phase is considered a normal part of development, difficulty arises when there is a mismatch between the teen's circadian pattern and demands of school, work, and other activities. (See Chapter 2, Developmental Aspects of Normal Sleep, and Chapter 10, Circadian Rhythm Disorders.)

DSPS is characterized by a very late sleep onset (often beyond midnight) with difficulty waking during the school week and sleeping late on weekend/nonschool days. School nights are associated with shortened sleep periods and weekends with sleep periods of 10 hours or more when possible (Hagenauer et al., 2009). DSPS results in sleep deprivation and difficulty waking for daily activities. Impaired learning and absenteeism occur in more severe cases (Carskadon, Coon, Saletin, McInrue, & Arantes, 2008; Mindell et al., 2009).

Treatment for DSPS requires full participation from adolescents and parents to be effective. A sleep log and/or actigraphy can be helpful in planning the timing of intervention strategies that include gradual shifting sleep preparations and onset 15 minutes earlier, regular sleep schedules on weekdays and weekends, and identifying and modifying wake time activities as much as possible. It is helpful to work with teens to identify the most important school and social activities and limit time spent on theirs. The demands of ever-increasing wake time activities (e.g., work, school, extracurricular activities, and social activities) among many adolescents make this a very challenging task.

As in adults, sleep hygiene strategies involving modifications of the sleeping environment, avoiding stimulation before bedtime, and avoiding caffeine consumption are useful in children. (See Chapter 6, Insomnia, and Chapter 18, Sleep and Primary Care of Adults and Older Adults.) Parents and teens also need to be informed of the potential difficulties associated with interactive electronic screen exposure before bedtime. Computer use, phone text messaging, and video and computer gaming significantly delay sleep onset and increase the amount of N2 sleep at the expense of other sleep stages (Dworak, Schierl, Bruns, & Struder, 2007). This results in sleep deprivation and impaired daytime performance. Nurses can instruct parents to make sure that their children finish video gaming at least 1 hour prior to the desired sleep onset time. The child's bed should be used only for sleep and it is a good practice to avoid having electronic devices or media (e.g., computers, television, cellular phones) in the child's bedroom.

Bright light therapy at waking is useful in re-training patterns of melatonin release and circadian rhythms (Mindell & Owens, 2010). (See Chapter 10, Circadian Rhythm Disorders, and Chapter 12, Sleep and Psychiatric Disorders.) Investigators have also shown that melatonin is useful in reducing sleep-onset latency and prolonging sleep in some children with ADHD (see Bendz & Scates, 2010, for review). Melatonin (dosage range 1–3 mg) should be used with caution and monitored closely because the long term effects of melatonin supplements on the hypopituitary gonadotrophic axis and pubertal development are currently unknown (Owens et al., 2010). Because supplements are not regulated by the FDA, actual concentration levels of melatonin in over-the-counter preparations may not be consistent among brands (Bendz & Scates, 2010).

OBSTRUCTIVE SLEEP APNEA

Some reports suggest that 3% of the pediatric population has obstructive sleep apnea (OSA), but investigators have found its symptoms in up to 16% of children (Archbold et al., 2002; Meltzer, Johnson, Crossette, Ramos, & Mindell, 2009). Whether this discrepancy is a result of underdiagnosis is not known. OSA affects boys and girls at the same rate throughout childhood, but it may be more prevalent in boys during the teenaged years as body mass index (BMI) and body composition change during puberty. African American and socially disadvantaged children appear to be at a greater risk for developing OSA versus Hispanic or White children. (Mulvaney et al., 2006; Spilsbury et al., 2006). (See Chapter 15, Racial/Ethnic Health Disparities and Sleep Disorders.)

Unfortunately, the frequent failure of nurses and other health care providers to assess children for OSA results in underdetection and undertreatment of this condition (Meltzer et al., 2010). The current diagnostic criteria for pediatric OSA are listed in Table 13.2 and have been adapted from the ICSD-2 descriptions of pediatric OSA symptoms. Specific formal or standardized criteria for diagnosis are currently being developed and not yet uniformly applied by all sleep specialists (American Academy of Sleep Medicine, 2005).

The criteria for scoring respiratory events in children differ from those used for adults and are listed in Table 13.3 (Miano, Paolino, Castaldo, & Villa, 2010). To be classified as apneic events, apneas in infants or children do not need to be followed by arousals, wake, or oxygen desaturation. Central apneas that follow a sigh, snore, arousal, or respiratory event are not scored unless accompanied by a desaturation of 3% or more. Hypopneas cannot be classified as obstructive, central, or mixed (combination of obstructive and central) without a quantitative assessment of ventilatory effort. Obstructive hypoventilation is unique to children (Tapia et al., 2008) and occurs primarily in children who have brainstem abnormalities. Nonobstructive hypoventilation occurs in obese children. Both are associated with increases in end-tidal CO_2 levels (ETCO2) recorded at levels from more than 45 to 50 mm Hg for more than 10%–25% of total sleep time (Miano et al., 2010).

Etiology of Obstructive Sleep Apnea (OSA) in Children

Unlike adults, the most common cause of OSA in children is enlarged adenoid and tonsillar tissues that decrease the upper airway diameter (Halbower & Marcus, 2003). Snoring, the most common symptom of OSA in children from infancy through adolescence, results from turbulence in airflow through the narrowed oronasopharynx. Children who snore do not necessarily have OSA, but may have some degree of upper airway obstruction (primary snoring, upper airway resistance syndrome).

Table 13.2 ■ ICSD-2 *Description of Obstructive Sleep Apnea in Children*

- One or more scorable respiratory events (apnea or hypopnea) by polysomnography per hour
- Polysomnography demonstrates EITHER
  - at least one of the following
    a. Frequent arousals associated with increased respiratory effort
    b. Oxygen desaturations that are associated with apneic episodes
    c. Hypercapnea during sleep (>45–50 mm Hg)
    d. Markedly negative esophageal pressure

OR

- Periods of hypercapnea and desaturation during sleep associated with snoring and/or paradoxical breathing and at least one of the following:
  a. Frequent arousals from sleep
  b. Markedly negative esophageal pressure

Source: International Classification of Sleep Disorders: Diagnosis & Coding Manual (ICSD-2) (2nd ed.) by American Academy of Sleep Medicine, 2005, Westchester, IL: Author.

Table 13.3 ■ *Comparison of Adult and Pediatric Criteria for Respiratory Events*

Respiratory Event	Adult Criteria	Pediatric Criteria
Obstructive apnea	Duration of ≥10 seconds >70% reduction	Duration of two breaths 90% reduction in amplitude of nasal pressure flow signal Associated with continued or increased respiratory effort
Hypopnea	≥30% fall in amplitude from any airflow or effort-related signal Associated with a 4% desaturation	≥50% fall in amplitude of nasal pressure signal two breaths duration Associated with arousal, awakening, or 3% desaturation
Central apnea	Duration >10 seconds	Duration of two breaths or 20 seconds or longer associated with an arousal or awakening or 3% desaturation
	Absent respiratory effort	Absent respiratory effort
Respiratory effort-related arousal (RERA)	Drop in inspiratory airflow, increase in respiratory effort, and EEG arousal but do not meet apnea or hypopnea criteria	Duration of two breaths Decrease in nasal pressure sensor amplitude <50% Flattening of nasal pressure wave form Accompanied by snoring, noisy breathing, increased CO_2 level or work of breathing
Obstructive and nonobstructive hypoventilation	Not seen in adults	Continuous partial airway obstruction Paradoxical respiratory effort Increased $ETCO_2$ can be associated with hypoxemia Occurs in conjunction with or separate of diagnosis of OSA

Sources: International Classification of Sleep Disorders: Diagnosis & Coding Manual (ICSD-2) (2nd ed.), by American Academy of Sleep Medicine, 2005, Westchester, IL: Author. "Visual Scoring of Sleep: A Comparison Between the Rechtschaffen and Kales criteria and the American Academy of Sleep Medicine Criteria in a Pediatric Population With Obstructive Sleep Apnea Syndrome," by S. Miano, M. C. Paolino, R. Castaldo, and M. P. Villa, 2010, *Clinical Neurophysiology, 121*(1), pp. 39–42.
Abbreviation: OSA, obstructive sleep apnea.

Snoring is associated with impaired scholastic and behavioral patterns (Chervin, Ruzicka, et al., 2006; Melendres, Lutz, Rubin, & Marcus, 2004; Montgomery-Downs, O'Brien, Holbrook, & Gozal, 2004; O'Brien et al., 2007; O'Brien, Mervis, et al., 2004).

Overweight and obesity are significant risk factors for OSA in children. The large increase in the proportion of obese children and adolescents is associated with increases in the incidence and prevalence of OSA. This is a major public health concern given the implication of both obesity and sleep apnea for metabolic and cardiovascular disorders. (See Chapter 7, Sleep-Related Breathing Disorders.)

Manifestations of OSA in Children

The etiology of OSA seems to have a differential impact on daytime function in children. Obese children with OSA are most likely to present with symptoms of excessive daytime sleepiness, failing or poor quality school work, behavioral problems, and falling asleep in class (Kheirandish-Gozal & Gozal, 2009; Louis, Redline, & Auckley, 2009; Moore et al., 2009; Ross et al., 2009; Spruyt et al., 2010). On the other hand, children with enlarged lymphoid tissues or other non–obesity-related etiologies are more likely to display hyperactive and disruptive behaviors in school and at home. These symptoms may mimic the behaviors of attention deficit hyperactivity disorder (ADHD). Paradoxically, sleep-deprived children with OSA can display patterns of inattention and disturbed social skills that may be similar to conduct disorder (CD), oppositional defiant disorder (ODD), and other difficult behaviors (Blunden & Chervin, 2010; Giordani et al., 2008; Hodges et al., 2008; Montgomery-Downs et al., 2004; Spruyt et al., 2010).

Children with OSA may have enuresis due to the decreased amount of N3 sleep, the stage in which urine is concentrated due to the release of antidiuretic and growth hormones. Effective treatment of OSA often eliminates enuresis and may lead to growth spurts as a result of the increase amounts of N3 sleep and secretion of growth hormones (Chervin, Ruzicka, et al., 2006; Dillon et al., 2007).

Gastroesophageal reflux may also be present, although its prevalence is not known in children. Gastroesophageal reflux can result in sleep fragmentation due to discomfort, pain, coughing, and dyspepsia (Kusano, Kouzu, Kawano, & Ohara, 2008; Parish, 2009).

Like obese adults, children with a high BMI are susceptible to hypoventilation and airway collapse during sleep (Ross et al., 2009). CPAP is an effective treatment for OSA in obese children, but diet, exercise, and education regarding the effects of obesity on OSA and daily function are also important interventions (Fennig, 2006; Spruyt et al., 2010) and have additional health-related benefits.

Treatment of OSA in Children

Adenotonsillectomy results in "cure" of OSA in 60% of children who have this risk factor (Hoban, 2010) and leads to improvements in behavior (Chervin, Weatherly, et al., 2006; Hodges et al., 2008). However, in some children adenotonsillectomy improves, but does not eradicate OSA. These children may require use of continuous positive airway pressure (CPAP). Although an apnea/hypopnea index of greater than or equal to 5 is generally the point at which treatment for pediatric OSA is initiated in the clinical setting (Marcus, 1997, 2008), research is needed to determine the specific level of pediatric OSA at which treatment is likely to completely improve negative behavioral, cognitive, and cardiovascular sequelae.

A landmark study (Gozal, 1998) found that scholastic performance (measured by year-end grades) improved by an entire grade level 1 year after adenotonsillectomy, compared with children with OSA who did not have adenotonsillectomy. Subsequent studies have consistently demonstrated the negative effects of OSA on children. These include alteration of endothelial function, sleep architecture, decreased attention, mental flexibility, and impaired behavior patterns (Bhattacharjee, Dayyat, Kheirandish-Gozal, Capdevila, & Gozal, 2009; Chervin, Archbold, Dillon, Panahi et al., 2002; Chervin, Dillon, Archbold, & Ruzicka, 2003; Gozal et al., 2010).

OSA is associated with inflammatory and altered metabolic processes in children (Gozal et al., 2010; Gozal & Kheirandish-Gozal, 2008). Obese children with OSA have increased levels of systemic inflammation and increased platelet-leukocyte-endothelial processes that have both been associated with cardiovascular disease. Treatment of obese children who have OSA with adenotonsillectomy led to decreases in serum c-reactive protein and other inflammatory biomarkers, but BMI and blood glucose levels remain unchanged (Gozal & Keirhandish-Gozal, 2008). There is a great need for further study of the treatments for OSA, including weight loss in obese children, and the ways in which treatment improves metabolic and cardiovascular health outcomes for children with OSA.

CPAP (see Chapter 7, Sleep-Related Breathing Disorders) is a second-line treatment for OSA in children and significantly reduces AHI when used for 6 hours at least 5 nights per week (Koontz, Slifer, Cataldo, & Marcus, 2003; Marcus, 1997). Children generally learn to tolerate the therapy well with sufficient support from providers and parents (Koontz et al. 2003). Further study is needed of the extent to which CPAP improves sleep patterns, behavior, and cognition among children.

There is little evidence to support the use of dental appliances to treat OSA in children (Guilleminault, Pelayo, Leger, Philip, & Ohayon, 2001), but research is underway to assess the utility of upper palate distraction (use of dental bridgework to gradually expand the width of the upper palate) in children with OSA who have high-arched palates (Holty & Guilleminault, 2010). Maxillofacial surgery is used in some children to correct facial structures that contribute to a narrowed airway (i.e., micrognathia,

retrognathia, genetic syndromes with facial morphologies), but surgical procedures are not used in children who have normal craniofacial structure.

Special Pediatric Populations and OSA

Children with developmental disabilities and genetic syndromes (e.g., Trisomy 21, Prader-Willi Syndrome, Craniofacial abnormalities) are generally at greater risk for OSA due to many factors, such as upper airway shape, alterations in airflow, and excessive weight gain that occurs with certain genetic profiles. Much has yet to be learned about the factors that place these children at increased risk for sleep disorders and the impact of sleep disorders on the developmental trajectory in these children. For example, children with ADHD have special needs and issues surrounding sleep health that practitioners should be aware of. In fact, parents have reported concerns with sleep behaviors in up to 50% of these children (Owens et al., 2010).

As many as 35% of children seen in a pediatric psychiatric clinic have symptoms suggestive of OSA and PLMD (Chervin & Hedger, 2001). Children with ADHD, ODD, and CD are at increased risk for OSA and PLMD (Dillon, Ruzicka et al., 2002; Gaultney, Merchant, & Gingras, 2009). Although there is a need for extensive research on the best ways to address this problem, there is evidence that treatment of OSA with tonsillectomy eliminated problematic behaviors associated with ODD/CD diagnoses (Dillon et al., 2007). Although OSA and PLMD or other sleep disorders are not described on standard clinical algorithms used to identify and diagnose problems such as ADHD (Reynolds, Redline, & Workgroup, 2010), the behavioral effects of OSA are often similar to those of ADHD. Children with ADHD and those with problematic daytime behavior patterns should always be screened for OSA, periodic limb movement disorder (PLMD) and/or restless legs syndrome (RLS) (Gaultney et al., 2009; Mehl et al., 2006; O'Brien et al., 2007; O'Brien et al., 2009; Paruthi & Chervin, 2010).

PARASOMNIAS

Parasomnias vary in incidence from 10% to 45% of children, as reported by parents (Archbold et al., 2002). Bruxism (grinding of the teeth) is the most common. Sleep terrors and sleep talking or walking are also commonly reported. In general, children outgrow their parasomnias over time, and parasomnias usually disappear altogether by age 8 (Archbold et al., 2002; Guilleminault, Palombini, Pelayo, & Chervin, 2003; Meltzer et al., 2009; Meltzer et al., 2010). (See Chapter 8, Sleep-Related Movement Disorders and Parasomnias.)

Bruxism

Bruxism is the repetitive, nonfunctional grinding or clenching of the teeth during sleep. It generally occurs during Stages N1 and N2 of sleep, but is infrequently associated with increased arousals during sleep. The age of onset is around 3 years, with disappearance of symptoms around age six. Bruxism can lead to tooth enamel erosion, temporomandibular joint pain and maxillary hypertrophy, and sensitivity and pain in the masseter and/or temporal muscles of the face and skull (Quintero et al., 2009; Velez et al., 2007).

Children with bruxism should be referred for dental evaluation. Intraoral appliances are useful in reducing enamel erosion. However, children with these devices should be carefully assessed because these devices may lead to alterations in the growth and development of jawbone and palatal structures (Quintero et al., 2009; Velez et al., 2007). Further research is needed into the long-term effects of devices used to treat pediatric bruxism (Zhang, McGrath, & Hagg, 2008).

Sleep Terrors

Sleep terrors occur at least once in about 46% of children. They are characterized by screaming loudly and inconsolability. In these cases, the child cannot be calmed by parental presence, and sleep terrors are most often the source of the disturbing behavior (Archbold et al., 2002; Guilleminault et al., 2003; Meltzer et al.,

2009). Although it is terrifying for parents and caregivers to witness sleep terrors, children are unable to recall the occurrence of the screaming episode the following morning. Amnesia for the event is the distinguishing feature of sleep terrors and differentiates them from nightmares or nightmare episodes (Bloomfield & Shatkin, 2009; Snyder, Goodlin-Jones, Pionk, & Stein, 2010).

Sleep terrors occur during non-REM sleep, during the first half of the nighttime sleep period. In contrast, nightmares occur during the second half of the night, when REM sleep is more prevalent. Sleep terrors usually occur during the transition from N1 to N2 sleep, or vice versa and are generally benign. Sleep terrors are most frequently outgrown by the time a child is 6 years old and rarely persist into adolescence (Guilleminault et al., 2003).

Sleep-Walking

Approximately, 20% of children walk in their sleep before the age of 8 years (Archbold et al., 2002; Meltzer et al., 2009) and this behavior is more common in boys than girls (Mahendran, Subramaniam, Cai, & Chan, 2006). Like sleep terrors, sleep-walking is primarily a disorder of N3 sleep in the first part of the night. Children are not aware of their surroundings when they walk in their sleep and are usually amnestic for the event the following morning. They may appear groggy, confused, and perform bizarre actions such as trying to hang a coat up on a bare wall or urinating in the hallway. In general, sleep-walking is benign, but safety is the most pressing concern. Care must be taken to keep dangerous or sharp objects out of the sleep-walking child's way, as children have been reported to walk out into a snowy yard during their sleep and step out onto a roof or balcony. Children who exhibit stereotypical behaviors (lip-smacking, hitting head with hand), drooling or tongue-biting during what appears to be a sleep-walking episode should be evaluated for nocturnal seizures especially if more than one of these behavioral events happens during the night (Davey, 2009; Silvestri & Bromfield, 2004).

MOVEMENT DISORDERS

Periodic Limb Movement Disorder (PLMD)

PLMD affects up to 12% of children between the ages of 3 and 18 (Meltzer et al., 2009). It is associated with regular kicking and jerking of the legs and/or arms during sleep at frequent intervals during the early hours of the evening (Grigg-Damberger et al., 2007; O'Brien et al., 2009). However, PLMD can be as subtle as a plantar flexion of the foot that occurs at regular intervals (every 20–40 seconds) during the night.

In children, PLMD is diagnosed when there are at least five episodes of limb movements per hour of sleep. The limb movements results in fragmented and disturbed sleep and symptoms of sleep deprivation (e.g., hyperactivity, inattention, and social misbehavior) that may be mistaken for ADHD (Chervin, Archbold, Dillon, Pituch, et al., 2002; Dillon et al., 2002).

PLMD is believed to be associated with decreased levels of iron in the blood or metabolic dysfunction of the iron metabolic and storage systems in the mid-brain. Alterations in iron metabolism and storage occur and contribute to central dopaminergic dysfunction (O'Brien et al., 2009). Therefore, serum ferritin levels should be evaluated in all children seen in clinic for disruptive behavior disorders and inattention problems. A serum ferritin lower than 50 ng/ml warrants treatment with iron supplementation in children with behavior and sleep problems. Polysomnography (PSG) is the only way to fully document and diagnose PLMD (Grigg-Damberger et al., 2007). PLMS are often seen in conjunction with OSA and narcolepsy, but PLMS that occur in these conditions are not diagnostic of PLMD. (See Chapter 8, Sleep-Related Movement Disorders and Parasomnias.)

Restless Legs Syndrome

RLS occurs in 2% of children between the ages of 2 and 12 years. Presentation of symptoms in children is similar to those reported by adults with RLS. (See chapter 8, Movement Disorders) Two notable differences in the presentation of RLS symptoms are the words and phrases

often used by children to report the feelings of discomfort in their legs. Children tend to use terms like "soda pop bubbling in my legs," "bugs crawling on my legs," and squeezing, tugging, pulling, or itching to describe the discomfort in their legs before bed (Mindell & Owens, 2010). Because RLS can significantly disrupt sleep patterns, the child with RLS usually presents with behavioral difficulties such as inattention, hyperactivity, and other social or academic difficulties (Chervin et al., 2002).

ASSESSMENT OF SLEEP IN CHILDREN

Sleep assessment should be customized to children's ages and developmental stages. (See Chapter 2, Developmental Aspects of Normal Sleep and Chapter 5, Conducting a Sleep Assessment.) The "BEARS" acronym is useful in guiding sleep assessment in children, as well as adults. (See Chapter 5, Conducting a Sleep Assessment.) Specific questions are useful in identifying symptoms of the most prevalent sleep disorders in all children and should be used to trigger the need for further sleep assessment. They should always be asked of every child and accompanying adult at every clinic visit or well-child assessment (see Tables 13.4 and 13.5).

Assessment of children for OSA is an important component of pediatric care. Nurses should assess children for the occurrence of snoring, obesity, and adenotonsillar hypertrophy. Hyperactive behavior and problems with school performance and daytime sleepiness are important signs and symptoms to document (Gozal & Kheirandish-Gozal, 2008).

Reports of frequent snoring (most nights per week) and/or any witnessed apneas should be further evaluated with more specific questions to further probe for the presence of OSA and the need for referral for polysomnography. A detailed algorithm for assessment of snoring is provided in Chapter 17, Sleep Promotion in Child Health Settings. Questionnaires are somewhat useful (see Chapter 5, Conducting a Sleep Assessment), but not diagnostic in many cases. The nurses should also elicit the presence of enuresis and gastric irritation and daytime sleepiness, often manifested in children as increase motor activity and hyperactivity, inattention, problematic social and scholastic behavior patterns, falling asleep in school, and daytime napping (O'Brien, Mervis et al., 2004).

Children usually outgrow the need for napping by the time they are 4 or 5 years of age (see Chapter 2, Developmental Aspects of Normal Sleep.) Therefore, napping in older children, especially preteens and adolescents may be a sign of excessive daytime sleepiness associated with a sleep disorder.

Positive responses to questions about PLMS should trigger investigation of the presence of PLMD. At present, PSG is the only way to diagnose PLMD (Grigg-Damberger et al., 2007).

Table 13.4 ■ *Sleep Assessment to Guide Screening and Identification of Specific Sleep Disorders*

Sleep Disorder Target	Questions to Ask:
Sleep apnea	Does your child snore? If yes, how often?
Sleep apnea	Have you ever noticed your child stop breathing during sleep, turn blue, or need to be awakened in order to breathe?
PLMD	Do you notice regular jerking movements of your child's arms or legs when he or she is sleeping?
Sleep apnea, PLMD, insomnia, delayed sleep phase syndrome	Is your child having any problems with paying attention in class, staying awake or interacting with their peers?
OSA, PLMD, insomnias, delayed sleep phase syndrome	How many hours of sleep does your child get each night? Does he or she nap or need to nap during the day?
OSA, PLMD, insomnias, delayed sleep phase syndrome	Do you have any concerns or questions about your child and his or her sleeping habits that I can help you with?

Abbreviations: OSA, obstructive sleep apnea; PLMD, periodic limb movement disorder.

Table 13.5 ■ *Pediatric Sleep Questionnaire (PSQ)*

The instrument contains a validated, reliable, 22-item SDB scale including a 4-item subscale for snoring and another for excessive daytime sleepiness. Scores are calculated as the proportion of symptoms that are endorsed as present. A criterion score for the SDB scale is 0.33. Answers are scored as "1" if the answer is "yes," and "0" if the answers are "no" and "don't know.". A criterion score for the SDB scale is 0.33. For PLMD, positive answers to the questions A12 through A14 would suggest further evaluation for symptoms associated with PLMs.	
WHILE SLEEPING, DOES YOUR CHILD. . .	
A2	. . . snore more than half the time?
A3	. . . always snore?
A4	. . . snore loudly?
A5	. . . have "heavy" or loud breathing?
A6	. . . have trouble breathing, or struggle to breathe?
HAVE YOU EVER . . .	
A7	. . . seen your child stop breathing during the night?
DOES YOUR CHILD . . .	
A24	. . . tend to breathe through the mouth during the day?
A25	. . . have a dry mouth on waking up in the morning?
A32	. . . occasionally wet the bed?
DOES YOUR CHILD . . .	
B1	. . . wake up feeling unrefreshed in the morning?
B2	. . . have a problem with sleepiness during the day?
B4	Has a teacher or other supervisor commented that your child appears sleepy during the day?
B6	Is it hard to wake your child up in the morning?
B7	Does your child wake up with headaches in the morning?
B9	Did your child stop growing at a normal rate at any time since birth?
B22	Is your child overweight?
THIS CHILD OFTEN . . .	
C3	. . . Does not seem to listen when spoken to directly.
C5	. . . Has difficulty organizing tasks and activities.
C8	. . . Is easily distracted by extraneous stimuli.
C10	. . . Fidgets with hands or feet or squirms in seat.
C14	. . . Is "on the go" or often acts as if "driven by a motor."
C18	. . . Interrupts or intrudes on others (e.g., butts into conversations or games).

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Abbreviation: PLMD, periodic limb movement disorder.

Like OSA, PLMD leads to sleepiness and is often accompanied by disruptive behavior patterns and difficulties with academic work and inattention in structured environments. (Blunden & Chervin, 2010; Chervin et al., 2003; Chotinaiwattarakul, O'Brien, Fan, & Chervin, 2009; Dillon et al., 2007; O'Brien et al., 2007; O'Brien, Mervis, et al., 2004; Spruyt et al., 2006).

When parents describe patterns of bedtime resistance, frequent night-waking episodes after the age of 6 months, and/or difficulties with initiating and maintaining sleep in their children, and answers to questions 1–3 in Table 13.3 are negative, the nurse should be concerned about delayed sleep-phase syndrome (especially for the young adolescent and teenaged patient),

behavioral insomnias, and patterns of sleep hygiene. With sleep diaries, actigraphs are useful in identifying patterns in sleep schedules and responses to repatterning (Johnson et al., 2007). (See Chapter 1, Physiological and Behavioral Aspects of Sleep, and Chapter 5, Conducting a Sleep Assessment.) Both parents and children should complete diaries because discrepancies in reports can be common and resolved with actigraphy data or vice-versa (O'Brien & Carskadon, 2008). Wrist actigraphy is useful, and its use as a diagnostic strategy has been reimbursed by some insurance carriers in sleep clinics (Goodlin-Jones, Waters, & Anders, 2009; O'Brien & Carskadon, 2008). (See Chapter 5, Conducting a Sleep Assessment.) PSG is only used in diagnosing these conditions to rule out OSA or PLMD. Comorbidity of these conditions is common. (Johnson et al., 2007).

Physical Assessment

Physical assessment of the child should always start with weight, height, and calculation of BMI. Head and neck circumferences should be plotted on growth charts and percentiles monitored. BMI should be calculated based on national public health standards of percentile for age. The child at either end of the BMI spectrum (obese vs. growth delay) should be assessed for sleep disturbances because both groups of children are particularly at risk for development or presence of OSA, PLMD or behavioral insomnias. (See Chapter 5, Conducting a Sleep Assessment.)

Otolaryngological assessment includes evaluation of tonsil size, shape of the hard palate, recurrent ear and throat inspection, and inspection of the nose. The presence of craniofacial characteristics that may suggest chronic mouth breathing (micrognathia, actual mouth breathing during exam, retrognathia) should be assessed. (See Chapter 4, The Nature of Sleep Disorders and Their Impact.)

Careful nursing assessment of the child's gastrointestinal system should be performed in order to document symptoms suggestive of increased stomach acid production, heart burn, and/or diarrhea that may be associated with gastric irritation. Serum ferritin levels are helpful in the documentation and management of PLMs and PLMD in children. Lead levels should be evaluated because heavy metal poisoning is associated with ADHD-like behaviors and neurological disruption (Ferber, 1996; Stores, 1996).

Blood gas values or oximetry readings obtained in the clinic may not be particularly helpful because they do not reflect values while the child is asleep. Given the cardiovascular consequences of OSA, assessment of blood pressure is also important. A complete neurological examination should be performed if nocturnal seizures are suspected or sudden, unexplained changes in sleep patterns (such as sudden onset of unexplained excessive daytime sleepiness, past history of closed head injury, etc.) (Ferber, 1996). (See Chapter 5 for more information.)

CONCLUSIONS

Sleep disorders are common among children and adolescence, with the prevalence varying according to developmental stage. The importance of incorporating a developmental approach to sleep assessment and treatment into nursing care is underscored by the profound effects of sleep and sleep disorders on children's behavior, learning, and daily function. Many sleep disorders can be addressed by the nurse and members of the interdisciplinary team with behavioral treatments. Others require referral and specialist treatment. Research is urgently needed on the efficacy of sleep disorders treatment in children and adolescents.

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Complementary and Alternative Medicine and Sleep

14

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As many as 38.3% of U.S. adults use some type of complementary and alternative medicine (CAM) to promote health or to reduce the symptom burden from chronic conditions (Barnes, Bloom, & Nahin, 2008), including insomnia and poor sleep quality. The purpose of this chapter is to review the evidence for the effects of CAM therapy for promoting sleep. Implications for nursing practice are discussed.

The term "complementary" refers to the use of therapies that are outside of the realm of conventional western medicine, while the term "alternative" refers to use of modalities in place of conventional western medicine. In this chapter, the review of research on the effects of CAM modalities on sleep is organized according to four CAM domains: mind–body, biological based, manipulative and body-based, and energy. CAM whole medical systems cut across all domains (NCCAM, 2010f). Table 14.1 provides an overview of CAM domains, examples of modalities in each category, and the specific modalities selected because of their potential relevance to sleep and sleep disorders discussed in this chapter.

CAM CLASSIFICATIONS

Whole Medical Systems: Acupuncture

Acupuncture is one of the oldest CAM practices and a part of traditional Chinese medicine. Acupuncture providers insert small needles into various parts of the body manually, with or without the use of electrostimulation (NCCAM, 2010a). The location of the acupuncture needles is selected according to the body's meridians, defined as body channels that guide the flow of body energy. The choice of specific meridians used for the insertion of the acupuncture needles is based upon an assessment that the acupuncturist completes, with the assessments varying from practitioner to practitioner. Acupuncture is usually performed once a day for consecutive days or weeks, based upon patient response and individual practitioner practice. Approximately 1% of U.S. adults used acupuncture in 2007. This is a slight increase since 2002 (Barnes et al., 2008).

Acupuncture seems to be an efficacious treatment for insomnia, as reported in several systematic reviews (Cao, Pan, Li, & Liu, 2009; Cheuk, Yeung, Chung, & Wong, 2007; Huang, Kutner, & Bliwise, 2009; Kalavapalli & Singareddy, 2007; Yeung, Chung, Leung, Zhang, & Law, 2009). Of 30 studies reviewed, 93% showed beneficial effects on sleep (Huang et al., 2009). Acupuncture was consistently superior to no treatment and to sham acupuncture, a placebo treatment that mimics acupuncture. There were improvements in sleep onset latency (SOL), wake after sleep onset (WASO), and total sleep time (TST), as measured with actigraphy (Huang, Kutner, & Bliwise, 2009). The effects of acupuncture on sleep are superior to benzodiazepines, with mean improvements of 91% for acupuncture versus 75% for benzodiazepines (Yeung et al., 2009); sleep hygiene, sham acupuncture, or no treatment (Cao et al., 2009; Cheuk et al., 2007). The use of acupuncture in addition to sedating medications led to greater

Table 14.1 ■ *Major Classifications of CAM Modalities*

CAM Categories	Specific Modalities	Examples in Chapter
Whole Medical Systems	Homeopathic Naturopathic Chinese medicine Ayerveda	Acupuncture
Mind–body	Support groups Cognitive–behavioral therapy Mindfulness meditation Expressive arts	Mindfulness meditation Expressive arts (music)
Biologically based	Herbs Vitamins and minerals	Valerian Lavender Melatonin
Manipulative and body-based practices	Massage Chiropractic manipulation Tai Chi Qigong Yoga	Massage Tai Chi Qigong Yoga
Energy-based modalities ■ Biofield ■ Bioelectromagnetic	 Healing touch Pulsed fields Static magnetic fields	 Healing touch Pulsed fields Cranial–electrical stimulation

improvements in sleep duration than medication alone (Cao et al., 2009). In the majority of studies the "effective rate," or proportion of participants who had at least some improvement in their sleep after therapy, was the only outcome reported, but the improvements in specific sleep attributes were not described (Cheuk et al., 2007). The reasons for the absence of improvement in self-reported insomnia in three studies are not known, but these differences may be due to differences in patient populations and choice of outcome measures (Cheuk et al., 2007). Although acupuncture may improve insomnia and specific characteristics of sleep, more rigorous randomized controlled trials are needed. While individual results vary, acupuncture treatments appear to be well tolerated and have few adverse effects. The most frequently reported side effects are pain and erythema at the sites of needle insertion. In future studies, more rigorous randomization of study participations, standardized procedures for "sham" acupuncture control groups, and the inclusion of intent-to-treat analysis are warranted.

Mind–Body Modalities

Mind–body modalities are practices that focus on the interrelationships between the brain, mind, body, and behavior, with the intent to use the mind to change physiology and promote health (NCCAM, 2010f). Many mind–body interventions are considered to be complementary and alternative, but others, such as cognitive behavioral therapy and support groups, have entered the mainstream of health care. Cognitive–behavioral therapy for insomnia (CBT-I) is efficacious and is now a first-line treatment for insomnia (Schutte-Rodin, Broch, Buysse, Dorsey, & Sateia, 2008). Because CBT-I is discussed in detail in Chapter 6, it is not addressed here. Mindfulness mediation and music have also been used to promote sleep.

Mindfulness Meditation

Meditation includes a variety of techniques or practices intended to focus or control attention. Mindfulness meditation or mindfulness-based stress reduction (MBSR), an often used

technique, is used to reframe and reduce negative thought processes that might otherwise escalate into stress reactivity (Bishop, 2002). Practitioners of this method believe that it improves insomnia through its beneficial effects on relaxation and reductions in arousal. Several studies and two systematic reviews documented improvements in sleep quality, SOL, TST, and sleep disturbances from use of MBSR (Klatt, Buckworth, & Malarkey, 2009; Winbush, Gross, & Kreitzer, 2007; Yook et al., 2008). Although two studies reported that increased practice of MBSR was associated with greater improvements in sleep, one study found no association (Smith, Richardson, Hoffman, & Pilkington, 2005; Winbush et al., 2007).

Given the generally promising results of the available studies, nurses could recommend mindfulness meditation (MBSR) to patients as a supportive therapy to help manage sleep problems. Nurses could take courses to learn MBSR for their own use, and further training is available to learn to teach MBSR. One advantage in using MBSR to help improve sleep is that once trained, patients can use this without continued appointments with a MBSR trainer, making it a cost-effective modality. None of the studies reported adverse effects after using MBSR.

Expressive Arts—Music

Because of music's ability to contribute to a calm environment and a relaxing state, music may be effective in reducing psychological presleep arousal and may be helpful in shortening SOL. In a recent meta-analysis of the efficacy of music-assisted relaxation to improve sleep quality in adults and older adults, data from five randomized, controlled trials showed that music-assisted relaxation had a moderate effect on sleep quality of patients with sleep complaints (de Niet, Tiemens, Lendemeijer, & Hutschemaekers, 2009). Music-assisted relaxation improved sleep latency and sleep efficiency in patients who had schizophrenia (Bloch et al., 2010). On the other hand, the effects of music on sleep (longer SOL, more WASO, and decrements in sleep efficiency) (Lazic & Ogilvie, 2007) were found to be inferior to the use of auditory tones. While the findings regarding the efficacy of music to improve sleep parameters are mixed, no studies report adverse effects. Most often, the type of music that is employed in studies of music as an intervention for sleep is based upon patient preference. While most persons report that slow, rhythmic music is relaxing, the effect is highly individual. Because the use of music is generally low cost and safe, nurses should recommend this modality to persons who complain of difficulty with falling asleep. Music may be useful as an adjunctive therapy in combination with other non-pharmacological and pharmacological therapies for treatment of insomnia. However, further study of these combined treatments is needed.

Biological-Based Products

Herbs and Natural Substances

Herbs and natural substances are found in nature (NCCAM, 2010f). Several herbs, including kava, chamomile, lavender, and St. John's Wort have been used to treat insomnia, but none has proven efficacy (NCCAM, 2010d). Although one placebo-controlled trial found that kava improved subjective ratings of sleep quality (Lehrl, 2004), the herb that has been most rigorously examined is extract of valerian. The natural product that has been studied the most in relationship to sleep is melatonin.

Valerian

Extract of valerian (*Valeriana officinalis*) has been used to treat insomnia symptoms in Europe and in the United States. Approximately 1% of U.S. adults used valerian within the past week (Barnes, Powell-Griner, McFann, & Nahin, 2004). Although valerian is safe and has only rare adverse effects (mild dizziness, headache, drowsiness, nausea, diarrhea), its efficacy for insomnia treatment is unproven (Bent, Padula, Moore, Patterson, & Mehling, 2006; Fernandez-San-Martin et al., 2010; Taibi, Landis, Petry, & Vitiello, 2007). For example, there were no statistically significant improvements in subjective or objective sleep characteristics in participants who used valerian, compared with placebo, in

a recent randomized, clinical trial with a crossover design (Taibi et al., 2009).

Limitations of many of the valerian studies include wide variation in valerian protocols (e.g., type of valerian preparation, dose, and length of treatment), limited use of randomized controlled designs, small samples, and failure to use intent-to-treat analytic strategies. Although six studies reported significant improvement (relative risk of improved sleep was about 1.8%), there was evidence of publication bias (Bent et al., 2006).

Unlike the studies in persons with insomnia that report nonsignificant results, one small study ($n = 37$) found improvements in subjective daytime sleepiness (Cuellar & Ratcliffe, 2009) and restless legs syndrome (RLS), but both the experimental and placebo groups improved on sleep quality. Further study of valerian in the treatment of RLS is warranted.

Aromatherapy

Aromatherapy is the therapeutic use of inhaled essential oils from flowers, herbs, or trees to promote health and well-being (NCCAM, 2010). Usually the oils are used in oil burners, in bath water, or are gently massaged into the skin, where the oil evaporates and stimulates the olfactory sense (Basch et al., 2004). Although little is known about the neurophysiological mechanisms for the effects of aromatherapy, there is some expert consensus that aromatherapy stimulates the limbic system and leads to relaxation and mood enhancement.

Despite the growing popularity of aromatherapy as a sleep-promoting modality, research on its efficacy is limited, with trials of lavender being most frequently undertaken. Participants who inhaled lavender continuously overnight reported improvements in self-reported insomnia symptoms, compared with participants in the control group who inhaled sweet almond oil (Lewith, Godfrey, & Prescott, 2005). However, lavender oil combined with massage was no more effective than massage alone in improving sleep of cancer patients in a hospice (Soden, Vincent, Craske, Lucas, & Ashley, 2004b), and use of lavender as aromatherapy did not improve sleep in dementia patients (Holomes & Ballard, 2004). Definitive conclusions about the effects of aromatherapy on sleep cannot be made.

Melatonin

Melatonin, a neurohormone produced by the human pineal gland, is isolated from the pineal glands of beef cattle or chemically synthesized (Buscemi et al., 2006). Data on the efficacy of melatonin for treatment of sleep disorders are mixed, with results depending on the nature of the sleep disorder (Buscemi et al., 2004, 2006). There were clinically significant improvements only in SOL in persons with delayed sleep phase syndrome and in TST in persons with circadian rhythm disorders (i.e., shift-work or jet lag) (Buscemi et al., 2004). Three small studies did not find any clinically important differences in sleep in the melatonin groups compared to placebo groups in either persons with Alzheimer's or Parkinson's (Dowling et al., 2005; Dowling et al., 2008; Gehrman et al., 2009). (See Chapter 10, Circadian Rhythm Disorders, and Chapter 6, Insomnia.) Side effects were rare and mild. Therefore, melatonin appears to be safe with short-term use (3 months or less). Because the results of clinical trials have been mixed regarding the efficacy of melatonin to relieve sleep disturbances, nurses should recommend the use of melatonin for select cases of persons who are suffering from circadian rhythm disturbances. (See Chapter 6, Insomnia.)

Manipulative and Body-Based Modalities

Massage therapy encompasses many different techniques. In general, therapists press, rub, and/or otherwise manipulate the muscles and other soft tissues of the body (NCCAM, 2010c). There are several types of massage: (1) Swedish massage (use of long strokes, kneading, deep circular movements, vibration, and tapping); (2) deep tissue massage, focused on manipulation of the deep muscles and connective tissues; and (3) reflexology, in which the practitioner applies pressure to the feet (or sometimes the hands or ears), to promote relaxation or healing in other parts of the body (NCCAM, 2010c).

Self-reported sleep quality improved with massage in five controlled studies (Field, Hernandez-Reif, Diego, & Fraser, 2007; Hernandez-Reif et al., 2005; Lawler & Cameron, 2006; Richards, 1998; Soden, Vincent, Craske, Lucas, & Ashley, 2004a). These studies included a variety of patient populations (e.g., persons with low back pain or migraines, critically ill patients, and hospice patients who reported insomnia). In the only study that employed polysomnography to examine the effects of massage in hospitalized male cardiac patients, Richards found improvements in TST and sleep efficiency (14.7%), compared to a control group who received visualization and imagery (1998). The studies of positive effects of massage contrasted with a study in which there were no statistically significant effects (Smith, Kemp, Hemphill, & Vojir, 2002), but in the latter study, the massage group had less deterioration in sleep compared to the control group in hospitalized cancer patients. It is possible that the lack of improvement in sleep quality may be related to the lower dose of the massage and the relative acuity of the hospitalized cancer participants when compared with other studies with significant results.

Given that most studies demonstrated generally positive sleep outcomes after use of massage, nurses could use therapeutic massage with their patients and recommend it as supportive therapy to help manage sleep problems. None of the studies reported any side effects from the use of massage; thus, therapeutic massage performed by trained nurses or massage therapists appears to be safe.

Tai Chi and Qigong

Tai chi is called moving meditation because Tai Chi practitioners move their bodies slowly, gently, and with awareness, while breathing deeply (NCCAM, 2010e). Tai Chi incorporates the Chinese concepts of yin and yang (opposing yet complementary forces described in traditional Chinese medicine). A major tenant of Chinese medicine is that health is achieved through balancing yin and yang; disease is caused by an imbalance leading to a blockage in the flow of qi (vital energy or life force). Practicing Tai Chi is said to support a healthy balance of yin and yang, thereby aiding the flow of qi (see http://nccam.nih.gov/health/taichi/).

Tai Chi improved self-reported sleep quality, duration, efficiency, and sleep disturbances in older adults who had sleep problems and in persons with fibromyalgia who had insomnia (Caldwell, Harrison, Adams, & Triplett, 2009; Chen, Hsu, Chen, & Tseng, 2007; Chen et al., 2009; Irwin, Olmstead, & Motivala, 2008). The effects of Tai Chi were superior to the effects of health education and low-impact exercise on the above sleep problems (Irwin et al., 2008). Tai Chi was as effective as Pilates in college students (Caldwell et al., 2009; Chen et al., 2007). However, Tai Chi did not improve sleep quality in older adults who were already good sleepers prior to beginning Tai Chi (Chen et al., 2007).

Qigong is a traditional Chinese mind/body exercise that uses slow and precise body movements with controlled breathing and mental focusing to improve health by improving the flow of "qi" or life force. Among patients with insomnia, 8.7% reported that Qigong improved their sleep (Lee et al., 2003); however, the investigators in this retrospective study did not report the type of qigong or the dose. Participants who performed Qigong in a randomized clinical trial (Manzaneque et al., 2004; Manzaneque et al., 2009) had slightly longer sleep duration, compared with those who had no treatment, but had no other improvements in sleep. It is premature to recommend Qigong as a treatment for sleep problems due to the paucity of research on this modality.

Given the promising results of available studies and the absence of adverse effects, nurses could recommend Tai Chi to patients as a supportive therapy to help manage sleep problems. Nurses could take Tai Chi courses to learn the technique for their own use, and further training is available to learn to teach Tai Chi. Once trained, patients can use Tai Chi at home. Therefore, Tai Chi is a cost-effective modality.

Yoga

Yoga is steadily gaining popularity in the United States. The various styles of yoga used for health

purposes combine physical postures, breathing techniques, and meditation, or relaxation (NCCAM, 2010g). Hatha yoga, the most commonly practiced type of yoga in the United States and Europe, emphasizes postures (*asanas*) and breathing exercises (*pranayama*). Hatha yoga incorporates various styles, including Iyengar, Ashtanga, Vini, Kundalini, and Bikram yoga.

Investigators examined the effects of yoga on sleep in people with chronic illnesses, including cancer (Carson, Carson, Porter, Keefe, & Seewaldt, 2009; Cohen, Warneke, Fouladi, Rodriguez, & Chaoul-Reich, 2004), hemodialysis (Yurtkuran, Alp, Yurtkuran, & Dilek, 2007), and insomnia (Khalsa, 2004); potential sleep problems related to life span (older adults [Chen & Tseng, 2008; Chen et al., 2009; Manjunath & Telles, 2005]; pregnant women in the second trimester [Beddoe, Lee, Weiss, Kennedy, & Yang, 2010]; and healthy individuals [Sulekha, Thennarasu, Vedamurthachar, Raju, & Kutty, 2006; Vera et al., 2009]).

The majority of investigators reported improvements in self-reported sleep disturbances in patients who used yoga (Beddoe et al., 2010; Carson et al., 2009; Chen & Tseng, 2008; Chen et al., 2009; Cohen et al., 2004; Vera et al., 2009; Yurtkuran et al., 2007). Some studies reported improvements in sleep quality, efficiency, duration, and latency after yoga participation (Chen et al., 2009; Cohen et al., 2004; Khalsa, 2004; Manjunath & Telles, 2005). In one study that included polysomnographic measures of sleep, long-term yoga practitioners had decreased REM latency and more slow-wave sleep (SWS) than age-matched controls who did not practice yoga (Sulekha et al., 2006).

There is promising evidence for the efficacy of yoga in improving self-reported and objective measures of sleep, and there have been no reported adverse effects. Thus, nurses could recommend yoga to patients as a supportive therapy to help manage sleep problems and may undergo training to teach this modality.

Energy Modalities

Healing Touch

Healing touch utilizes the hands to clear, energize, and balance human and environmental energy fields (King, 2005). Very few studies have investigated the effects of healing touch on sleep parameters and available studies are of poor quality. Two studies reported significant decreases in self-reported sleep disturbances after healing touch in persons with musculoskeletal disorders (Weze, Leathard, & Stevens, 2004) and in persons with cancer (Weze, Leathard, Grange, Tiplady, & Stevens, 2004). However, another study (Danhauer, Tooze, Holder, Miller, & Jesse, 2008) found no differences in sleep quality in hospitalized patients who had leukemia. The use of healing touch for improving sleep cannot be recommended at this time due to the paucity of research studies and the generally poor quality of the studies available.

Bioelectromagnetic-Based Modalities

Pulsed Electromagnetic Fields

Pulsed electromagnetic fields (PEMFs) are generated by electricity flowing through wire coils and produce magnetic fields. Magnetic fields are thought to influence the polarity and energy of cell membranes and facilitate healing of damaged cells. Low strength PEMFs are less than the Earth's magnetic field, which is about 0.5 G, and much weaker than the magnets used for MRI machines (approximately 15,000 G or higher) (NCCAM, 2010b). The premise of PEMFs is that the human body adapts to magnetic fields that are static, but the body does not adapt to the variability in field strength of PEMFs. Therefore, PEMFs may lead to greater physiological change than static magnetic fields.

Very few studies have investigated the effects of PEMFs on sleep. One study found improvements in WASO and non-rapid eye movement sleep (NREM), but SOL was unchanged (Borbely et al., 1999). Other studies have reported negative results (Graham, Sastre, Cook, & Gerkovich, 2000; Wrobel et al., 2008). Because most of the studies are of low quality, it is premature to draw any conclusions on the effects of PEMFs on sleep.

Cranial Electrical Stimulation

Practitioners of cranial electrical stimulation (CES) deliver low levels of electrical stimulation

to the brain. CES devices are about the size of MP3 players and clip to the earlobes. Although the precise mechanism of action for CES is unknown, animal models suggest that CES may inhibit the reuptake of norepinephrine, serotonin, and dopamine (Shealey, Cady, Culver-Veehoff, Cox, 1998). Because the electrical current is not perceptible, it is feasible to use sham (placebo) CES in randomized, controlled trials. Use of the Alpha-Stim, (Electromedical Products International, Inc.), a commercially available device that delivers a very low level of electrical stimulation (usually 100 microamps), reportedly improved sleep in patients with a variety of diagnoses, including insomnia and fibromyalgia (Cork et al., 2004; Lichtbroun, Raicer, & Smith, 2001; Tyers & Smith, 2001). However, the Alpha-Stim did not improve the sleep of caregivers of persons with dementia (Rose, Taylor, & Bourguignon, 2009). At the current time, the use of CES for improving sleep cannot be recommended due to the scarcity of research studies and the generally poor quality of the studies that are available.

SUMMARY AND IMPLICATIONS FOR NURSING PRACTICE

CAM-related modalities are widely available, and use of these treatments accounts for more than 33.9 billion dollars in expenditures (Nahim, Barnes, Stussman, & Bloom, 2009). However, convincing evidence in support of their use as efficacious treatments for sleep is sparse. Several CAM modalities, including acupuncture, yoga, massage, Tai Chi, and mindfulness meditation, show promise of improving some aspects of sleep, such as sleep quality, duration, latency, WASO, and self-reported sleep disturbance. These treatments are safe and cost-effective. Well-designed, randomized, and controlled trials are needed to further support the use of these modalities.

Many people use herbs and other natural substances for sleep and other conditions, regardless of the absence of high quality evidence in support of their use. It is important to carefully assess patients for the use of these substances, their potential side effects, and interactions with prescribed and over-the-counter medications. Due to the absence of FDA regulation of these products, they may contain potentially harmful ingredients that are not listed on the label. Most herbs and natural substances have not been tested in pregnant women, nursing mothers, or children, and the use of these substances in these patient populations is not recommended.

Resources and products are available to support nurses and other health care providers in providing CAM treatments (see Table 14.2). However, treatment with CAM modalities is often not reimbursable by third-party payors.

Table 14.2 ■ *Internet Resources on CAM Products and Practices*

Resource	Description
http://nccam.nih.gov (National Center on Complementary and Alternative Medicine); 1-888-644-6226	Includes data on all aspects of CAM for providers and lay persons.
http://www.herbs.org (Herb Research Foundation)	Information on use of herbs.
http://www.herbalgram.org (American Botanical Council)	Information on use of herbs.
http://naturalstandard.com	Database on all aspects of CAM; accessed by subscription only.
http://ods.od.nih.gov (NIH Office of Dietary Supplements)	Information on dietary supplements.
http://www.quackwatch.com	Information on quackery, fads, questionable therapies.
http://www.ncahf.org (National Council Against Health Fraud)	Information of health misinformation, fraud, and quackery.
http://www.consumerlab.com	Identifies best quality health and nutritional products through independent testing.

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Racial/Ethnic Health Disparities and Sleep Disorders

15

Carol M. Baldwin and Luxana Reynaga Ornelas

O sleep, O gentle sleep, nature's soft nurse . . .
—Wm. Shakespeare

Disturbed sleep is associated with increased health care utilization and costs, risks to public safety, reduced quality of life, and morbidity and mortality in the general population (Institute of Medicine [IOM], 2006; Ram, Seirawan, Kumar, & Clark, 2009). Evidence is emerging that suggests that some sleep disorders may be more prevalent among racial and ethnic minority groups, and these disorders may be associated with excess disease burden. Whether the risk for sleep disorders and their negative consequences is due to ethnic or racial group membership per se, or health disparities associated with these groups, is not clear and research is underway. Nevertheless, an understanding of extant data informs the perspectives of nurses working with people from these groups, assists in identifying health disparities, and may focus sleep assessment and guide culturally relevant sleep promotion. The purpose of this chapter is to review data related to racial and ethnic health disparities in sleep disorders and to discuss the implications for nursing.

This chapter focuses on American populations for whom there is available data on sleep, which includes Latino, African, and Asian. The terms of use here—"African American," "Asian-American," and "Latino/Hispanic American"—refer to racial/ethnic residents of the United States unless otherwise described. The term "Caucasian" refers to Non-Hispanic White people.

For comparative purposes, the Caucasian population represented 66% of the total United States population in 2007 and was older than the overall population, with a median age of 40.8 compared to 36.6 years (U.S. Census Bureau, 2008). Other ethnic groups include American Indian/Native American and Alaska Natives. These populations rose by 1% between 2006 and 2007 with a median age of 30.3 years for both groups (U.S. Census Bureau, 2008). They are the largest minority groups in Alaska, Montana, North and South Dakota. Although these groups also suffer from health disparities (See Froese et al., 2008; Villanueva, Buchanan, Yee, & Grunstein, 2005), there is insufficient information available on sleep disorders to draw valid conclusions to report here. Recommendations for future practice and research address the broader issues related to culture and racial/ethnic health disparities in sleep disorders and their treatment.

HEALTH DISPARITIES AND SLEEP DISORDERS AMONG LATINO AMERICANS

Demography of Latino Americans

Latinos are the fastest growing minority group in the United States and represent at least 20 different countries from Latin America and the Caribbean; 40% were born outside the United States (U.S. Census Bureau, 2004). The term "Hispanic," used in the U.S. Census since 1980 (Gibson & Jung, 2002), suggests direct lineage from Spain. "Latino" is now the preferred descriptor for persons from Latin American countries who have Spanish ancestry. Subgroups include Mexican, Cuban, Puerto Rican, Colombian, Dominican, Salvadoran, and

Spanish-Americans, among others. In 1998, there were 30 million persons of Latino heritage representing 11% of the population. In 2007, the population reached 45.5 million or 15% of the total U.S. population of 301.6 million (U.S. Census Bureau, 2008). Nearly 34% of this population was younger than 18 years of age, compared to 25% of the total population (U.S. Census Bureau, 2008). By the year 2050, Latinos will number 97 million and constitute 25% of the U.S. population, with Mexican Americans representing the largest subgroup (64.3%) of the Latino population (U.S. Census Bureau, 1996). The increasing size of the Latino-American population underscores the significance of understanding the prevalence and impact of sleep disorders in general here and for their subgroups.

Sleep Disorders Among Latino Americans

Sleep is the best cure for waking troubles.
—Miguel de Cervantes

Studies of the prevalence of sleep disorders or comorbid conditions among United States Spanish-speaking Latinos are only beginning to emerge. However, there is some evidence that Latinos are at higher risk for several sleep disorders, and these sleep disorders may increase the burden of chronic conditions, such as diabetes, cardiovascular disease, substance abuse, and depression—problems for which Latino Americans are known to be at particularly high risk.

Sleep-Disordered Breathing

The true prevalence of sleep-disordered breathing among Latinos is not known, but increased rates of overweight and obesity and use of alcohol and tobacco associated with acculturation to the United States may contribute to its development (Loredo et al., 2010). (See Chapter 7, Sleep-Related Breathing Disorders.)

An early racial/ethnic study of sleep-disordered breathing estimated that 16.3% of Latinos and other racial minorities had ≥20 oxyhemoglobin desaturation events per hour of sleep (a sign of moderate or higher sleep-disordered breathing, as assessed with home monitoring) compared to 4.9% of middle-aged Caucasians; similar to other groups, predictors for sleep-disordered breathing were body mass index, age, and male sex (Kripke et al., 1997). In another study, nearly 15% of the Latino participants had apnea-hypopnea indices (AHI) ≥ 5, suggesting that they had at least mild sleep-disordered breathing (Mahmood et al., 2009).

The 10-year-long multi center Sleep Heart Health Study of adults 40 years of age and older (n = 6,441) demonstrated that Latino men and women had substantially higher odds of snoring, a focal sign of sleep apnea, compared to their Caucasian counterparts, but did not show other characteristics of sleep-disordered breathing (O' Connor et al., 2003). These findings cannot be generalized to all Latinos because participants, primarily of Mexican heritage, were English-speaking and had higher educational levels than Mexican Americans in the general U.S. population. However, the snoring rate for Latinos in the Sleep Heart Health Study replicated findings from a population-based survey of New Mexico Hispanics (Schmidt-Nowara, Coultas, Wiggins, Skipper, & Samet, 1990).

Parental reports of snoring were more commonly reported for Mexican American children between the ages of 4 and 11 compared to Caucasian children in the Tucson Children's Assessment of Sleep Apnea (TuCASA) study (Goodwin et al., 2003). Latino children in the TuCASA study also showed less Stage N3 sleep and more Stage N2 sleep (Quan et al., 2003). Although the reasons for these differences in sleep architecture are not known, they may be explained by snoring, a sign of sleep-disordered breathing.

The potential importance of sleep-disordered breathing among Latinos is underscored by the excessive burden of diabetes, cardiovascular disease, and obesity in these groups. For example, diabetes is nearly twice as common in Mexican Americans compared to Caucasians with documented rates of 8.2% for Cubans, 11.9% for Mexican Americans and 12.6% for Puerto Ricans (National Diabetes Information Clearinghouse, 2007). Untreated or undiagnosed

hypertension is also more common among Mexican Americans than Caucasians or African Americans (Harris, 2001), and cardiovascular disease is the leading cause of death. Evidence that an AHI of >20 was associated with higher rates of diabetes among Latinos than Caucasians suggests that sleep-disordered breathing may confer excess risk for the disorder (Surani, Aguillar, Komari, Surani, & Subramanian, 2009).

High rates of obesity among Latinos and the powerful association between obesity, sleep-disordered breathing, diabetes, and cardiovascular disease found in the general population suggest that obesity may be an important part of the causal pathway for each of these chronic condition conditions. (See Chapter 7, Sleep-Related Breathing Disorders.) The extent of excess risk conferred by Latino-American ethnicity beyond those racial/ethnic groups with similar degrees of obesity or overweight is not known. Two large NIH-funded studies are underway to examine these and other sleep-related questions among Latino Americans (Loredo et al., 2010).

Sleep Architecture

Rapid eye movement (REM) density (more frequent rapid eye movement), a marker of depression, was noted to be higher in Latino adults (Rao et al., 1999) and Mexican American adolescents, particularly females, compared to other racial and ethnic groups, after controlling for relevant variables (Rao, Hammen, & Poland, 2009). Although the clinical implications of increased REM among this small group ($n = 19$) are not certain, epidemiological studies found associations between REM density and psychiatric symptomatology (Breslau, Roth, Rosenthal, & Andreski, 1996; Buysse et al., 2008). Additional research is needed to evaluate whether REM density among Latinos is associated with increased rates of depression. Given the dearth of available objective sleep data for Latino adults and children, further studies are also needed to examine relationships between sleep architecture and comorbid conditions.

Insomnia

In several studies, insomnia was more common among Latinas than women of other ethnic backgrounds. As many as 73% of Dominicans, compared with 34% of English-speaking Caribbeans and 33% of Haitians, reported insomnia (Jean-Louis et al., 2008). (See Chapter 6, Insomnia.) Latinos, mostly of Mexican heritage, were more likely to report insomnia (36%) compared to Caucasians (30%) and African Americans (28%), as reported in data obtained in the Sleep Heart Health Study. Latinos with frequent snoring, insomnia symptoms, or daytime sleepiness had significantly poorer mental health compared to Caucasians (Baldwin et al., 2010).

Latinas who were postmenopausal reported less sleep and more depression compared to Caucasian women (Kripke et al., 2004), and investigators for the Study of Women's Health across the Nation (SWAN) found that Latinas reported less total sleep and were twice as likely to report depression as Caucasians or Asians (Hall et al., 2009). Although the reasons for the higher rates of insomnia in Latinas are not completely known, stressors associated with acculturation and socioeconomic status may play a role (Baldwin et al., 2009; Loredo et al., 2010).

It is possible that sleep disorders, including insomnia, may increase the already high rates of mental health disorders, particularly anxiety and depression, found among U.S. born Mexican Americans (Cabassa, Zayas, & Hansen, 2006; Escobar, Hoyos, Nervi, & Gara, 2000). The finding that depression predicted an increased incidence of adverse health outcomes in older Mexican Americans with type 2 diabetes (Black, Markides, & Ray, 2003), coupled with strong evidence of the bidirectional relationship between depression and insomnia, suggests the complex interrelationships among these conditions (Szklo-Coxe, Young, Peppard, Finn, & Benca, 2010).

Little is known about cultural perspectives on insomnia or the meaning of sleep among people from diverse cultures, especially those whose primary language is not English, which makes assessment and treatment of insomnia

challenging for English-speaking health professionals. One extant qualitative study suggests that Latino elders in Texas considered sleep problems as a normal part of aging (expressing a fatalistic view) and were less likely to seek health care in the prior year, while elderly Latinos who felt nothing could be done to treat sleep problems (expressing a nihilistic view) were less likely to have a primary provider (Goodwin, Black, & Satish, 1999). Findings from this work and the paucity of studies regarding cultural meanings highlight the need for such research in order to enhance sleep interventions that are sensitive to health beliefs among racial/ethnic groups.

Sleep Duration

Both short and long sleep duration are risk factors for negative health outcomes. (See Chapter 4, The Nature of Sleep Disorders and Their Impact.) Data from the cross-sectional National Health Interview Survey (Hale & Do, 2007) showed non-Mexican Latinos to have increased odds of being short sleepers compared to Caucasians after adjusting for socioeconomic status, health, and urban living. Mexican Americans had higher odds of long sleeping compared to Caucasians, although this relationship was not statistically significant after adjusting for socioeconomic status. The variables associated with long sleep included diabetes, cardiovascular disease, depression, and limitations in activity. The 2005–2006 National Health and Nutrition Examination Survey (n = 6,139) showed Latinos and Caucasians to have longer sleep durations than African Americans (Ram et al., 2009). These findings are similar to those of Krueger and Friedman (2009), who reported short sleep durations for Latino "Other," and long sleep durations for Mexican Americans. Latinos were slightly less likely than Caucasians to have short sleep duration at baseline in 1965 (12%; CI = 9–15), but were more likely than Caucasians to have short sleep (37%; CI = 28–47) at follow-up in 1999 (Stamatakis, Kaplan, & Roberts, 2007). The finding that Mexican Americans born in the United States were 40% more likely to be short sleepers compared to Mexican immigrants suggests that stress, smoking, and other factors associated with acculturation may contribute to their rates of short sleep (Hale & Rivero-Fuentes, 2009). Causality cannot be inferred from these cross-sectional studies; future studies need to address design issues that include lifestyle and other factors known to influence sleep duration and its consequences. However, these findings suggest that sleep duration may be an important health-related risk factor among subgroups of Latinos.

Restless Legs Syndrome

There are few data regarding the prevalence of restless legs syndrome among Latino populations (Baldwin, Mays, Márquez-Gamiño, Caudillo-Cisneros, & Quan, 2008; Baldwin et al., 2009). Extant data does suggest that prevalence here is somewhat lower among Latinos (0.1%) compared to African Americans (0.4%) and Caucasians (0.5%); however, these rates are based on physician-diagnosis and may not fully reflect the true prevalence of restless legs syndrome because of potential differences in access to care, language, subjective interpretation of the survey questions, socioeconomic status, and other factors (Ram et al., 2009).

Excessive Daytime Sleepiness

There is a paucity of data regarding daytime somnolence for Latino Americans. Nonetheless, in their study of Latinos residing in New Mexico, Schmidt-Nowara et al. (1990) reported an association between snoring and daytime sleepiness that suggested a greater occurrence of upper airway obstruction. Latinos who reported greater daytime somnolence in the Sleep Heart Health Study showed significantly poorer mental health compared to Caucasian American participants (Baldwin et al., 2010). These findings warrant further investigation to determine causes and consequences of excessive daytime sleepiness within and between Latino subgroups in the United States.

Disparities and Sleep Risk Among Latino Americans

There is ample documentation that Latino Americans often suffer from limited access to health care, poorer health, and high rates of stress due to higher rates of unemployment, lower income and education levels, more food insecurity, lower levels of leisure-time physical activity, and less access to transportation compared to Caucasian Americans (DuBard & Gizlice, 2008; Escarce, Morales, & Rumbaut, 2006; Fitzgerald, Damio, Segura-Pérez, & Pérez Escamilla, 2008). These sources of stress are likely to contribute to the development of insomnia and may partially explain high levels of depression. Dietary consumption and sedentary lifestyles contribute to obesity and the risk of sleep-disordered breathing.

Language barriers play a significant role in health disparities for the 45 million Latinos whose primary or second language is Spanish (Agency for Healthcare Research and Quality, 2006). There is an ongoing need for linguistically relevant, translated, validated, and psychometrically sound Spanish-language sleep assessment tools (Baldwin et al., 2008; 2009) for clinical and research purposes. Latino Americans are also less likely to have health insurance (Harris, 2001) and more likely to mistrust the health care system. Given the high costs of sleep screening and treatment in specialized sleep disorders center, health insurance and language issues are significant barriers to addressing sleep disorders in this population.

Raising awareness about the implications of sleep disorders among Latino Americans is an important goal, as demonstrated by the emphasis in *Healthy People 2010*. Rates of recognition for sleep disorders in community-based primary care settings have traditionally been low compared to the estimated population prevalence, especially among Latino Americans (Rosen, Zozula, Jahn, & Carson, 2001). Knowledge of health and sleep disparities that confront Latinos in the United States may contribute in important ways to the identification, evaluation, and treatment of sleep disorders in the Latino population. The paucity of information specific to sleep among Latinos underscores the continued need to raise awareness among health care providers, including nurses, of the importance of sleep assessment. These efforts are ongoing, but more work is needed. Table 15.1 lists nursing implications resulting from the evidence regarding sleep disorders in Latino Americans.

Table 15.1 ■ *Nursing Implications Related to Sleep Pathology Identified Among Latinos*

- Include body habitus, duration and loudness of snoring, witnessed apneas during sleep, daytime sleepiness and drowsy driving in the nursing assessment
  - These signs may be especially important as proxy measures in patients who do not have health insurance.
  - Sleep apnea has been associated with diabetes, stroke, cardiovascular disease, hypertension and mood disorders (Baldwin & Quan, 2002; Gottlieb et al., 2005, 2006; Surani et al., 2009).
- Query Latino patients and parents of Latino children regarding frequency and loudness of snoring (Al-Delaimy et al., 2002; O'Connor et al., 2003; Schmidt-Nowara et al., 1990).
  - Snoring may be associated with restless sleep, daytime sleepiness, learning problems, and hyperactivity in school-age children (Goodwin et al., 2003; Quan et al., 2003).
- Include patient and family history of mood disorders in nursing history
  - Increased REM sleep and REM density have been linked with higher rates of depression and psychopathology in adults, adolescents and by parental history (Breslau et al., 1996; Buysse et al., 2008; Rao et al., 1999, 2009).
- Nursing assessment should include average hours of sleep to determine short and long sleepers in order to develop a plan of care that promotes sleep health.
  - Short sleep has been related to higher rates of obesity, diabetes, and insulin resistance while long sleep has been associated with cardiovascular disease, diabetes and limitations in physical and mental HR-QOL (Gottlieb et al., 2005, 2006; Ram et al., 2009).
- Consider educational level and limitations in translated measures; utilize translators if needed.
  - Few sleep measures have been adequately translated in a manner that addresses health and reading literacy among Spanish speakers (Baldwin et al., 2008, 2009).
  - Assess patients for use of folk practices (e.g., herbal remedies) and beliefs that could influence sleep disorders treatment.

HEALTH DISPARITIES AND SLEEP DISORDERS AMONG AFRICAN AMERICANS

Demography of African Americans

African Americans (single race or multiracial) are the second largest group in the United States at 40.7 million; this population increased by 1.3% between 2006 and 2007 (U.S. Census Bureau, 2008). Approximately 31% of the African American population was younger than 18 years compared to 25% of the total population (U.S. Census Bureau, 2008). Available data do not distinguish among groups categorized under the umbrella term *African American*. For example, subgroups include direct immigrants from Africa and those who are U.S. born. Likely variations in cultural orientation and acculturation factors between these groups are not acknowledged in the existing data.

Sleep Disorders Among African Americans

> *There are some nights when sleep plays coy, aloof and disdainful. And all the wiles that I employ to win its service to my side are useless as wounded pride, and much more painful.*
>
> —Maya Angelou "Insomniac"

The growing and substantive literature on sleep disorders among African Americans suggests important interactions between sleep disorders and prevalent chronic health conditions. This section provides a review of the most commonly identified sleep disorders in this population.

Sleep-Disordered Breathing

Studies of the prevalence of sleep-disordered breathing among African Americans are often conflicting. Prevalence rates of 4% in men and 2% in women for sleep-disordered breathing were similar among African Americans compared to other ethnic groups in the Sleep Heart Health Study (Young et al., 2002). Other researchers, however, reported higher sleep apnea severity in young and elderly African Americans after controlling for age, sex, obesity and familial clustering (Ancoli-Israel et al., 1995; Redline et al., 1997). Oxyhemoglobin desaturations were nearly three times as frequent among African Americans and other minorities compared to Caucasians, and body mass index was the most important predictor (Kripke et al., 1997). Frequent snoring was also more common among African American women compared to Caucasian women (O' Connor et al., 2003). These findings were consistent with results of a study of "probable" obstructive sleep apnea based on subjective symptoms and anatomic findings that suggested that sleep apnea may be more common in African Americans (Friedman et al., 2006).

Similar to the results of studies in other populations, African American patients with or without a family history of hypertension had higher respiratory disturbance indices than normotensive patients. Hypertensive patients with a positive history were more likely to have higher blood pressure, body mass index, and severe sleep apnea (Jean-Louis, Zizi, Casimir, DiPalma, & Mukherji, 2005).

Sleep Architecture

Several studies have shown that African American adults (Mezick et al., 2008; Redline et al., 2009) and adolescents (Rao et al, 2009), have a higher percentage of Stage N2 sleep (light stages that may contribute to feeling unrefreshed on arising) and lower percentages of Stage N3 (deep, restorative sleep) compared to their Caucasian and Latino American counterparts. Longer sleep time and more time in REM sleep have also been noted; however, findings on the degree to which these differences result in excessive daytime sleepiness are inconsistent (see below).

Failure to consider health disparities, including socioeconomic status, health literacy, and access to care issues may contribute to the inconsistent sleep findings of African Americans compared to Caucasians (Fiorentino, Marler, Stepnowski, Johnson, & Ancoli-Israel, 2006; Phillips & Manino, 2005; Profant, Ancoli-Israel, & Dimsdale, 2002). Differences in sleep architecture may also be due to genetic, psychosocial, environmental, behavioral, or stress-related factors (Basta, Chrousos, Vela-Bueno, & Vgontzas,

2007; Rao et al., 2009). Racial discrimination and stress may play a role. For example, participants who reported experiencing greater racial discrimination showed less Stage N3 sleep (Thomas, Ancoli-Israel, Bardwell, & Dimsdale, 2006).

Insomnia and Sleep Quality

African Americans reported more difficulty falling asleep, reduced sleep satisfaction, and frequent napping compared to Caucasians (Ancoli-Israel et al., 1995; Redline et al., 1997), and as many as 71% of urban-dwelling African American women (Jean-Louis et al., 2008) reported insomnia. As noted in a comparative epidemiological literature review, African Americans reported poorer sleep and sleep quality, longer sleep onset, more light and less deep sleep, napped more frequently, and exhibited increased risk factors for poor sleep, including higher body mass index (Durrence & Lichstein, 2006).

Insomnia is known to increase the likelihood for depression and anxiety. For example, people with insomnia had 9.82 and 17.35 times the risk for depression and anxiety, respectively, in a study that used empirically validated diagnostic criteria (Taylor, Lichstein, Durrence, Reidel, & Bush, 2005). African Americans were noted to be 3.4 and 4.8 times more likely to have clinically significant depression and anxiety respectively, and these mental health indices are consistent with a history of insomnia. African Americans with insomnia symptoms also reported significantly poorer physical and mental health related quality of life, compared to Caucasians and Latinos in data obtained from the Sleep Heart Health Study (Baldwin et al., 2010).

Sleep Duration

African Americans were significantly more likely to experience both short (≤5 hours) and long (≥9 hours) sleep, and less likely to report sleeping 7 hours per night compared to Caucasians (Nunes et al., 2008). These findings were consistent with another self-report study (Krueger & Friedman, 2009) and a study that utilized wrist actigraphy and in-home polysomnography (Mezick et al., 2008). The risk of short sleep doubled from 26% to 54% between 1965 and 1999 among African Americans, after adjusting for age; income and educational levels were also predictors (Stamatakis et al., 2007). Hale and Do (2007) reported that sleep durations were associated with increased mortality in African Americans. In contrast, Phillips and Mannino (2005) found that African American race was associated with reduced risk of sleep disturbances. Further studies are needed.

Restless Legs Syndrome and Periodic Limb Movements During Sleep

Investigators only recently included racial/ethnic groups in studies of restless legs syndrome or periodic limb movement disorder, and available data are conflicting. The prevalence of restless legs syndrome was 4.7% for African Americans and 3.8% for Caucasians in the Baltimore Health and Mental Health Study (Lee et al., 2006), in contrast to the prevalence rates were 0.4% and 0.5%, respectively in the 2005–2006 National Health and Nutrition Examination Survey (Ram et al., 2009). However, there were higher rates of periodic limb movements, a condition that often coexists with restless legs syndrome, among African Americans compared to Caucasians (Scofield, Roth, & Drake, 2008). Large multi-racial epidemiological studies should be undertaken to validate these findings and systematically examine factors known to be associated with restless legs syndrome, including genetics, peripheral neuropathy, history of depression, and iron levels to explain these differences and evaluate the consequences. (See Chapter 9, Narcolepsy.)

Excessive Daytime Sleepiness

Several studies have suggested that African Americans report higher rates of self-reported daytime sleepiness, as measured with the Epworth Sleepiness Scale (see Chapter 5, Conducting a Sleep Assessment) compared to Caucasians (Knutson, Rathouz, Yan, Liu, & Lauderdale, 2006; O'Connor et al., 2003).

Although African American participants in a normative population-based study had elevated Epworth scores compared to Caucasians, the effect sizes were small, and the Epworth scale did not discriminate between persons with and without insomnia (Sanford et al., 2006). Whether or not these apparent differences are due to true differences or differences in response to the Epworth Sleepiness Scale is not clear because researchers found that African Americans held different interpretations of the scale items than Caucasians (Hayes, Spilsbury, & Patel, 2009). Understanding the true extent of daytime sleepiness is important because it is associated with poorer physical and mental health-related quality of life (Baldwin et al., 2001; Baldwin et al., 2010). Little is known about differences in objective measures of daytime sleepiness (e.g., Multiple Sleep Latency Test) among African Americans.

Disparities and Sleep Risk Among African Americans

It is well established that socioeconomic (low income, low educational levels, no or underinsurance), environmental (e.g., crime, pollution and stress associated with living in urban environments), and lifestyle factors (e.g., diet, sedentary lifestyle) contribute to poor health outcomes among African Americans (Morenoff et al., 2007; Paschal, Lewis-Moss, Sly, & White, 2009). For example, the prevalence of cardiovascular disease, including hypertension, among non-Hispanic African Americans exceeds that of other racial/ethnic groups in the United States (Glover, Greenlund, Ayala, & Croft, 2005). African American adults are less likely to be diagnosed with coronary heart disease, but more likely to die from it (Office of Minority Health, 2008). In 2006, African Americans were over twice as likely to die from diabetes compared to Caucasians (Office of Minority Health, 2008). Overall, African Americans were 1.4 times as likely to be obese compared to Caucasians; 39% of African American women were overweight or obese—a rate higher than all other groups (Office of Minority Health, 2008). African American children between ages 6 and 17 years were 1.3 times as likely to be overweight compared to Caucasian children (Office of Minority Health, 2008). Although African American race alone was associated with reduced risk of sleep disturbances, health problems, socioeconomic status, and mood and unhealthful behaviors in this population increased the risk of sleep complaints (Phillips & Mannino, 2005). Taken together, these factors may escalate the risk of chronic conditions, including sleep disorders.

In order to build culturally relevant programs for assessment and treatment of sleep disorders, there is a need for better understanding of perceptions among African Americans about the relevance of sleep and sleep disorders to their lives, health and function (e.g., Goodwin et al., 1999). Community-based awareness campaigns are needed to educate residents about the risk of sleep disorders and to counter misconceptions. For example, African American bed partners were more likely to accept snoring (Friedman et al., 2006) than members of other ethnic/racial groups.

Levels of stress, mental health disorders, and substance abuse are high in urban environments where African Americans of low socioeconomic status often reside. These problems are intimately associated with sleep disorders among children and insomnia and mental health disorders in adults. (See Chapter 12, Sleep and Psychiatric Disorders.) For example, Caldwell and Redeker (2009) found that stress, anxiety, and depression were associated with poor sleep in minority women living in the inner city. There is a need for insomnia screening and access to affordable efficacious treatment for these individuals, who often are un- or under-insured. Working with parents to promote adequate sleep among children is an important priority and may have an impact on school and social performance. (See Chapter 2, Developmental Aspects of Normal Sleep.) Table 15.2 lists nursing implications relevant to sleep disorders identified in African Americans.

Table 15.2 ■ *Nursing Implications Related to Sleep Pathology Identified Among African Americans*

- Incorporate a focus on diet, nutrition, sedentary lifestyle, and other risk factors in nursing care plans
  - Body mass index was the leading predictor for sleep apnea among African Americans (Kripke et al., 1997).
- Assess for signs of sleep apnea (snoring, witnessed apneas, daytime sleepiness) in hypertensive African Americans; refer for specialized sleep evaluation
  - Hypertensive African Americans with or without a family history of hypertension are at risk for worse respiratory disturbance indexes (Jean-Louis et al., 2005)
- There is a need for culturally-responsive cognitive behavioral therapy for insomnia
  - African Americans may have high rates of insomnia, possibly reflected in their sleep architecture (more stage N2 and less stage N3-N4 sleep), comorbid depression and anxiety, and significantly poorer mental and physical HR-QOL (Baldwin et al., 2010; Redline et al., 1997; Taylor et al., 2005)
- Assess for extremes in sleep duration (long or short) in light of lifestyle behaviors, health problems, socioeconomic status, and mood
  - African Americans exhibit extremes in sleep duration that contribute to increased morbidity and mortality (Hale & Do, 2007; Krueger & Friedman, 2009; Nunes et al., 2008).
- Assess for daytime sleepiness to determine risk for accidents and difficulties with daytime performance
  - African Americans have reported greater daytime sleepiness compared to Caucasians that may be a result of short sleep, greater prevalence of sleep apnea, higher prevalence of comorbid conditions, or other factors.

HEALTH DISPARITIES AND SLEEP DISORDERS AMONG ASIAN AMERICANS

Demography of Asian Americans

Asian Americans are the second fastest growing minority group in the United States with growth of nearly 3% between 2006 and 2007 (U.S. Census Bureau, 2008). In the United States, over 4% of the population is of Asian origin. Chinese Americans comprise over 23%, followed by Philippinos (18%), Asian Indians (16%), Vietnamese (10.9%), and Koreans (10.6%) (U.S. Census Bureau, 2007). The number of people who self-identify as Asian or Asian in combination with one or more races will increase 153% between 2008 and 2050 compared to a 44% increase in the population as a whole (U.S. Census Bureau, 2009). Asians had a median age of 35.4 years, compared to 36.6 for the general U.S. population (U.S. Census Bureau, 2008). Of the Chinese Americans, 81% are foreign-born and 19% are born in the United States (U.S. Census Bureau, 2009). Chinese is second only to Spanish as the most widely spoken non-English language in the United States and, as of 2007, approximately 2.5 million people 5 years of age and older spoke Chinese at home (U.S. Census Bureau, 2009).

Sleep Disorders Among Asian Americans

The loss of one night's sleep is followed by 10 days of inconvenience.

—Asian proverb

A majority of studies that examined sleep problems of Asian populations were conducted outside the United States. In Asian countries, sleep disorders are not well recognized, awareness of sleep disorders by health professionals is low, and few sleep laboratories can deal with sleep disorders other than obstructive sleep apnea in China (Han, 2009).

Sleep-Disordered Breathing and Sleep Architecture

Among Chinese, Japanese and Korean and Caucasian patients, male gender was a significant risk factor for sleep-disordered breathing, while obesity was a less significant risk factor among Asians compared to Caucasians who had severity of the respiratory disturbance index (Li et al., 1999). Differences in cranio–facial morphology may explain the excess risk of sleep apnea in Asian men (Li et al., 1999; Li, Kushida, Powell, Riley, & Guilleminault, 2000). (See Chapter 7, Sleep-Related Breathing Disorders.) Asian adults residing in San Diego had three times more oxyhemoglobin desaturations than Caucasians; however, body mass index was the major predictor in this study (Kripke et al., 1997). Prevalence rates for self-reported snoring

among students attending a California college were 37% for Asians, 31% for Hispanics, 27% for Caucasians, and 24% for African Americans, although another 20% did not identify with any ethnic group (Patel et al., 2008). Snorers were significantly more likely to report a parental history of snoring and had higher body mass indices—signs that could represent early presentation of adult sleep apnea (Patel et al., 2008). These somewhat contradictory findings suggest that male gender and obesity play a role in sleep-disordered breathing among Asians, but in some persons, cranio–facial abnormalities may also contribute. (See Chapter 7, Sleep-Related Breathing Disorders.) Slow-wave sleep was notably reduced in midlife Chinese women compared to Caucasians in the Women's Health across the Nation (SWAN); however, this finding is considered preliminary and mechanisms are unclear (Hall et al., 2009).

Insomnia, Sleep Quality, and Excessive Daytime Sleepiness

The prevalence of insomnia among Japanese American men in the Honolulu Heart Study (32%) was similar to Caucasians and associated with depression, chronic health conditions, and benzodiazepine use; however, the prevalence of daytime sleepiness (8.9%) was similar to native Japanese but lower compared to older Caucasian adults (Babar et al., 2000). Sok (2008) reported that Korean-American immigrant older adults described sleep interruption and dissatisfaction with their sleep. Similar to other populations, poor sleep was associated with depression and comorbid illness among community-dwelling Chinese residents of Seattle (Hsu, 2001).

Disparities and Sleep Risk Among Asian Americans

Chronic health conditions associated with poor diet, obesity, and sedentary lifestyles are increasing among U.S. born versus immigrant Asians (Dey & Lucas, 2006; Lee et al., 2008; Mak & Zane, 2004). Acculturation seems to play an important role. For example, the prevalence of smoking, cardiovascular diseases, and hypertension are also higher for U.S. born compared to Chinese immigrant adults. Only 15% of Chinese immigrants consumed adequate fruits and vegetables, 31% engaged in regular exercise, and 21% of male respondents smoked (Taylor et al., 2007). The Behavioral Risk Factor Surveillance System reported strong associations between body mass index and risk for chronic health conditions among Asian-Americans 50 years and older that underscored the need for culturally appropriate health promotion messages, as well as research documenting health status and needs (Balluz, Okoro, & Mokdad, 2008).

Chinese and other Asian American groups have high rates of depression (Lee et al., 2008; Mak & Zane, 2004). Both depression and anxiety (Lubetkin, Jia, & Gold, 2003a) were noted among low-income immigrant Chinese Americans that were associated with generally poorer health-related quality of life (Lubetkin, Jia, & Gold, 2003b). Acculturative stress contributes to depression among Asian-Americans (Hwang & Ting, 2008), but the extent to which this increases the risk of poor sleep quality is not known. Because Chinese Americans may manifest depression via somatic complaints (Kung & Lu, 2008) that may include poor sleep, complaints of poor sleep may be cues to evaluate patients for mental health problems. (See Chapter 12, Sleep and Psychiatric Disorders.)

A major barrier to health care and treatment among immigrant populations is the inability of the majority of health care providers in the United States to query patients in their native language. Asian Americans with low English proficiency have limited access to health care or routine preventive services, report higher rates of medication complications, are less satisfied with clinical communication and overall health care, and oftentimes avoid care when ill (Green et al., 2005). There is no reason to believe that these limitations in health care encounters, communication and follow-up do not also extend to reporting sleep symptoms and obtaining evaluation, treatment and follow-up care for sleep disorders. Because foreign-born Asian Americans are less likely to be insured and less likely to have a usual source of care compared to their U.S. born counterparts

(Dey & Lucas, 2006), they may be less likely to be screened and treated for sleep disorders.

In addition to the need for availability of health care providers who can communicate in immigrant's native languages, there is a need for sleep questionnaires in patients' native languages. An example is the translation of the General Sleep Disturbance Scale (GSDS) for use with Chinese-American parents of hospitalized infants (Lee, 2007). The General Sleep Disturbance Scale showed acceptable reliability and validity for the total and subscale scores in Chinese-speaking parent pairs residing in the San Francisco area. Table 15.3 summarizes nursing implications related to sleep disorders of Asian-Americans.

HEALTH DISPARITIES AND SLEEP DISORDERS IN RESEARCH AND PRACTICE

Sleep disorders increase the risk for chronic conditions, such as diabetes, depression, and cardiovascular disease that, in turn, contribute to burgeoning health care costs. Sleep disorders are also associated with decrements in daytime performance in school and the work place. Taken together, the high rates of diabetes, hypertension, obesity, poor dietary intake, and sedentary lifestyle (Sharma, Malarcher, Giles, & Myers, 2004), as well as the performance-related effects of sleep disorders combine to make knowledge of culture and racial/ethnic aspects of sleep and sleep disorders a high priority for nurses in clinical and research settings.

There is a pressing need for better understanding of cultural differences in perceptions about sleep and sleep disorders and their impact, as well as culturally relevant and cost-effective strategies to assess and treat sleep disorders. For example, cognitive–behavioral therapy (CBT-I) is an efficacious treatment for insomnia. (See Chapter 6, Insomnia.) However, barriers to treatment may include language, lack of evidence of its cultural acceptability, and limited access due to limited trained providers and the cost of multiple sessions. There is a need for research on the effectiveness of this treatment in ethnically and racially diverse populations, therapists who can communicate in patients' native languages, and cost-effective ways to expand access in community based settings. Table 15.4 summarizes several pressing research priorities related to health disparities and sleep disorders.

Use of complementary and alternative (CAM) therapies and traditional folk practices to treat insomnia may also be desirable. For example, 25% of Korean-Americans are reported to have used them, and women preferred to use their traditional Korean health care practices (Sok, 2008). However, few of these treatments have been adequately studied (see Chapter 6, Insomnia and Chapter 14, Complementary and Alternative Medicine (CAM) and Sleep); some CAM therapies may not be compatible with other treatments. Therefore, evaluation of patients' perspectives about these treatments and their compatibility with other therapies should be considered.

Table 15.3 ■ *Nursing Implications Related to Sleep Pathology Identified Among Asian Americans*

- Incorporate evaluation of cranio-mandibular factors in assessing patients for risk of sleep-disordered breathing (See Chapter 7, Sleep-Related Breathing Disorders)
  - Asian Americans may have cranio-mandibular factors not necessarily seen in other racial/ethnic groups (Li et al., 1999, 2000). Obesity is not necessarily associated with sleep apnea among Asian American populations.
- Valid and reliable assessment measures for depression, anxiety and acculturation should be included in the nursing assessment of Asian Americans with presenting complaints of insomnia, unrefreshing or insufficient sleep (Hsu, 2001; Hwang & Ting, 2008; Kung & Lu, 2008).
- Consider Asian American client's educational level, availability of translated measures and translation resources obtainable in the clinical setting.
- Asian Americans may utilize traditional medical practices, including acupuncture and herbal remedies to manage their sleep problems. Nurses should become familiar with these practices and use them as potential adjuncts to sleep interventions.

Table 15.4 ■ *Implications for Further Research Related to Health Disparities and Cultural Issues Related to Sleep*

- Develop sleep-measurement instruments that are psychometrically sound, culturally appropriate, linguistically relevant; these measures must also account for literacy level.
- Conduct longitudinal and cross-sectional studies to examine associations between disturbed sleep, comorbid conditions, and sleep-related quality of life of racially and ethnically diverse populations with limited English proficiency across the life span.
- Incorporate evaluation of associations between acculturation, access to care, cultural racism, education level, income, urban/rural dwelling, region of the country, lifestyle factors, and other variables needed in studies of sleep in racially/ethnically diverse populations.
- Conduct qualitative studies related to cultural interpretations of the meaning of sleep and sleep disorders in order to develop and implement education and intervention protocols that are sensitivity to health beliefs.
- Develop and test, culturally responsive sleep health promotion and intervention protocols.
- Assess the extent to which the results studies of sleep disorders conducted in Asian populations outside of the United States are generalizable to Asian Americans.

The documented associations between obesity and sleep-disordered breathing, as well as diabetes and heart disease, suggest the importance of improvements in diet and exercise that prevent increased weight and reduce overweight and obesity if they occur. (See Chapter 7, Sleep-Related Breathing Disorders.) Although difficult to initiate and sustain, lifestyle change programs may have an important impact on multiple obesity-related outcomes, including the development of sleep apnea. While positive airway pressure is the most successful treatment, averting sleep apnea by preventing overweight and obesity is preferable and likely more cost-effective in the long run, albeit more difficult to accomplish. Therefore, there is a need for better access to community based and culturally relevant programs to address this problem.

Although few data are available on Native American populations regarding sleep, levels of obesity, diabetes, depression, and substance abuse are high, and access to health services is limited. Hence, these groups may also be at high risk for sleep disorders and their negative consequences.

Numerous population-based studies have identified associations between sleep disorders, quality of life, daytime performance (e.g., sleepiness, cognitive function, school and work performance), and morbidity and mortality. Other studies have addressed treatment outcomes for sleep disorders. Too few of these studies have included minority group members (especially members of lower socioeconomic status) in the proportion at which they are represented in the population, and even fewer have focused on understanding the specific needs of these groups.

Despite the need for further studies, extant research documents the powerful relationships between poor sleep and daytime function. Given the difficulties many African and Latino Americans of low socioeconomic status face in obtaining employment and succeeding in academics, it seems logical that sleep disorders assessment and treatment should have a high priority in schools, community-based centers, and the workplace. Cost-effective strategies are needed to assure their dissemination to populations at high risk for health disparities (see Unit III).

SUMMARY

Research findings on health disparities in sleep and sleep disorders and their consequences among racial/ethnic groups in the United States are emerging. Latino, African, and Asian-Americans may be at greater risk to have or to develop sleep disorders, including sleep-disordered breathing, insomnia, and short sleep duration than their Caucasian counterparts. Given that these groups are already at high risk for sleep-related comorbidity (e.g., hypertension, diabetes, obesity, and depression), sleep disorders may increase this risk. Risk for developing these comorbidities may be compounded by cultural barriers, language, acculturation, stress, low income, low education, and lack of health insurance. Well-designed studies are needed to adequately and appropriately evaluate the prevalence of sleep disorders and their

associations with morbidity, mortality, function, quality of life and to evaluate sleep disorders treatment outcome. These studies must employ culturally responsive self-report and objective measures. There is a particular need for studies of cultural perceptions about sleep disorders that can be used to develop treatments that are sensitive to health beliefs of target groups (e.g., Goodwin et al., 1999).

Available data suggest the need for an emphasis on assessment of sleep and its negative consequences among minority group members, especially persons who do not speak English, have low levels of education, or limited income. There is a particular need for culturally relevant translation of sleep questionnaires and diaries into languages that are understood by minority group members. Web sites with potential resources are listed in Table 15.5.

Table 15.5 ■ *Web-Based Resources*

Cross-Cultural Health Care—Case Studies (A Day in the Sleep Clinic): http://support.mchtraining.net/national_ccce/case5/home.html
Harvard Sleep and Health Education Program: http://healthysleep.med.harvard.edu/portal/
Minority Nurse: http://www.minoritynurse.com/
National Center on Minority Health and Health Disparities: http://ncmhd.nih.gov/default.html
National Center on Sleep Disorders Research—Racial and Ethnic Disparities: http://www.nhlbi.nih.gov/health/prof/sleep/res_plan/section4/section4b.html
National Coalition of Ethnic Minority Nurse Associations: http://www.ncemna.org/
National Sleep Foundation: http://www.sleepfoundation.org/
National Sleep Foundation—Spanish: http://www.sleepfoundation.org/secondary-links/en-español
Nursing Programs for Minorities: http://www.allnursingschools.com/faqs/diversityfaq.php
Sleep and Ethnicity 2010 Sleep in America Poll: http://www.sleepfoundation.org/article/sleep-america-polls/2010-sleep-and-ethnicity
Sleep Disorder Sites (Rated by Stanford): http://www.sleepnet.com/links.htm
Stanford Sleep Research Center Links: http://med.stanford.edu/school/psychiatry/humansleep/links.html
Transcultural Nursing Society: http://www.tcns.org/

Gaps in the literature on racial/ethnic aspects of sleep can serve as heuristics for future research studies, and culturally responsive sleep education, intervention and promotion activities.

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16 Sleep Promotion in the Childbearing Family

Kathryn A. Lee

During the childbearing phase of life, a woman's sleep can be profoundly altered, with increased risk for physical and mental health problems. Poor sleep can adversely affect labor and delivery, lead to poor maternal–infant interactions or poor relationships with other adults, and even affect the infant's growth and development. Hormonal alterations during early pregnancy, enlargement of the fetus during late pregnancy, and a newborn with random patterns of sleeping and feeding contribute to her disrupted sleep. This chapter briefly reviews normal physiological and anatomical changes that occur during pregnancy and discusses common sleep disorders that can occur during pregnancy as well as postpartum. The adverse effects of poor sleep on labor and delivery outcomes are briefly discussed, and postpartum sleep patterns are reviewed within the context of risk for postpartum depression. Throughout this chapter, the nurses' role in sleep promotion is discussed in relation to sleep hygiene behaviors that can be adapted for pregnant women and new mothers and their families during the first 6 months postpartum.

For the majority of healthy women, nursing attention toward promoting sleep during pregnancy should be focused on comfort measures as part of any healthy sleep hygiene protocol. A recent national survey of 1,000 women in the United States revealed that about 30% rarely or never got a good night's sleep during their pregnancy (National Sleep Foundation, 2007). Poor sleep is understandable during this time in a woman's life, given the hormonal and anatomical changes as well as anxiety associated with fear of painful or prolonged labor, the upcoming life-changing event of becoming a mother, or fear of having an unhealthy baby. Most women are reluctant to discuss a sleep problem with their health care provider, however, because they do not want to consider taking any medication during pregnancy or while breast-feeding. This chapter describes alternative strategies nurses can use to safely promote sleep during the perinatal period. However, if daytime sleepiness becomes hazardous during pregnancy or postpartum, the cause must be medically treated and not be dismissed or assumed to be a normal symptom of pregnancy. Restless legs syndrome (RLS) and sleep-disordered breathing (SDB) or obstructive sleep apnea (OSA) are discussed in this chapter as they are two of the most critical causes of disturbed sleep during pregnancy and require urgent assessment and referral for effective and non-pharmacologic interventions. For women at risk of SDB or OSA, screening and treatment is imperative for the health of the fetus. For those at risk of RLS, nurses can offer nutritional advice and comfort measures to promote sleep.

The final section of this chapter describes sleep during the postpartum period, when women's experience with sleep deprivation is expected. Strategies to promote sleep and reduce the risk of excessive daytime sleepiness are discussed. Chronic sleep loss may be a risk factor for postpartum depression. Given the increased risk of postpartum depression, all pregnant and postpartum women should be screened for depressive symptoms. In particular, complaints about difficulty falling asleep should be explored further in a new mother who normally would be sufficiently sleep-deprived and have no trouble falling asleep at bedtime.

PHYSIOLOGICAL AND ANATOMICAL CHANGES ASSOCIATED WITH SLEEP

Beginning early in pregnancy, placental secretion of estrogen, progesterone, and prolactin hormones increases. Progesterone, the hormone essential for maintaining the pregnancy, also increases core temperature (deMouzon, Testart, Lefevre, Pouly, & Frydman, 1984) and drowsiness (Heuser, Ling, & Kluver, 1967). Progesterone's inhibitory effects on the nervous system result in a calming effect in which women feel drowsy or sedated, and anesthesiologists are well aware of progesterone's potentiating effects on anesthetics and analgesics (Selye, 1941).

Progesterone's effect on smooth muscle causes women to begin experiencing urinary frequency early in the first trimester, long before the uterus begins pressing on the bladder. By the third trimester, the discomfort and resultant sleep disturbance is most frequently attributed to the large uterus, which not only presses on the bladder to reduce capacity and increase urinary frequency, but also makes it difficult to change positions during sleep without arousing to consciously find a more comfortable sleeping position. To make room for the enlarging uterus, the diaphragm elevates and breathing becomes shallower; progesterone increases minute ventilation, and women feel short of breath. The intestines and esophageal sphincter are displaced, resulting in esophageal reflux and heartburn, particularly while lying down.

Fluid volume increases by about 7 L during pregnancy. The increased level of estrogen is thought to be responsible for the vascular changes, but the net result is nasal congestion and swelling in the extremities. Changes in connective tissue and joints can be uncomfortable and result in vision changes or larger foot size. Women may complain about these changes, but are more accepting of them because they are temporary, the cause is obvious, and there will be a joyful outcome. However, as discussed later, the increased risk for snoring and sleep-disordered breathing should not be neglected, particularly when there is excessive abdominal weight.

NORMAL SLEEP DURING PREGNANCY

Sleep studies in a controlled laboratory environment consistently document that pregnant women have about 45–60 minutes more wake time during the night compared to about 5 minutes in healthy, nonpregnant women of the same age (Driver & Shapiro, 1992; Signal et al., 2007). In the home sleeping environment, more awakenings are likely due to other family members, noise, light, the bed partner's movement, longer trips to the bathroom, or other uncontrollable environmental factors.

When healthy pregnant women were compared with their own prepregnancy sleep patterns in the home environment with ambulatory polysomnography monitoring, a similar increase in wake time was also seen, as well as a reduction in deep sleep stages (slow-wave sleep Stages 3–4) (Lee, Zaffke, & McEnany, 2000). Since deep sleep is associated with higher arousal threshold for noise, less time in deep sleep stages would likely make pregnant women more susceptible to awakenings. Even in longitudinal studies such as this, it would be difficult to determine whether the discomforts of pregnancy cause the awakenings and thus less deep sleep, or whether the reduced deep sleep results in more wake time. Regardless of cause or effect, this gradual reduction in deep sleep and more wake time over the course of pregnancy may go unnoticed as women adapt over time. For those who maintain their prepregnancy bedtimes and wake times, however, the outcome is about 1 hour less total sleep time (TST) during the night, and the clinical implications of sleep loss for labor and delivery outcomes are discussed later in this chapter.

Most (80%) women with a prior pregnancy loss reported having dreams associated with anxiety about labor and delivery outcomes (Van, Cage, & Shannon, 2004). Similar dreams are described by other pregnant women, and at about the same rate (90%), indicating that dreams are more likely remembered because of the frequent arousals during sleep (Nielsen & Paquette, 2007). Dream recall may increase over the course of the pregnancy, but the

frequency of nightmares is similar to prepregnancy baseline rates in prospective longitudinal studies (Baratte-Beebe & Lee, 1999). Since rapid eye movement (REM) sleep has not been shown to vary over the course of pregnancy, and REM sleep is when dreams are occurring, a woman's dreams and nightmares may be an important indicator of her anxiety and mental health status.

The prevalence of symptoms associated with disrupted sleep by trimester is summarized in Table 16.1. In general, these are common complaints that increase in frequency or severity as gestation increases (Schweiger 1972), and many strategies have been described in the literature to cope with these symptoms. Very little research has been done to provide the definitive evidence for implementing these strategies, but most are comfort measures that are nonpharmacologic and easily implemented and discarded if not effective.

A recent pilot study that tested the effects of yoga and meditation revealed a particularly promising line of inquiry for sleep promotion and symptom reduction during pregnancy. Beddoe and colleagues enrolled 15 healthy pregnant women in a 7-week group yoga class with a mindfulness-based stress reduction (MBSR) component. Wrist actigraphy was used to objectively monitor their sleep before and after the intervention, self-report measures of sleep, and symptoms were also collected, and the yoga poses were modified specifically for pregnant women. There were no effects for sleep or pain for the eight women who began the sessions during the third trimester, but they did lower their level of anxiety (Beddoe, Yang, Kennedy, Weiss, & Lee, 2009). The seven women who began the intervention during their second trimester had fewer awakenings and less wake time during the night after the intervention. The second trimester group also had better sleep and less pain at the end of their yoga intervention, now into their third trimester, compared to the third trimester women's baseline measures (Beddoe, Lee, Weiss, Kennedy, & Yang, 2010). This was a small pilot study that demonstrated safety and efficacy, but it is not clear whether it was any specific yoga poses, the MBSR component, or the social support that could be provided by a group intervention. More research is needed to replicate these findings before recommending that women begin yoga early in pregnancy to reap the potential benefits. The various components of a yoga intervention would complement sleep hygiene strategies. The sleep hygiene strategies described in Table 16.2 are all categorized as comfort measures to reduce symptoms and promote sleep during pregnancy. They are organized using the acronym, *B.E.T.T.E.R* to help

Table 16.1 ■ *Summary of Sleep Complaints During Pregnancy*

Trimester	Symptoms Associated with Sleep Disruption
First trimester	■ Less than 5% have joint pain, nightmares, or anxiety. ■ 12%–15% report disturbed sleep due to nausea, vomiting, or backache. ■ About 20% begin to experience sudden awakening from painful leg cramps. ■ 50%–52% report frequent nocturnal voiding.
Second trimester	■ About 20% begin to report that fetal movements and heartburn disrupt sleep. ■ 30% report a new onset of snoring during pregnancy (Izci-Balserak, 2005).
Third trimester	■ Less than 5% have nightmares or anxiety. ■ About 5% report awakening from fetal movement. ■ 19%–25% experience restless legs syndrome. ■ 20%–25% have joint pain. ■ 25% suffer from heartburn or esophageal reflux (Rey et al., 2007). ■ 45%–50% report frequent nocturnal voiding. ■ 75% experience awakenings from leg cramps.

Table 16.2 ■ *Sleeping B.E.T.T.E.R. Hygiene for Promoting Sleep During Pregnancy*

***B*edroom:**

- Make bedroom dark, cool, safe, and comfortable.
- Use night light in the bathroom to decrease arousal time.
- Use white noise machine or fan to mask noise from traffic or bed partner.
- Use pillows between the knees, under abdomen and behind the back for comfort.
- Sleep side-lying, preferably left lateral position, to improve blood flow to fetus and improve maternal renal function.
- Elevate head of bed to reduce heartburn and snoring.
- Use nasal dilator strips or saline nasal washes to relieve nasal congestion.

***E*ating:**

- Drink lots of water during the day, but cut down before bedtime to reduce nocturia.
- Keep dry crackers at the bedside to ease morning or evening nausea.
- Eat a balanced diet (include magnesium, potassium, calcium).
- Rather than hot chocolate in the evening, consider drinking Ovaltine with hot milk.
- Take vitamin C and eliminate caffeine to increase folate absorption from food.
- Avoid carbonated beverages to reduce risk of leg cramps.
- Take prenatal vitamins with iron and folate to reduce risk of restless legs syndrome.
- Avoid caffeine, spicy, acidic, and fried foods to reduce heartburn.
- Eat small meals during the day to reduce heartburn.

***T*ension:**

- Practice relaxation techniques to reduce tension.
- Use massage, local heat, and pillow support for low back pain.
- Consider drinking non-caffeinated hot tea in the evening.
- Consider the sedative effects of lavender pillows or bubble bath in the evening.

***T*ime in Bed:**

- Plan a regular sleep-wake schedule, prioritize sleep as "sleeping for two."
- Extend your time in bed by 10 minutes each month of pregnancy, so there is at least 8 hours in bed to get a minimum of 7 hours of sleep during the last month of pregnancy.
- Consider a nap during the day unless you have problems falling asleep at bedtime.
- Stay in bed only when sleepy.

***E*xercise**:

- Exercise daily to control weight gain, improve circulation, reduce tension and stress.
- Consider a yoga class with poses and breathing specific to pregnancy.

***R*hythm:**

- Get daylight exposure every day.
- Eat meals and snacks at consistent times.
- Have a consistent nightly bedtime and morning wake time.
- Get up out of bed at the same time every morning.

patients remember to think about their *B*edroom environment, what they *E*at and drink, how to reduce *T*ension, be aware of the *T*ime they spend in bed, the importance of regular *E*xercise, and keeping a consistent circadian *R*hythm. These are all useful strategies for promoting sleep in any adult, and can be reinforced for the woman's entire family.

Despite the best of sleep hygiene practices, pregnancy can place women at risk for specific sleep disorders. If the nursing assessment reveals that daytime sleepiness is occurring despite healthy sleep hygiene behaviors, the potential for a sleep disorder must be investigated for the health and safety of the mother and fetus. The next sections discuss two of the

more common sleep disorders seen during pregnancy. There are other less common disorders that can also impact daytime functioning, but in general, any complaint of daytime sleepiness should be evaluated and if necessary, referral to an accredited sleep disorders center should be considered.

SLEEP DISORDERS DURING PREGNANCY

Sleep-Disordered Breathing in Pregnancy

Epidemiological studies and laboratory polysomnography studies find that sleep-disordered breathing (SDB) is less common in women than men for any given age. Given the normal adaptive changes in pulmonary function during pregnancy, most healthy women will not develop SBD. Nevertheless, a pregnant woman and her fetus are at greater risk than other women of childbearing age because of increased body weight and edema of the nasal mucosa (for a recent review, see Champagne, Kimoff, Barriga, & Schwartzman, 2010). Symptoms of SDB range from light or heavy snoring to apneic events with sudden awakenings and choking sensations. (See Chapter 7, Sleep-Related Breathing Disorders.) With increased body weight and obesity comes greater risk for obstructed airways while sleeping in a supine position. By the third trimester, the prevalence of snoring ranges from 10% to 46%, depending on whether the data come from a pregnant woman's self-report or from her bed partner, and depending on whether or not she snored prior to pregnancy (Izci-Balserak 2008; Izci et al., 2005).

It has been estimated that 1 in 10 pregnant women may be at risk for sleep apnea during pregnancy (Pien, Fife, Pack, Nkwuo, & Schwab, 2005). However, when over 260 women were screened in the second trimester, none were diagnosed with OSA (Guilleminault, Querra-Salva, Chowdhuri, & Poyares, 2000). Even though SDB improves with weight loss after delivery (Edwards, Blyton, Nennessy, & Sullivan, 2005), it is critical to recognize and treat SDB because ongoing hypoxic events have been associated with intrauterine growth retardation, small placentas, or newborns who are small for gestational age (Franklin et al., 2000). It may not be cost-effective to evaluate all women for OSA during pregnancy, but women especially at high risk (those with pregnancy-induced hypertension or preeclampsia, gestational diabetes, or unexplained intrauterine growth retardation) should be screened by self-report (see Chapter 7, Sleep-Related Breathing Disorders) and referred to an accredited sleep disorders center if warranted (Champagne et al., 2010). OSA can be adequately and safety treated with safe, non-pharmacologic strategies such as positioning, nasal dilator strips, or continuous positive airway pressure (CPAP) devices (Guilleminault et al., 2007).

Leg Cramps and Pregnancy-Related Restless Legs Syndrome

Women have a variety of leg aches and pains during pregnancy as abdominal pressure on the pelvis increases and metabolism of calcium, magnesium, and other micronutrients are directed toward fetal development. The intense pain from a leg cramp is the most common reason for sudden awakenings during pregnancy. Compared to prepregnancy when leg cramps were reported at about 10%, the incidence increased to 21% in the first trimester, 57% in the second trimester, and 75% during the third trimester in one longitudinal study (Lee, Zaffke, & Baratte-Beebe, 2001). Few clinical trials have been done to address treatment strategies for reducing leg cramps, but anecdotal evidence would suggest nightly stretching before bedtime, reducing phosphorous-containing carbonated beverages, heat or cold, and gentle leg massage are safe and may be useful comfort measures. A slow hyperextension of the calf muscle, by flexing the foot toward the knee, is useful at the onset of the cramp in order to reduce the duration of the cramping. An analysis of placebo-controlled trials concluded that there was no benefit from use of calcium, sodium chloride, or calcium with sodium chloride; the magnesium found in multivitamins may be beneficial (Young & Jewell, 2002).

RLS is typically experienced in the evening before bedtime while trying to fall asleep. It is described as a strange uncomfortable sensation, accompanied by an irresistible urge to move the legs. (See Chapter 8, Sleep-Related Movement Disorders and Parasomnias.)The sensation is then relieved by getting up and walking, which delays sleep onset, reduces TST, negatively affects mood, and is described as "pure torture" by some women wanting desperately to fall asleep. Circadian rhythms for iron metabolism and dopaminergic pathways converging in the evening hours make it more of an evening or nightly experience, and low levels of folate or iron may be the culprit as they decrease over the course of pregnancy (Lee et al., 2001). Women with prepregnancy low normal ranges for hemoglobin, ferritin, or folate are at increased risk, and even taking prenatal vitamins make it difficult to increase levels. Thus, pregnant women have three times the rate of RLS compared to the general population, particularly if they are not taking nutritional supplements of vitamins and iron (Tunc, Karadag, Dogulu, & Inan 2007). Those with multiple gestation (twins or triplets) are at especially high risk. In one study of 10 women with multiple gestation, all 10 had periodic leg movements (PLMs) during sleep, and 4 of the 10 went on to experience RLS (Nikkola, Ekblad, Ekholm, Mikola, & Polo, 1996).

Pregnancy-related RLS is usually only a temporary condition until delivery. The prevalence of pregnancy-related RLS varies by age, race, and family history (Xiong et al., 2010) but is about 20% by the third trimester in those who did not have RLS prior to pregnancy, and reverts back to 0% after delivery (Harano et al., 2008; Lee et al., 2001; Manconi et al., 2004; Suzuki, Ohida, Sone, Takemura, Yokoyama, et al., 2003). Women who experienced previous pregnancy-related RLS have an increased risk (30%–40%) in their next pregnancy, particularly if there is short spacing between the two pregnancies and iron stores are depleted (Manconi et al., 2004). Since the risk of RLS is increased in diabetic patients, women with gestational diabetes may be at greater risk of RLS (Bosco et al., 2009). For an in-depth review of this topic, see Hensley (2009) and Ekbom and Ulfberg (2009).

In addition to iron and folate nutritional supplements and prenatal vitamins, comfort measures to minimize symptoms are well worth the effort, and dopamine or iron therapy may be warranted in severe cases. To cope with these RLS symptoms, light leg massage, hot or cold compresses, walking, and gentle stretching in the evening are safe and may be useful. Increased intake of vitamin C can facilitate iron metabolism, and caffeine or other stimulants should be avoided to improve uptake of iron and folate. Rather than a cup of hot chocolate in the evening, a cup of warm milk or Ovaltine would be more beneficial and avoids stimulants found in chocolate.

PRENATAL SLEEP DURING HOSPITALIZATION

Very little sleep research has been conducted on hospitalized patients, particularly those in antepartum units. Women hospitalized during the prenatal period are usually admitted for preterm labor, placental abnormalities, or pregnancy-induced hypertension. In a sample of women with multiple gestation on bed rest precautions, complaints about sleep were as common (over 80%) as backache and dry lips (Maloni, Margevicius, & Dalmato, 2006). Sleep was not objectively monitored in this sample, but results are very clinically important, particularly given that bed rest would allow for extra sleep if needed, but hospital environments are not typically conducive to sleep promotion due to noise, excessive lighting, and frequent patient care activities. (See Chapter 20, Sleep in Adult Acute and Critical Care Settings.) More research is needed with this population to determine whether it is the environment, medication side effects, or lack of physical activity that result in complaints of poor sleep.

When women were studied with wrist actigraphy to monitor their sleep in the hospital unit, 30%–40% reported that their sleep was very bad, and the objective actigraphy data validated their self-reports. This sample

of hospitalized women had many different diagnoses, but awakenings ranged from 9 to 32 times each night, and naps averaged about 2 hours during the day (Gallo & Lee, 2008). The implications of this type of sleep loss on labor and delivery outcomes have not been adequately studied.

LABOR AND DELIVERY

With the discomforts associated with healthy pregnancy, sleep quality diminishes, particularly in the few weeks prior to labor (Evans, Dick, & Clark, 1995). When the fetus drops lower into the birth canal, breathing is easier for the mother-to-be, but physiological preparations are underway to begin the labor and delivery process. When sleep was monitored with wrist actigraphy for 2 weeks prior to women's expected due dates, sleep was most disrupted during the 5 days prior to delivery, and especially fragmented during the night before hospital admission (Beebe & Lee, 2007). This was seen in those who experienced spontaneous labor, but unexpectedly, it was also seen in those women the night before being admitted for a scheduled induction. In a qualitative study with 20 women, all described being unable to sleep once contractions began (Kennedy, Gardiner, Gay, & Lee, 2007).

Women's self-reported sleep quality did not appear to influence labor or delivery outcomes in one study (Evans et al., 1995). Self-report measures may not totally capture one's estimate of total time spent asleep, however. When amount of sleep was documented with wrist actigraphy at about 3 weeks prior to delivery for women having their first baby, those who slept less than 6 hours at night went on to have significantly longer labor (12 hours on average), and they were 4.5 times more likely to have a cesarean delivery compared to women who slept more than 7 hours (Lee & Gay, 2004).

The sleep loss that results from being in labor during the night (Wilkie & Shapiro, 1992) can result in a higher risk of emotional distress during the early postpartum period. In the early stages of labor, when contractions do not seem to be regular or progressing, morphine sulphate has been commonly administered to induce sleep and reduce uterine contractions. The laboring woman then typically awakens in more active labor (Conkin, 1998). When a woman is admitted into the Labor and Delivery unit and not progressing during initial labor onset, nurses should evaluate how much, or how little, sleep she has been getting in the past few days and evaluate whether a few hours of morphine-induced sleep could be beneficial before she begins her vigorous labor activity.

POSTPARTUM SLEEP

Postpartum begins with the birth and continues throughout lactation and until the infant is consistently sleeping through the night. Disturbed sleep during the postpartum period is common and particularly problematic for primiparas (74.4%) and for women after cesarean (73%) compared to vaginal (57%) delivery (Tribotti, Lyons, Blackburn, Stein, & Withers, 1988). When new mothers had their infants in the same room on their first postpartum night after vaginal delivery, 25% of the night was spent awake compared to 5% for healthy controls, but no significant differences in amount of REM or slow-wave sleep (SWS) were noted (Zaffke & Lee, 1992).

In one of the first studies to examine sleep in the postpartum unit, Lentz and Killien (1991) used observation techniques to monitor sleep and described the common reasons for awakening. Most awakenings were for infant feeding, but there were other awakenings when nurses entered the room to check vital signs. This was at a time when women were typically in the hospital for 48 hours prior to discharge, and it would be interesting to compare women's sleep at home to see which environmental setting is more conducive to promoting sleep in the early postpartum period.

Mothers with a newborn admitted to the neonatal intensive care unit (NICU) are bound to be more anxious and have problems sleeping. In a study of 21 postpartum mothers with 3–5-day-old infants in the NICU, sleep time averaged only about 4 hours for the 6 women who had a cesarean delivery and were still

hospitalized (Lee & Lee, 2007). In comparison, sleep time averaged 6.5 hours for the 15 women in the sample who had a vaginal delivery and were sleeping at home (Lee & Lee, 2007). These findings were based on wrist actigraphy measures, and wake time was also significantly different for the two groups; the hospitalized postoperative cesarean women had 34% wake time during the night, whereas the other group had only 14% wake time sleeping at home. Both groups were in their first postpartum week, and all mothers were breast-feeding, but differences in sleep could be due to the hospital environment, surgical recovery, or other factors such as parity and the home situation with other family members and children. Given this critical time for healing and need for restorative sleep, nurses working in postpartum units should make sleep a priority issue for new mothers recovering from surgery, particularly after a traumatic cesarean birth that results in admission to a NICU for the infant or longer hospital stay for the mother.

It is during the early postpartum period when novice new mothers (primiparas) have substantially more interrupted sleep during the night than experienced (multipara) new mothers (Lee, Zaffke, & McEnany, 2000; Signal et al., 2007). Although sleep efficiency averages about 90% during the third trimester, it drops to about 77% during the first postpartum month in novice mothers, but only drops to about 84% in experienced mothers. For both groups at 1 month postpartum, deep slow wave sleep (SWS) is increased compared to pregnancy levels, and light sleep Stages 1 and 2 are decreased (Driver & Shapiro, 1992; Lee et al., 2000).

Breast-Feeding

There is scant research on polysomnography-recorded sleep differences between women who breast-feed and women who formula feed during the postpartum period. Petre-Quadens and DeLee (1974) studied fewer than 10 women and reported a difference in the pattern of REM sleep during the first 2 weeks postpartum for breast and formula feeding mothers. There was a gradual decrease in REM sleep for formula feeding mothers, whereas REM sleep remained high for the breast-feeding mothers. Blyton, Sullivan, and Edwards (2002) studied women in the home environment with portable polysomnography, and the 12 lactating women in their sample had less light sleep (Stages 1 and 2), fewer arousals, and more SWS compared to the seven non-lactating postpartum women. There was no difference in the amount of REM sleep or TST between the two groups (Blyton et al., 2002). More SWS may be one added benefit for breast-feeding mothers, but subjective measures were not included and more research is needed with a larger sample to confirm these results.

The increase in SWS for breastfeeding mothers is hypothesized to be due to the influence of prolactin hormone. For lactating women, basal levels of prolactin are high, and there is a burst of prolactin secretion at the onset of each breast-feeding event, regardless of when sleep occurs, but the bursts seem to be of a higher magnitude in the evening compared to morning (Noel, Suh, & Frantz, 1974). Both basal levels and bursts diminish to prepregnancy levels by about 3 months postpartum. Within 24 hours of weaning, prolactin levels return to low basal levels and to the circadian sleep-associated patterns seen in healthy adults (Uvnäs-Moberg, Widström, Werner, Matthiesen, & Windberg, 1990).

The effect of lactation on sleep patterns of mothers and infants has also been examined with wrist actigraphy in the home environment. Actigraphy measures of more wake time during the night were seen in women who exclusively breast-fed at 3–4 weeks after birth, but the mother's perception of her sleep was unrelated to type of infant feeding (Quillan, 1997). In the largest study to date, Doan, Gardiner, Gay, and Lee (2007) analyzed sleep data from wrist actigraphy and from self-report measures to compare new mothers who were exclusively breast-feeding and new mothers who used supplementation during the night at 3 months postpartum. The fathers' sleep was also assessed in this study. In the exclusive breast-feeding group, both parents had more sleep time (about an extra 45 minutes) compared to the formula feeding parents, and self-report findings were

consistent with the wrist actigraphy data. These findings from 48 hours of wrist actigraphy in the home at 3 months postpartum differ from the findings of a similar TST for breast-feeding and formula feeding mothers in the small sample studied with polysomnography for one night in the home setting at a similar average age (11 weeks), but infant ages ranged from 6 to 28 weeks old (Blyton et al., 2002).

Co-Sleeping and Bed-Sharing

Co-sleeping is defined as children sleeping with their parents, either in the same room or the same bed. In most cultures, various forms of co-sleeping exist, ranging from mother and baby sharing the same bed to mother and baby sleeping in the same room. Large epidemiological studies would indicate that bed-sharing is more prevalent than pediatricians and family practice experts would expect from what a parent might report, and not just among low-income families with limited space in the home. Bed-sharing rates rose in the United States from less than 10% in the 1990s to between 35% and 40% in more recent surveys (Brenner et al., 2003; Willinger et al., 2003). After the American Academy of Pediatrics spoke out against bed-sharing in 2005, the rate has dropped substantially, but remains at about 20% based on self-report from one area in the United States (Norton & Grellner, 2010) to 72% in a Canadian sample (Ateah & Hamelin, 2008).

During pregnancy, most parents indicate that they do not plan for the infant to sleep in the parent's bed. Parents may not intend to bed-share, but resort to this strategy as a way of coping with sleepless nights. This short-term strategy is not only an unsafe practice but has little potential for improving a parent's sleep. As discussed in the next paragraph, parents have more arousals from sleep and suffer more fatigue over time. Nurses can be proactive in suggesting that a bedside crib be placed next to the parent's bed during the early postpartum weeks to avoid unplanned bed-sharing.

McKenna et al. (1993) used polysomnography to study co-sleeping in three women who were between 2 and 4 months postpartum. In a controlled laboratory setting with the infant in bed with the mother, they found increased arousals and more frequent changes in sleep stages, but no difference in sleep efficiency compared to previous nights when sleeping alone. Further research from this group in a larger sample revealed that sleep was indeed affected, with less SWS and more arousals for mothers who shared a bed with their infant (Mosko, Richard, & McKenna, 1997).

When a newborn sleeps in its own crib but in close proximity to mother, both are likely to have more consolidated sleep and more TST, because less time is spent in arousals and awakenings. Nishihara and Horiuchi (1998) studied 10 primipara Japanese mothers. They used ambulatory polysomnography in the home setting and reported results from 8 of the 10 mothers. Interestingly, sleep efficiency for these novice mothers at 3 weeks postpartum (86%) resembled the sleep efficiency (84%) for the experienced mothers in the Lee and colleagues' (2000) study. In the Japanese sample, co-sleeping with the infant within "arms reach" on an infant futon is an accepted cultural practice. These studies would support the use of a bedside crib as a safe and effective strategy for sleep promotion for novice new mothers.

Sleep Deprivation and Postpartum Depression

An occasional night of poor sleep is well tolerated by most adults, particularly if there is an opportunity for a nap during the day. Chronic sleep deprivation, however, can have profound implications for maternal and infant health and safety. Automobile crashes, infant abuse, and infant death from hyperthermia in unattended vehicles may be directly related to sleep deprivation, but there is scant research on this topic. Most poignant is the qualitative data from Lamott (1993), in a published memoir about life with her infant son, Sam. Many new parents share this type of experience, but are not as eloquent:

> We had another bad night. We finally slept for two hours at 7 AM. What a joke. I feel like thin glass, like I might crack. I was very

> rough changing him at 4 when he wouldn't stop crying. I totally understand child abuse now. I really do ... I can't stop crying. I cried all night ... (Lamott, 1993, entry for October 5th)

Research finds that poor maternal sleep is associated with depressive symptoms (Dennis & Ross, 2005; Goyal, Gay, & Lee, 2007). This is a difficult area to study, however, because most self-report measures of depressed mood have overlapping items related to sleep, and most sleep measures have items about daytime functioning and lack of energy or enthusiasm. Many measures of depressed mood contain items that are normal somatic symptoms of pregnancy or postpartum, such as "difficulty getting going" or "tires easily."

The overall prevalence of depression among women of all ages is about 20%, and the childbearing years are no exception. After the first few weeks postpartum when the "baby blues" is a very common experience, symptoms of depression can emerge with even more distinction. Symptoms of postpartum depression are similar to the depressive symptoms experienced at other times in a woman's life, but there are three distinguishing questions that should be included in assessing childbearing women. The first question is whether or not the new mother is worried about hurting her infant. The second question is whether or not she has trouble falling asleep at her normal bedtime, and the third question is whether she has trouble sleeping when the baby is sleeping.

Coble et al. (1994) were the first to really explore the relationship between sleep patterns and depression in childbearing women. They recruited pregnant women at risk for depression due to their prior history of mental health problems, and compared their sleep with healthy controls in the home setting using polysomnography. They found no association between mental health outcomes and sleep during the first 8 months postpartum for the at-risk group of women. In fact, none of their samples developed postpartum depression during the first 8 months. An interesting question emerges from their findings: To what extent does being a valued subject in a longitudinal research study, where someone is monitoring your sleep and other aspects of your life on a monthly basis, provide a protective or supportive intervention for women, even those at high risk for depression?

In a similar protocol with healthy women at low risk for depression, no one developed postpartum depression either, but depressive symptoms were associated with more awakenings during the night and an earlier onset of REM sleep (Lee, McEnany, & Zaffke, 2000). Compared to those with low scores for depressive symptoms who slept a similar amount in both the third trimester and at 1 month postpartum, the group with more depressive symptoms slept 80 minutes less on average at 1 month postpartum than they were sleeping during the third trimester (Lee et al., 2000).

Using sleep diary reports, Wolfson, Crowley, Anwer, and Bassett (2003) found a different type of sleep pattern for those who had more depressive symptoms. Total amount of sleep and bedtime did not differ at 1 month postpartum, but prior to delivery they did have later final wake times in the morning and were more likely to nap during the day. In contrast, wrist actigraphy monitoring indicated that mothers with higher depressive symptom scores were those who had less than 4 hours of TST between midnight and 06:00 AM and mothers who napped for less than an hour during the day (Goyal et al., 2007). Difficulty falling asleep (Posmontier, 2008) and daytime sleepiness (Huang, Carter, & Guo, 2004) are the most common sleep issues for mothers with higher depressive symptom scores.

There was no relationship between their depressive symptoms and the infant's sleep pattern (Goyal, Gay, & Lee, 2009), but recent research is suggesting that the sleep of newborns is more disrupted even in the first few weeks of life if the mother has a history of depression (Armitage et al., 2009). The new father is also at higher risk for depression if the mother has postpartum depressive symptoms (Goodman, 2004).

Regardless of whether sleep is assessed with polysomnography, wrist actigraphy, or self-report and diary methods, findings from these studies would suggest that mothers with more fragmented sleep and less TST are more susceptible to depression, particularly if they complain about difficulty falling asleep. Difficulty falling asleep may be explained by recent evidence for differences

in circadian rhythms for healthy and depressed childbearing women. As a marker for circadian rhythm, Parry et al. (2008) compared melatonin levels during pregnancy and postpartum and found significantly lower levels during the night in those with major depression compared to healthy controls. To what extent postpartum depression is related to sleep loss (Hunter, Rychnovsky, & Yount, 2009), the release of inflammatory cytokines due to sleep loss (Chang, Pien, Duntley, & Macones, 2010), or alterations in circadian rhythm in vulnerable populations of women, needs further research. Nurses should consider all women and their infants vulnerable to adverse health outcomes associated with sleep loss.

CONCLUSIONS

Figure 16.1 summarizes the many ways in which sleep loss can occur during pregnancy or postpartum. Nurses need to consider the potential adverse outcomes as they relate to a woman's physical and psycho-social health. Pregnancy is a time when women are highly eager for new information to improve the health of an unborn child, and highly motivated to make changes to improve their health and well-being. Tables 16.2 and 16.3 contain suggestions for strategies that can be very effective at promoting sleep during the childbearing years, not only for the women themselves, but for their entire family.

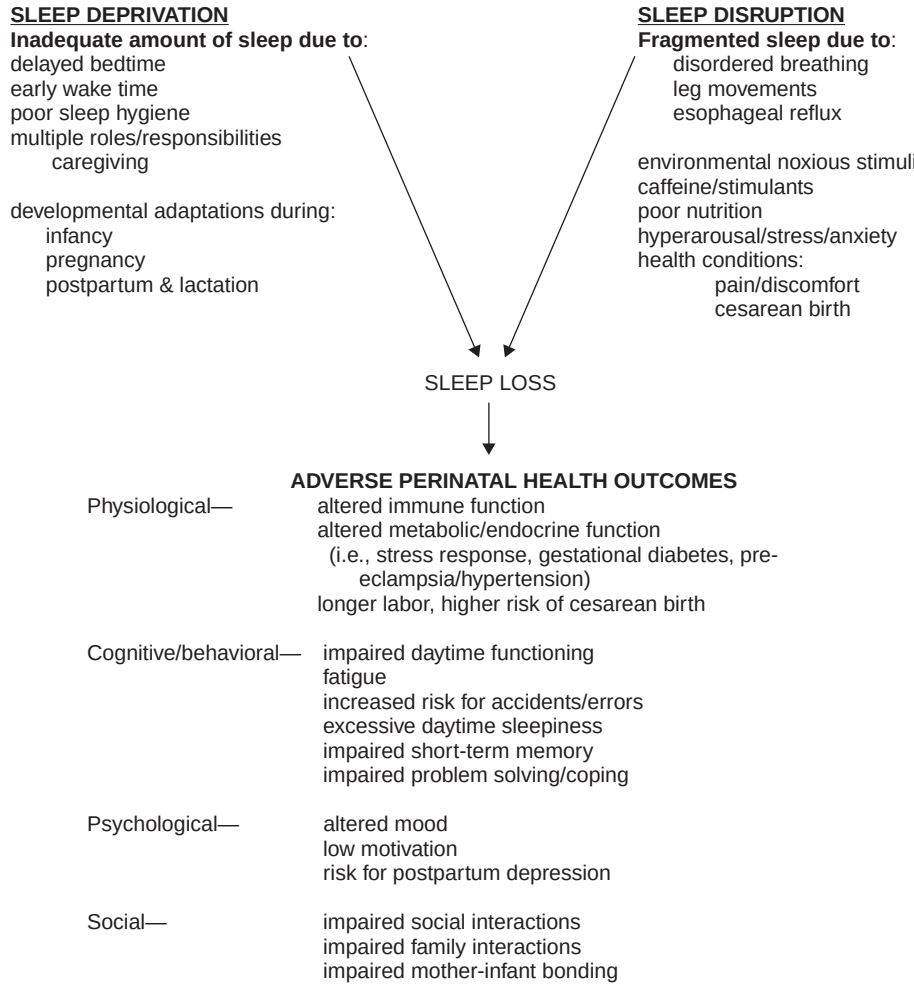

Figure 16.1 ■ Conceptual model of impaired sleep for the childbearing family.

Table 16.3 ■ *Sleeping B.E.T.T.E.R. Hygiene Strategies for New Mothers and Infants*

***B*edroom:**
- Keep room dark, cool, safe, and comfortable during the night.
- Keep the newborn in a bedside crib next to mother's side of the bed.
- Do not sleep with the infant in the parental bed for safety reasons.
- Use a night light at the bedside crib to safely perform infant care and feeding during the night.
- Use a white noise machine or fan to mask the noises from the baby or bed partner.

***E*ating:**
- Avoid caffeine and alcohol, spicy, acidic, or fried foods, or other foods that may affect breast milk and infant sleep.
- A high-calorie diet assures sufficient calories for lactation and breast milk production.
- Have a friend or family member take responsibility for the food shopping and preparing the evening meal.

***T*ension:**
- Practice relaxation techniques.
- Plan a shower or bath when the baby's father gets home from work to allow for father-baby interactions.
- Rather than the father doing nighttime infant care and risking sleep deprivation, have the father leave work an hour early and bring dinner home from the workplace cafeteria.

***T*iming:**
- Plan regular sleep-wake schedule, prioritizing sleep. Nap when the baby naps.
- Stay in bed only when sleepy.
- Limit daytime sleep if you have difficulty falling asleep at bedtime.
- Get up at the same time every day, including weekends, holidays, and days off from work.

***E*xercise:**
- Regular exercise should include walking outdoors (alone or with the baby in a stroller) and adequate light exposure.
- Cleaning the house can be a form of physical activity, but should not be a priority activity.

***R*hythm:**
- Get a daily dose of sunlight at about the same time every day, even in winter or on cloudy days.
- New mothers need daily light exposure to maintain their circadian rhythms and promote positive mood and mental health.
- Infants need daily light exposure and dark sleeping environments as cues to develop their circadian rhythm and sleep through the night.

Implications for Research

1. Women need to be recruited into longitudinal studies based on past sleep history to determine whether sleep improves or worsens with pregnancy.
2. Attention to parity is essential in sample design or statistical analyses in order to generalize findings to all pregnant and postpartum women.
3. The symptoms experienced during various stages of pregnancy need to be examined for their influence on sleep loss.
4. The contributions of internal physiological changes during pregnancy, assessed with inflammatory markers or melatonin circadian rhythm markers, need to be compared with factors in the external environmental when sleep is studied during the childbearing years.
5. More research is needed on the benefits of breast-feeding for promoting maternal and infant sleep.
6. The severity of sleep disturbance and depressive symptoms in the early postpartum period needs to be studied in new mothers, new fathers, and infants.
7. The effects of co-sleeping on mothers, fathers, and the infants need to be studied in Western and non-Western cultures.

Recommendations for Practice

1. When women are seen for prenatal care, emphasizing "eating for two" and "sleeping for two" should have equal weight in patient teaching and childbirth preparation.
2. The positive and negative effects of co-sleeping should be discussed with women prior to birth. Even if it is not planned,

bed-sharing can occur from desperation for sleep and the family should have alternative strategies in place for that event.
3. Women should be counseled during pregnancy and reminded during postpartum visits that sleep deprivation is similar to driving while intoxicated. In the first month postpartum, sleep loss puts them at risk for impaired cognitive functioning, postpartum depression, and negative interactions with other family members.

CASE STUDY

Ms. Knapp is a 28-year-old Caucasian woman at 8 weeks postpartum who brought her infant into the pediatrics clinic for immunizations. She complains of fatigue and looks like she has not slept for a week, with her hair unwashed and uncombed, and food stains on her skirt. The infant is fussy but appears healthy and vigorous and gaining the expected weight. When asked how she is doing with being a new mother, she replies, "I just wish someone would knock me out with a sleeping pill or something so I can get some sleep." Upon further probing, she says the baby is sleeping and eating well, but that she has no time to take a shower or shop for groceries during the day, and she cannot fall asleep at night. She adds that now the baby is sleeping through the night, but she still cannot fall asleep, and says that her husband takes over baby care during the night so she doesn't have to be awakened.

Ms. Knapp should be screened for postpartum depression and treated appropriately with either antidepressant therapy or counseling. The nurse should inquire about her usual bedtime, how long it takes her to fall asleep, and how much sleep and physical activity she is getting. Ms. Knapp should be referred to psychiatry for further evaluation, and pharmacological treatment may be considered if symptoms do not resolve.

CRITICAL THINKING QUESTIONS

1. You cannot determine whether Ms. Knapp is depressed or just disorganized and unable to find time for her own grooming. Discuss how screening for sleep problems is related to postpartum depression. How would you approach her to assess for depression?
2. You are the charge nurse in a labor and delivery unit. Discuss how you would implement a sleep hygiene information program prior to discharging new mothers home with their infant. What key symptoms would you alert them to focus on if they are worried about getting postpartum depression?
3. What nursing strategies would you use to promote sleep during pregnancy or during postpartum in order to reduce the risk of postpartum depression?

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Sleep Promotion in Child Health Settings

17

Kristen Hedger Archbold

Sleep is one of the most important functions of normal growth and development during childhood. Unfortunately, children's sleep disorders often go undetected and untreated, and there is a wide gap between parent-reported sleep symptoms and the number of children who are diagnosed with these conditions (Meltzer, Johnson, Crosette, Ramos, & Mindell, 2010). For example, more than 12% of children experience symptoms suggestive of obstructive sleep apnea (OSA), yet studies report an OSA prevalence of only 3% in school-aged children (Archbold, Pituch, Panahi, & Chervin, 2002; Bhattacharjee, Dayyat, Kheirandish-Gozal, Capdevila, & Gozal, 2009), and parents report symptoms suggestive of sleep disorders in as many as 45% of children (Archbold et al., 2002; Meltzer, Johnson, Crossette, Ramos, & Mindell, 2009).

The gap between formal diagnosis of sleep disorders and the prevalence of symptoms might be explained by epidemiological strategies; but studies have also shown that health care providers rarely assess children for sleep disorders (Meltzer et al., 2010). Assessment and treatment of sleep and sleep disorders is an important, but often overlooked component of nursing care in all settings in which nurses encounter pediatric patients. The purpose of this chapter is to provide practical strategies for nursing care related to sleep promotion and prevention and treatment of sleep disorders in pediatric primary care settings, acute care settings, and schools. These strategies build on the information provided in Chapter 13 (Pediatric Sleep Disorders), Chapter 5 (Conducting a Sleep Assessment), and Chapter 2 (Developmental Aspects of Normal Sleep). Additional information about the critical elements of sleep assessment and treatment to include in well-child visits during the first 2 years of life is provided in a recently published text (Mindell & Owens, 2010).

PEDIATRIC PRIMARY CARE SETTINGS

Children seen in primary care settings may present with a wide variety of sleep disorders. The prevalence depends partly on the age and developmental stage of the child. (See Chapter 2, Developmental Aspects of Sleep and Chapter 13, Pediatric Sleep Disorders.) For example, a teen-aged patient who presents to the clinician with academic and social difficulties in school and sleeping during class may be assessed for delayed sleep phase syndrome (DSPS). Toddlers may present with bedtime resistance. A 4-year-old child who presents to the clinic with frequent sore throats and daytime behavior problems may have OSA. (See Chapter 13, Pediatric Sleep Disorders, for more information.) Because of the close association between children's sleep and development and the impact of sleep disorders on daytime function and behavior, it is important to understand the developmental stage of the child and to include both parents and guardians and the children themselves into assessment and treatment.

Sleep Health Assessment During a Well-Child Visit

The nurse in the pediatric primary care setting can readily incorporate sleep assessment into annual well-child visits. Detailed information

on sleep assessment is provided in Chapter 5. Parents are a critical source of information for infants and very young children. However, the nurse should ask both parents and older children about their sleep. It is important to question children directly whenever possible and to provide them with the opportunity to describe any episodes of waking during the night, difficulties with falling asleep at night, or any other concerns he or she may have about their own sleep patterns. Like most parentally reported data, the level of agreement between parent reports and child self-reports of sleep disturbance symptoms is often small (Goodlin-Jones, Tang, Liu, & Anders, 2009; Gregory, Van der Ende, Willis, & Verhulst, 2008). For example, a recent study found that children reported waking an average of at least once per night, while parents reported that the child was not waking up at all during the night (Ward et al., 2009).

The focus of assessment of sleep during a well-child visit is on patterns of sleep, potential disturbances, and the potential consequences of sleep disturbance. Use of the "BEARS" Framework for assessment as described in Chapter 5 is a helpful way to organize the interview and assessment. Another approach is based on the acronym "AAA": "*Ask*" patients and families about the presence of sleep disturbances, snoring, length of sleep periods, and so on; "*Assess*" physical characteristics that can be associated with sleep disorders in children; and "*Act*"—refer patient to sleep clinic when complex patterns of sleep disturbances suggest the presence of sleep-disordered breathing, nocturnal seizures, limb movements, and/or daytime behavior difficulties are present (see Tables 17.1 and 17.2).

Table 17.1 ■ *"3A" Mnemonic for Sleep Disorders Assessment*

"Ask"	"Does your child snore?" Determine the presence or absence of snoring. "Do you have any concerns about your child's behaviors when he or she naps, sleeps at night, or prepares for bed?" Determine presence of napping that is age-inappropriate, tossing and turning, sleepwalking, night terrors, enuresis, or bedtime resistance. "Can you tell me about the room your child sleeps in and what activities other than sleep generally occur there?" Determine the nature of the sleep environment at home. "Is it difficult to wake your child up in the morning?" Determine presence of excessive daytime sleepiness and potential sleep disturbances such as OSA, PLMD, seizure activity, and so on.
"Assess"	Growth Patterns: Height, Weight, BMI percentile for age, developmental milestone achievement, Tanner stage General Impressions and Appearance: Alertness of child, irritability, hyperactivity, fatigue Head and Throat Exam: Head, Eyes, Ears, Nose, and Throat (HEENT), assess size of tonsil, shape of upper hard palate, symptoms of atopic dermatitis, allergies Neurological Exam: when nocturnal seizures may be suspected or severe, daytime sleepiness is reported and/or observed. Serum measurements: ferritin and lead levels (request these levels are measured if no previous data available and sleep disorder such as PLMD is suspected)
"Act"	Refer for in-lab PSG if: OSA or PLMD are suspected. excessive daytime sleepiness is present nocturnal behaviors such as seizures or chronic, repeated violent behavioral episodes are reported. Give parent and patient (as age permits) a 2-week sleep diary to complete at home and schedule return visit for follow-up to discuss appropriate treatment options for DSPS, behavioral insomnias if suspected. Discuss sleep hygiene and how to optimize sleep environment. Provide information and educational materials to child and parents. Refer to sleep clinic with pediatric expertise for further evaluation of complex sleep-related disturbances.

Source: Pediatric Sleep: Diagnosis and Management of Sleep Problems (2nd ed.), by J. A. Mindell and J. A. Owens, 2010, Philadelphia: Wolters Kluwer. Adapted with permission.

Table 17.2 ■ *Anticipatory Guidance for Sleep Issues in Children and Adolescents During Well-Child Visits*

Time Frame of Visit	Content
3–5 Years	Discuss: ■ Normal development of sleep and napping patterns ■ Effects of inadequate sleep on daytime behavior ■ Signs of sleepiness ■ Development of good sleep habits ■ Parental limit setting Discourage: ■ TV viewing at bedtime ■ TV or other screen-related media (i.e., computers, video games) in the child's bedroom
6–12 Years	Discuss: ■ Normal developmental sleep needs for school-aged children ■ Impact of inadequate sleep on school performance ■ Strategies to develop good sleep habits Encourage: ■ Adequate sleep ■ Appropriate bedtime Discourage: ■ TV at bedtime ■ TV or other screen-related media (i.e., computers, video games, text messaging) in the child' bedroom in bedroom ■ Use of electronic (e.g., computer) media within 2 hours of bedtime ■ Caffeine use
Adolescents	Discuss: ■ Average sleep needs (9–9 1/4 hours) in teenagers Encourage: ■ Obtaining adequate sleep and appropriate bedtime ■ Prioritizing school, leisure, sports, social, and work activities to assure adequate time for sleep Discourage: ■ Irregular sleep schedules ■ TV or other screen-related media (i.e., computers, video games, text messaging) in the child's bedroom Review: ■ Pubertal influences on sleep (phase delay) ■ Healthy sleep habits (regular bedtimes and waketimes, avoidance of caffeine, avoidance of TV set/computers in bedroom)

Source: Pediatric Sleep: Diagnosis and Management of Sleep Problems (2nd ed.), by J. A. Mindell and J. A. Owens, 2010, Philadelphia: Wolters Kluwer. Modified with permission.

A general way to begin a pediatric sleep assessment is with questions such as: "Do you (ask the child directly when possible) have any concerns about you/your child's sleeping routines or habits?" and "Does your child get at least (age-appropriate amount) hours of sleep each night?" (Chervin, Hedger, Dillon, & Pituch, 2000). Discussion and assessment of a child's sleep routines and habits should include determination of the presence or absence of enuresis, active tossing and turning or limb twitching during sleep, sleep-related behaviors such as sleep-walking, sleep-talking, and bruxism, and patterns of sleep/bedtimes and rise-times. Verbal questioning with these issues in mind is meant to elicit symptoms suggestive of periodic limb movement disorder (PLMD), parasomnias (sleep-walking, talking, bruxism), and DSPS or other behaviorally related insomnias (Chervin et al., 2002).

Careful assessment of the child's behavior and functioning at school and at home is an important component of sleep assessment. Children with sleep disorders are more likely to have difficulty with falling asleep in school and may have hyperactivity and behavioral problems or excessive daytime sleepiness. Sleep disorders are also common among children who have developmental disorders and attention deficit hyperactivity disorder (ADHD).

Given the high prevalence and profound consequences of OSA, it is critically important to assess for its symptoms. The question: "Does your child ever snore?" is an excellent way to begin assessment and discussions regarding pediatric OSA in any practice setting (Chervin et al., 2000). An affirmative answer to this question should prompt further questioning: "Does the snoring happen most nights, nearly every night?" "Does your child have any concentration difficulties or behavioral issues such as difficulty sitting still or resisting going to bed at night?" Although affirmative responses to these questions are suggestive of OSA, positive answers to these questions do not guarantee a diagnosis of OSA (Carroll, McColley, Marcus, Curtis, & Loughlin, 1995). Questionnaires may also be helpful in screening, but the diagnosis can only be made with polysomnography (PSG). (See Chapter 5, Conducting a Sleep Assessment.)

A detailed description of physical assessment related to sleep disorders in children is provided in Chapters 5 and 13. The primary focus is assessment of anthropometric characteristics, otolaryngological structure and function, and craniofacial morphology. Neurological examination may also be indicated, and, depending on the sleep disorder suspected, screening for serum ferritin and lead levels may be performed.

To Refer or Not to Refer?

Nurses who practice in pediatric primary care settings should feel comfortable in referring children who are suspected of having OSA, periodic limb movements during sleep, narcolepsy, or other complex sleep disorders to sleep disorders centers that have pediatric sleep specialists. Personnel in the sleep disorders center will further evaluate the child for specific sleep disorders and may suggest an overnight polysomnographic evaluation in the sleep laboratory setting or in the home. (See Chapter 5, Conducting a Sleep Assessment for more information on pediatric polysomnography and information regarding what families should expect in these settings.) There is, at the present time, no screening tool, questionnaire, or set of questions that can positively diagnose OSA or other sleep disorders, and only clinical and/or in-home PSG can provide or rule out this disorder.

The American Academy of Sleep Medicine (AASM) has not yet adopted guidelines on when to refer a child to a sleep center for further evaluation for OSA or other sleep disturbances (Miano, Paolino, Castaldo, & Villa, 2010). Similarly, there are no formal AASM guidelines to determine when children should have a postadenotonsillectomy PSG to determine the extent to which OSA has been improved with surgery.

Treatment of Sleep Disorders

Insomnia

Advanced practice nurses can effectively manage behavioral insomnias of childhood (BIC) and DSPS (see Chapter 10, Circadian Rhythm Disorders) in primary care settings (Morgenthaler et al., 2006). Sleep diaries are very helpful in evaluating sleep patterns and should be used for 2 weeks to provide a baseline for treatment. Sleep hygiene and cognitive behavioral therapy (CBT) are important treatment modalities that can be administered by advanced practice nurses. (See Chapter 13, Pediatric Sleep Disorders.) These CBT-I treatments should include the parents and family who are present in the child's sleep environment (Morgenthaler et al., 2006) and a developmentally appropriate approach. It is important to remember that insomnia symptoms and frequent nightmares in children may also be signs of underlying psychiatric mental health disorders (e.g., ADHD,

anxiety, and depression) and therefore, may be triggers to initiate referral to psychiatric or psychological providers for further evaluation and possible treatment.

Medications for Management of Insomnia

Although prescription medications are sometimes prescribed for pediatric sleep disorders, only chloral hydrate has been approved by the FDA for its hypnotic effects, but it is not recommended by sleep specialists. Emerging evidence suggests that a combination of behavioral therapy and hypnotic medications may eventually become the most helpful pathway of intervention for the child with insomnia (Owens, Rosen, Mindell, & Kirchner, 2010), but evidence from randomized clinical trials is still accumulating. In the meanwhile, critical thinking must guide the nurse in efforts to make behavioral modifications with ongoing patient education, the primary mode of nursing intervention for their pediatric patients with behaviorally related sleep disturbances.

Parasomnias

Children often have benign parasomnias (e.g., sleep terrors, occasional nightmares, sleepwalking, sleep talking) that may naturally resolve as the child develops. (See Chapter 8, Movement Disorders and Parasomnias and Chapter 13, Pediatric Sleep Disorders.) Primary care nurses can provide support, reassurance, and education, to the children and families with these problems. (See Chapter 13, Pediatric Sleep Disorders and Chapter 8, Sleep-Related Movement Disorders and Parasomnias.) The primary care nurse can also assess the time-course, presence and disappearance of parasomnias at follow-up visits in order to insure referral for further evaluation of unresolved parasomnia issues as may be necessary.

The primary care nurse should provide referral for PSG for parasomnias during which seizure activity is suspected (i.e., reports of stereotypical movements, lip smacking, drooling during a sleep-walking episode) or if enuresis and symptoms of OSA are present. Both primary (ongoing since birth) and secondary (sudden re-emergence after a time of absence) enuresis are associated with OSA in children (Spruyt et al., 2006).

Movement Disorders

Detailed information on movement disorders is provided in Chapter 8, Sleep-Related Movement Disorders and Parasomnias and Chapter 13, Pediatric Sleep Disorders. If a nurse suspects the presence of a movement disorder (PLMs or RLS), and symptoms of OSA are also present (i.e., frequent snoring, reports of witnessed apneas, daytime behavioral issues, scholastic problems, etc), then referral for PSG should be provided (Chervin et al., 2002). Assessment of scholastic and social behavioral issues should be made at each follow-up visit in order to document any changes or improvement in the child's daytime symptoms.

Medications for Management of RLS and PLMD

The primary care practitioner can coordinate management of any medications used to treat PLMD and RLS, and monitor the potential side effects of such compounds on a child's behavior and sleep. The medication that is most commonly prescribed for treatment of limb movement disorders in children is elemental iron. A dose of 5 mg/kg is the standard therapeutic dose given to children if their serum ferritin levels are lower than the targeted level of 50 (Simakajornboon, Kheirandish-Gozal, & Gozal, 2009). However, this should be used cautiously because of the risk of overdose. Dopaminergic agonist drugs are used in low doses to treat severe excessive limb movement patterns during sleep (Simakajornboon et al., 2009). (See Chapter 8, Movement Disorders and Parasomnias.) Dopamine agonists such as levodopa, ropinirole, pramipexole, and pergolide should be administered to children only in severe cases and when frequent follow-up visits (every 6–8 weeks) are possible because long-term effects in children are not well known (Simakajornboon et al., 2009).

Patient/Family Education Regarding Sleep

Education of children and parents is an important component of sleep promotion and also a component of the "*Act*," portion of the "*AAA*" acronym described above and in Table 17.1. Educational information should include an explanation of normal sleep consistent with the child's developmental stage and description of sleep disturbances that may occur frequently in normal development. For example, some sleep disorders, like sleep terrors or sleep talking, may be benign and resolve as the child develops. Parents should also be encouraged to report any snoring they notice in their child as snoring is not necessarily benign in children. Table 17.1 lists appropriate advice regarding normal sleep and healthy sleep behaviors according to developmental levels.

Sleep hygiene strategies are an important foundation for the treatment of many sleep disorders and important components of sleep promotion in children and families. Sleep hygiene education includes emphasis of the need for consistent bedtime and rise-times, regardless of the day of week or time of year (Mindell, Meltzer, Carskadon, & Chervin, 2009) and characteristics of normal sleep consistent with the child's developmental stage. Parents should be instructed to use a calming and reassuring approach and bedtime routines with their children that begin approximately 60 minutes prior to the desired sleep onset time. Routine cues established in the environment signal to the child that it is "time for bed" (e.g., closing of shades in bedroom or hallway, putting items from evening bath activities away in their proper locations). All electronic screen-related activities (computer games, text messaging) should be finished and put away a *minimum* of 2 hours prior to desired sleep onset. The ideal sleeping environment is dark and cool (66°–68° ambient temperature) whenever possible (Peixoto, da Silva, Carskadon, & Louzada, 2009). Education about the need to avoid caffeine and guidance on caffeine content of foods and beverage is an important component of this behavioral approach. (See Chapter 6, Insomnia and Chapter 10, Circadian Rhythm Disorders.)

Social and educational demands placed on children often limit the time available each night for sleep. Unfortunately, children's optimal sleep periods are often reduced in order to accommodate extracurricular and social activities such as team sports or employment (Touchette et al., 2007). Parents can be encouraged to structure their child's social environment around age-appropriate sleep needs and reduce extraneous extracurricular activities that may reduce the optimal sleep period length. Families can make a full night's sleep a priority activity within the household, and parents can be encouraged to model appropriate sleep hygiene techniques for their children in their own busy daily routines.

Children's bedtimes can also be influenced by parental work schedules. Therefore, it is helpful to ask about parental work schedules whenever late sleep onset times are present (Gau & Merikangas, 2004). Parents who come home from work late in the evening should be discouraged from keeping their children up past an appropriate bedtime in order that the parent can spend more time with the child each day. Parents will discover that the quality of time with their children is improved when the children have a good night's sleep and are well rested. This may contrast with the quantity of time with children who may suffer from the effects of sleep loss.

Families with children and adolescents face significant challenges related to sleep behavior on a nightly basis. Nurses can discuss these important issues with patients and families in the spirit of understanding that healthy and optimal sleep patterns provide key support for the growth and development of their child.

Children Who Have Special Needs

Structure and routine are also essential components of successful bedtime preparations (Andersen, Malow, & Barnes, 2007; Doo & Wing, 2006), especially in children who have special needs related to cognitive ability (e.g., Autism, Down's Syndrome). It may be helpful to suggest that parents take photographs

of their child enacting the desired activity at each stage in the bedtime preparation process (i.e., child brushing teeth, child putting on pajamas, child tucked into bed, child actually asleep). These pictures can then be laminated on cardstock for repeated use and shown to the child at the actual stage of the bedtime routine to remind them of the routine and what will occur at each step.

Follow-Up Care

CPAP is the second line of treatment for children who have OSA. (Removal of adenoid and tonsilar tissues is the first). (See Chapter 13, Pediatric Sleep Disorders.) CPAP is prescribed and titrated in the sleep center. Once this treatment is established, nurses in pediatric primary care settings will work with children who are using CPAP and their parents. Adherence and care of CPAP equipment at home (cleaning schedules, importance of cleaning, mask care) are two of the main issues nurses can support in the primary care setting. One study describes parental involvement and skilled nursing staff as crucial to ensuring a child's success with PAP therapy (Massa, Gonsalez, Laverty, Wallis, & Lane, 2002). The study also reported that common side effects of PAP therapy in children were nasal dryness and irritation from mask placement. Neither of these was found to be sufficient enough for discontinuation of therapy. Continued education regarding the need for adherence and clarification about parental concerns with the CPAP treatment is an important component of the nursing role.

Although much is known about issues that negatively affect CPAP adherence in adults with OSA, there is little, if any, data available about what these issues may be for children who use CPAP. Much more research needs to be done to determine the efficacy of CPAP in pediatric populations, and how to best support optimal adherence patterns for these children. Research is ongoing to determine the therapeutic effects of CPAP on sleep patterns, behavior, and cognition in children with OSA.

Upwards of 60% of children may have some degree of OSA after adenotonsillectomy that often requires further treatment with CPAP. It is important for parents to be aware that adenotonsillectomy may not completely cure the OSA and that PSG is needed to determine the extent to which OSA has been addressed by the surgical procedure.

ACUTE CARE SETTING

Acute care hospitalization is stressful for children and parents due to concerns about the illness, treatment, and prognosis. During hospitalization, children (and often parents) must sleep in an unfamiliar environment associated with noise from other patients, equipment, and health care personnel, as well as less than optimal patterns of lighting. The pathophysiology of the illness, pain, discomfort, and treatments may all contribute to poor sleep in children and their families.

Providing Comfort and Managing the Environment

Hospitalized children experience disturbed sleep patterns in the same ways they do at home, but problems may be magnified by the illness and treatment setting (Meltzer, Davis, & Mindell, 2008). Behavioral and environmental modifications are similar to those provided to adults with sleep disorders, but may need some specialized adaptations. Therefore, environmental modifications and assuring comfort, reassurance, and anxiety reduction are important components of nursing care.

Hospitalized children may benefit from dim lights 1 hour prior to normal home bedtime, darkened room, in-room monitors quieted or muted (as permissible) at the same time as the home bedtime (Meltzer et al., 2008). Care of the pediatric patient during the sleep period should be limited and clustered to minimize interruptions. Pain control should be maximized to facilitate comfortable sleep whenever

possible. Having a parent or care provider sleep in the room with the child may be an effective comfort and sleep-promotion mechanism. However, while there is a large body of research on this topic in settings in which adults are hospitalized (see Chapter 20, Sleep in Adult Acute and Critical Care Settings), there is little scientific evidence to support these strategies in children.

Obstructive Sleep Apnea

Children who use CPAP at home should be instructed to bring the CPAP equipment with them for use during hospitalization. Great care must be taken not to have a parent who has untreated OSA (i.e., loud snoring, apneaic pauses) sleep in the room with the child, as the child's sleep will likely be interrupted by the sounds of the snoring parent. Parents who use CPAP should also be encouraged to bring their equipment to the hospital if planning to stay overnight in the child's room.

Anesthesia, sedatives, and opioid medications present special concerns for children who are at-risk for or may already have been diagnosed with OSA, given their potential to cause respiratory depression. It is particularly important to assess children for the presence of OSA prior to administering these treatments. Obese children who are scheduled to undergo adenotonsillectomy may be at particular risk for respiratory complications (Brigance et al., 2009). Postanesthesia care nurses must be particularly vigilant in monitoring respiratory patterns in these children because referring surgeons may not have diagnosed the child with OSA prior to surgery (Weatherly, Ruzicka, Marriott, & Chervin, 2004). Obese children with OSA are at higher risk for postoperative complications, including serious bleeding, reintubation, respiratory failure, pulmonary hypertension (Sterni, Tunkel, & Heitmiller, 2009), than their average weight counterparts (Robb et al., 2009).

As in adults, postanesthesia and emergence from anesthesia are particular risk periods for respiratory complications in children (Punjabi et al., 2009). Nurses can obtain a thorough patient sleep history prior to surgery in order to elicit symptoms of OSA which may not have been previously identified. All surgical-team personnel can be alerted to the presence of these symptoms in the child, so close monitoring of pre- and post-surgical respirations can occur. The postanesthesia care nurse should be alerted to the potential need for CPAP if a child has pre-existing OSA symptoms and have a standing therapeutic order available for the delivery of CPAP equipment to the unit if possible (Brigger, Cunningham, & Hartnick, 2010).

SCHOOL HEALTH CARE SETTINGS

School nurses are in a critical position to assess children for behavioral problems associated with poor sleep and to observe children for signs of OSA and other sleep disturbances. They often play important roles as leaders in educating children, parents, and teachers regarding sleep promotion and prevention of sleep disorders. Unfortunately, their numbers continue to dwindle as public education budgets become smaller and smaller.

The "AAA" acronym is useful for every child who is administered stimulant and/or other medications for ADHD. The simple screening question for parents of these children: "does this child snore?" may open the diagnostic door for treatment of an underlying, undiagnosed sleep disorder that is at the root cause of a child's behavioral issues in the classroom (Chervin et al., 2000). A good example of a child who is at risk is the one who is medicated with stimulants for a diagnosis of ADHD, obese, falls asleep in class, or has other classroom behavioral difficulties.

Table 17.3 provides suggestions about ways in which school nurses can raise awareness among students, parents, teachers, and school administrators regarding the importance of sleep and strategies to develop "sleep-friendly" schools and communities. Important emphases include raising awareness about normal

Table 17.3 ■ *Strategies for Sleep Promotion in School-Health Settings*

- Initiate formal discussions in the school setting about the necessity and importance of sleep in the health and development of a growing child.
- Implement age-appropriate start-times for school days. Bus sharing is common among public school districts and can be assigned based on physiological sleep needs of the children, the district serves. The youngest of children may need to start at the earliest times. They may have less homework and extracurricular activities, and so, may be more easily encouraged to sleep earlier in the night, and start school the earliest times the following morning; middle-school should be given second-shift start times; adolescents should have the latest start times.
- Emphasize the importance of adequate sleep to promote cognitive performance, learning, and behavior. This provides an important foundation for elementary and secondary school, but also for preparation for college. Obtaining a full night of sleep has beneficial effects on retention of information and performance on tests. Staying up late at night to "cram" for examinations should be discouraged.
- Work with teachers and administrators to improve understanding of the benefits of adequate sleep and the potential influence of sleep disorders on children's behavior (e.g., irritability, restlessness, ADHD, daytime sleepiness)
- Work with teachers, families, and students to promote "sleep-friendly" scheduling of academic, sports, work, and social activities.
- Utilize public health strategies to promote adequate sleep.

sleep needs, circadian rhythms in childhood and adolescence, age-appropriate sleep duration guidelines, and the need to be aware that sleepiness, behavioral, and school performance issues may be signs of sleep disorders. Information should also be included in health education curricula.

School start times influence patterns of cognitive and scholastic performance in children. When high school aged children begin school at 9 AM or later, scholastic performance improves and absenteeism decreases (Mindell et al., 2009). School nurses may be in a direct position to affect school start times in their school districts, and can initiate and facilitate discussions that may lead to age and circadian-rhythm appropriate start times for elementary, middle and high school age groups for students.

SUMMARY

Pediatric sleep disorders are common, underdiagnosed, and often not effectively treated. In children with sleep disorders, inadequate sleep does not often result in excessive daytime sleepiness, but in behavioral difficulties such as inattention, hyperactivity, cognitive dysfunction, and/or scholastic problems. Because ADHD and other psychosocial mental illnesses are common pediatric diagnoses that mimic the symptoms of OSA and PLMD or RLS, assessment and evaluation of sleep patterns should always be performed prior to diagnosis of psychiatric conditions.

There are many opportunities to promote healthy sleep and to screen, evaluate, and treat sleep disorders in primary care, acute care, and school health settings. Nurses who see children in the primary care setting can take an active role in the evaluation and assessment of all children's sleep health and provide follow-up care and ongoing treatment monitoring for children who have sleep disorders. In the acute care setting, nurses can incorporate the regular treatment plans for a child's sleep disorder during hospitalization and should be aware of the potential for OSA-related perioperative complications for children undergoing adenotonsillectomy or other surgical procedures. School nurses have the opportunity to promote healthy sleep and improve behavior and school performance in children at risk. All nurses at the practitioner, school, and policy making levels can increase the amount of importance and attention given to the pediatric sleep health promotion strategies in order to provide the most appropriate strategies for intervention to insure a good night's sleep is obtained by all children (Table 17.4).

Table 17.4 ■ *Web-Based Resources*

Web site	Organization
www.aasmnet.org	**American Academy of Sleep Medicine (AASM)** One Westbrook Corporate Center, Suite 920, Westchester, IL 60154 (708) 492-0930
www.sleepfoundation.org	**National Sleep Foundation (NSF)**1522 K Street, NW, Suite 500, Washington, DC 20005 (202) 341-3471
www.narcolepsynetwork.org	**Narcolepsy Network** 79A, Main Street, North Kingstown, RI 02852 (888) 292-6522
www.rls.org	**RLS (Restless Legs Syndrome) Foundation** 1610 14th Street NW, Suite 300, Rochester, MN 55901 (507) 287-6465
www.sleepapnea.org	**American Sleep Apnea Association** A.W.A.K.E. Network 6856 Eastern Avenue NW, Suite 203, Washington, DC 20012 (202) 293-3650
http://www.kidzzzsleep.org/	Current information on sleep, sleep disorders, and the impact of sleep on health and behavior in infants, children, and adolescents to parents, pediatric health care and mental health practitioners, pediatric sleep researchers, and educators interested in pediatric sleep.

CASE STUDY 17.1 The Sleepy Teen

Your newest clinic patient is a 14-year-old female. She is an only child who lives at home with her mother and father. She comes to clinic with the complaint of daytime sleepiness. She can fall asleep very easily, often within seconds during her morning classes. Actually, this is the school's complaint and her mother's complaint—she is just here because "they" want her here. Patient reports not being bothered by her sleep problems other than it seems to be a problem for everyone else that she doesn't go to bed until 1 AM and would sleep until 12 PM each day if she could! Mother states this pattern of sleep and wake has been going on now for about 6 months, and her school grades are beginning to decrease from their usual "A" levels. When patient falls asleep in her classes, she reports having very real, weird, or frightening dreams that cause her to startle and wake herself back up (if the teacher doesn't do it first). Patient reports drinking two (2) 16 oz of energy drinks during lunch at school to help her "stay awake."

Further questions reveal that the patient does not snore, have periods of apnea, or problems with inattention at school (other than falling asleep in her morning classes!). Mother describes patient's bed linen as all twisted around and together. It appears to mom as though patient moves a lot during the night. Patient reports no sensations of discomfort in her legs, urgency to move them, or awareness of twitching during sleep. Patient denies falling down with laughing or during intense emotional periods. Patient denies lying awake in bed and feeling like she cannot move. When patient's mother sends her to bed early, she tosses and turns (thus, the messed-up sheets) until 1 AM when she finally falls asleep. Mother must wake patient with three attempts in order to awaken her for school every morning. Patient does not report wake periods during the night after she falls asleep. Patient will take a 60-minute nap 3 days a week when getting home from school. Patient reports going to bed at 2 AM on weekend nights, and sleeps until 2 PM the following afternoon. Patient appears healthy and in no acute pain or distress, except that she seems tired, pale, lethargic, and uninterested in what's going on around her today. Mom states patient is "doing this on purpose, just to make her father and I mad." Mother's job is an accounting professional during daytime hours, and Father is an industrial engineer who has worked the night shift at the local assembly plant for the past 10 years.

Patient's BP is 98/62, and BMI is 27—which puts her in the 75th percentile for her age. Patient's onset of menarche was 2 years ago and her LMP was 24 days ago. Patient does not report heavy flow or other problematic issues with her menstrual periods. Patient tonsils are 1+ bilaterally and other craniofacial morphology exam is negative. Other physical exam findings are unremarkable. Energy drink reported as consumed by patient contains approx, 120 mg caffeine. Patient reports having two of these drinks every day around 11:30 AM (Google.com search for caffeine content in drink).

Impressions. Patient is complaining of excessive daytime sleepiness. Patient does not complain of scholastic difficulties other than falling asleep in her morning classes. Patient is negative for snoring, witnessed apneas, has 1+ tonsil, and is mildly overweight. Bed linen is twisted around, and patient attributes this to tossing and turning in her bed until she finally falls asleep at around 1 AM each night. Patient denies discomfort in her legs or feeling an urge to move her legs at night. Patient is unaware of any twitching during sleep. Patient reports no significant issues with her menstrual period. School begins daily at 7:30 AM and patient must arise at 6:15 AM in order to prepare for arrival at school on time. Patient stays awake until 2 AM on weekend nights and sleeps until 2 PM the following afternoon. Patient drinks 240 mg of caffeine at 11:30 AM during school days in an attempt to "stay awake the rest of the day." Patient will take a 60-minute nap on 3 days after school each week. Patient reports vivid dreams and falling asleep easily during her morning classes, but does not report cataplexy or sleep paralysis.

Critical Thinking Questions

1. What symptoms in the patient's clinical presentation indicate a formal sleep disorder? Based on your assessment, formulate a plan to address the needs. Articulate the specific dimensions of the plan.
2. In your comprehensive assessment, you discover that the patient has anemia. What relationship exists between anemia and formal sleep disorder(s)? How does the finding of anemia shift your plan of care, if at all?
3. How does the patient's sleep/wake schedule differ from weeknights to weekend nights and what are the implications of these changes for overall sleep health?
4. From the perspective of circadian phase, describe the patient's sleep/wake pattern. What is the plan for intervention with this sleep/wake pattern, if any?
5. How does the patient's pattern of napping potentially affect her overall sleep/wake pattern? What is your teaching plan? What evidence base do you use for your plan?
6. In what ways might the parent's work schedules affect your diagnosis for this patient?
7. What symptoms are suggestive of a psychiatric condition? What is your intervention plan, if any?
8. Which sleep disorder(s) do you think might be the most appropriate for consideration in this patient's case?
9. How would you further assess symptoms to make your diagnosis of the suspected sleep disorder(s)?

This patient has DSPS, not narcolepsy. Would send her home with actigraph and sleep diary for 2 weeks to confirm. I would send a diary with parent for parental observations, to increase likelihood of substantiation of actigraphy findings. Review of the collected data to document DSPS symptoms and plan to begin treatment through advancement of sleep phase with sleep restriction, chronotherapy, and then promotion of sleep hygiene. May consider referral to behavioral health specialist if mood levels fluctuate or become problematic; a specialist for both mother AND daughter!

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Sleep and Primary Care of Adults and Older Adults

18

Catherine S. Cole

Clients seen in primary care settings spend approximately one-third of their life asleep. Unfortunately, 50–70 million Americans may have some type of sleep disorder and, for the majority, those sleep disorders are chronic (Institute of Medicine [IOM], 2006). "All types of sleep disorders probably exist in a higher prevalence among the primary care client population than in the general population, and very likely at higher levels of severity" (Dement & Netzer, 2000, p. 1). For example, epidemiologic studies suggest that although about 30% of the general population report insomnia, 50% of adults and older adults in the primary care setting report it (National Institutes of Health, 2005). The high prevalence of sleep disorders seen in the primary care setting is likely due to the fact that clients see their primary care providers because of the presence of chronic and acute conditions that place them at highest risk for sleep disorders (Dement & Netzer, 2000).

The association between sleep disorders and serious medical conditions is bidirectional (Bloom et al., 2009). Primary care clients with disordered sleep describe distressing symptoms, but they may not be aware that there are also a wide range of downstream health consequences, such as increased risk for hypertension, diabetes, obesity, depression, heart attack, and stroke (Bridges, 2006; IOM, 2006; Katz & McHorney, 2002). Conversely, coexisting health problems and iatrogenic treatment effects can lead to disordered sleep. Investigators have found that those at highest risk for sleep disorders often have painful conditions, mental illness, limited activity, and poor mental and physical health (Alattar, Harrington, Mitchell, & Sloane, 2007).

Insomnia is the most frequently reported sleep disorder, but 55%–73% of those with insomnia report that they do not discuss sleep with their primary care providers, thereby eliminating the possibility of optimal treatment (Ohayon, 2002). Likewise, The American Sleep Apnea Association indicates that 12 million Americans may have obstructive sleep apnea, but 10 million remain undiagnosed (Institute for Clinical Systems Improvement, 2010). The reported prevalence of restless legs syndrome ranges from 2.5% to 15% of the general population and increases with age (Zucconi & Ferini-Strambi, 2004). However, only 12.9% of those seeking health care for symptoms related to restless legs syndrome receive an accurate diagnosis and fewer receive adequate treatment (Hening et al., 2004). Regardless of the etiology or prevalence of specific sleep disorders, the consistent message is that sleep disorders are underdiagnosed, and it can be assumed that they are not effectively treated (IOM, 2006; Ram, Seirawan, Kumar, & Clark, 2010). When clients are misdiagnosed or undiagnosed, sleep disorders are likely to become more severe (Dement & Netzer, 2000). Primary care providers, including nurses and advanced practice nurses (APNs), are in the ideal position to screen, evaluate, and manage these problems. The purpose of this chapter is to describe nursing care for sleep disorders in the primary care setting.

ROLE OF THE NURSE IN THE PRIMARY CARE SETTING

The high prevalence of sleep disorders and their under-detection suggests the critical role of primary care providers in screening, assessment, treatment, and referral for these problems. Primary care providers must be able to provide evidence-based individualized care that includes screening, assessment, and treatment in a limited amount of time, recognize when to refer clients to sleep specialists, incorporate careful follow-up for chronic sleep conditions after treatment by sleep specialists, assess for the interactions between sleep disorders, comorbid conditions, and medication regimens (Bloom et al., 2009), and assist clients to prevent the negative consequences of sleep disorders, such as medical and psychiatric disorders, decrements in cognitive performance, and excessive daytime sleepiness. All clients, especially those with symptoms that are resistant to standard therapies, deserve skilled assessment and interventions that incorporate sleep promotion strategies even when they do not report specific sleep complaints.

In addition to direct patient care, the APN can implement and evaluate practice-based and system-wide changes through development of organizational systems of care and processes that reinforce and support incorporation of sleep promotion strategies on an ongoing basis. These changes might include provider education, protocols, computerized reminders, audits, and client education materials. Incorporation of sleep promotion into primary care settings is likely to lead to more timely, efficient, and cost effective care, while more consistently improving client outcomes. Addressing these factors at the practice or health system level is more likely to lead to maintenance of sleep promotion practices than focusing only on individual provider practice.

RECOMMENDATIONS FOR PRIMARY CARE PRACTICE

Sleep Assessment

Evaluation of sleep should be a standard component of a comprehensive review of systems. This discussion is frequently omitted unless the client identifies sleep as a problem (Kushida et al., 2000). In fact, medical record reviews conducted in primary care settings have documented the magnitude of this problem. The National Sleep Foundation found that 6 in 10 primary care physicians, nurse practitioners, and physician assistants reported that they did not have time to routinely discuss their clients' sleep during regular office visits (National Sleep Foundation, 2008). Discussion of sleep occurs most frequently when clients report distressing symptoms related to sleep disorders such as trouble falling and staying asleep (79%), depression, anxiety, or stress (76%), difficulty staying asleep (74%), and trouble falling asleep (70%) (National Sleep Foundation, 2008). Unfortunately, when clients present with long problem lists, sleep disorders may not be viewed as a high priority and opportunities to improve sleep-related outcomes are missed. Another barrier to incorporating sleep assessment and treatment into primary care is lack of knowledge among some providers.

The importance of routine assessment of sleep and associated daytime dysfunction is underscored by the fact that clients may not always acknowledge the importance of sleep. (Dement & Netzer, 2000). For example, older adults may believe that poor sleep is to be expected with aging and that they require less sleep than younger adults. This belief is contrary to recent findings that the oldest old (80–100 years) are more likely to have good sleep than the young old (65–79 years) (Gu, Sautter, Pipkin, & Zeng, 2010) and that poor sleep in the elderly is usually associated with comorbid illness and not aging per se. (See Chapter 2, Developmental Aspects of Normal Sleep.)

Incorporating a structured approach to sleep assessment into routine patient encounters is likely to improve ability to detect sleep-related problems. An example of a structured interview is provided in Table 18.1. Case study 18.1 is an example of an assessment conducted in the primary care setting. Other examples of structured sleep assessments that are appropriate for the primary care setting, such as the BEARS nomenclature, can be found on the American Academy of Sleep Medicine Web site (Table 18.2) by using the search feature. The BEARS acronym stands

Table 18.1 ■ *Questions to Ask in a Structured Sleep Assessment*

- What time do you go to bed at night and wake up in the morning?
- Do you often have trouble falling asleep at night?
- About how many times do you wake up at night?
- If you do wake up during the night, do you have trouble falling back asleep?
- Does your bed partner say (or are you aware) that you snore, gasp for air, or stop breathing?
- Does your bed partner say (or are you aware) that you kick or thrash about while asleep?
- Are you aware that you ever walk, eat, punch, kick, or scream during sleep?
- Are you sleepy or tired during much of the day?
- Do you usually take one or more naps during the day?
- Do you usually doze off without planning to?
- How much sleep do you need to feel alert and function well?
- Are you currently taking any type of medication or other preparation to help you sleep?

Table 18.2 ■ *Web Sites for Primary Care Patients and Health Professionals*

Information for Patients

National Heart Lung and Blood Institute/National Center for Sleep Disorders Research

- http://www.nhlbi.nih.gov/about/ncsdr/index.htm

National Sleep Foundation (many patient materials)

- http://www.sleepfoundation.org

American Family Physicians (searchable sleep-related topics, including video and text on major sleep disorders and treatment)

- http://familydoctor.org

American Family Physician journal, October 1, 2005

Patient handouts "information from your family doctor" insomnia, sleep apnea, sleep in older adults

- http://www.aafp.org/online/en/home/publications/journals/afp/afpsearch.html (available free online)

Information for Health Professionals: Professional Development, Practice Guidelines, Assessment tools

American Academy of Sleep Medicine

- http://www.aasmnet.org/ProfDev.aspx.
- http://www.aasmnet.org/MedSleep.aspx

National Sleep foundation (many resources, including statistics)

- http://www.sleepfoundation.org/sites/default/files/sleepdiscussionguide.pdf

Questionnaires & Assessment Tools

Berlin Questionnaire

- https://www.swclab.com/images/PDFS/Berlin-Questionnaire.pdf

Sleep Diary

- http://www.sleepeducation.com/pdf/sleepdiary.pdf

Epworth Sleepiness Scale

- http://epworthsleepinessscale.com

for Bedtime, Excessive sleepiness, Awakenings, Regularity, and Snoring and provides a user friendly organized approach to sleep assessment. The *Sleeping Smart Discussion Guide* is a brief six-item guide that can be distributed in the waiting room and then used as a guide to facilitate discussion of sleep disorders between the primary care client and provider (National Sleep Foundation, 2010). It is available for no cost on the National sleep Foundation's Web site (see Table 18.2). Additional helpful Web sites are summarized in Table 18.2.

The use of a sleep diary for 2 weeks assists the provider to identify patterns in the client's sleep and to help the patient identify their own patterns (Barthlen, 2002). (An example is provided in Chapter 5, Conducting a Sleep Assessment.) Sleep diaries are also available on many Web sites for free. A Web link is provided in Table 18.2. Use of a diagnostic algorithm for evaluating sleep disorders is helpful in guiding differential diagnosis (i.e., insomnia, sleep apnea, restless legs syndrome, etc.) and determining the need for referral to the sleep laboratory for further evaluation (see Figure 18.1) (Bloom et al., 2009).

Sleep Disorders In Adult Primary Care Settings

The most prevalent sleep disorders in adults and older adults, and those most commonly seen in primary care settings are insomnia, sleep-disordered breathing, and restless legs syndrome. A detailed description of each of these disorders and their associations with chronic medical and psychiatric conditions is provided in Chapters 6–12. Specific aspects of the approach to sleep disorders in adults and older adults who seek health care in the primary care setting are presented in succeeding sections.

Insomnia

Acute and chronic insomnia can be treated in the primary care setting. The questions listed in Table 18.1 are useful in establishing the diagnosis of insomnia. Insomnia does not require polysomnographic evaluation except to rule-out the influence of primary sleep disorders such as sleep apnea or periodic limb movements during sleep, precipitous arousals that occur with violent behavior, or as a follow-up if initial insomnia treatment is unsuccessful (Littner et al., 2003). The primary care provider must carefully explore all possible contributing factors, including medical and psychiatric conditions, medications, behavior, cognition,

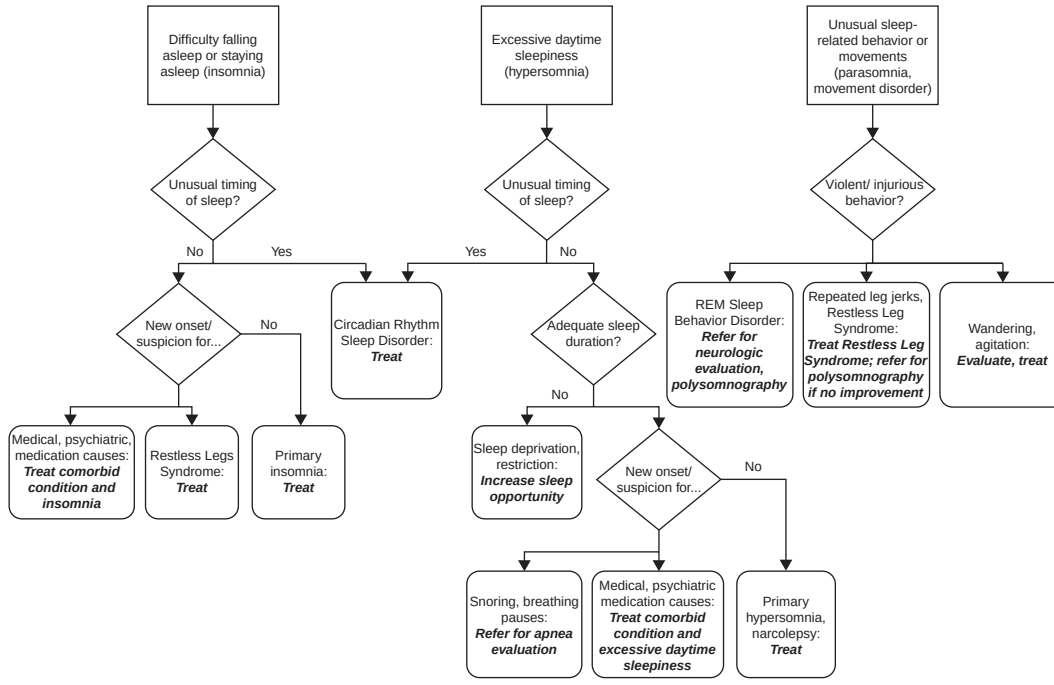

Figure 18.1 ■ Diagnostic algorithm for sleep disorders. *Source:* "Evidence-Based Recommendations for the Assessement and Management of Sleep Disorders in Older Persons," by H. G. Bloom, I. Ahmed, C. A. Alessi, S. Ancoli-Israel, D. J. Buysse, M. H. Kryger, et al., 2009, *Journal of the American Geriatrics Society, 57,* pp. 761–789. Reprinted with permission.

environment, cognition, circadian patterns, and primary sleep disorders in a structure manner. (See Chapter 6, Insomnia.) Given the high prevalence of comorbid conditions and associated medications in clients presenting in the primary care setting, especially older adults, comorbid insomnia is the most common form and may be multifactorial in origin (Bloom et al., 2009). Therefore, it is important for the primary care provider to consider all contributing factors and design a plan of care that prioritizes interventions and addresses them sequentially. Referral for psychological evaluation may be needed for insomnia patients who have complex comorbid psychiatric problems that require more intense intervention (Schutte-Rodin, Broch, Buysse, Dorsey, & Sateia, 2008; Pagel, 2007). Insomnia may also be the presenting symptom for a variety of psychiatric disorders. (See Chapter 12, Sleep and Psychiatric Disorders.) The primary care provider should consider referral when initial assessment indicates complex psychological factors or if initial treatment is unsuccessful.

Both pharmacological and behavioral methods are effective treatments for insomnia in primary care settings. (See Chapter 6, Insomnia.) However, hypnotics are most frequently used. One group of investigators reported that between the years 2006 and 2008, hypnotics were prescribed in 95 of 100 primary care insomnia cases (Charles, Harrison, & Britt, 2009). Hypnotics can be effectively used to treat short-term insomnia, but their long-term use is contraindicated, especially in older adults, and should be limited to 35 days or less. Only one medication (eszopiclone) was approved for use without a specified time limit, but significant safety concerns, including rebound insomnia, confusion, impaired memory, dizziness/falls, and fractures (Taylor & Weiss, 2009) limit its use by older adults (National Institutes of Health, 2005). The costs of medications and other treatments in the primary care setting are also a major concern. An overview of commonly used hypnotic medications and their approximate costs are listed in Table 18.3. Choosing the most efficacious drug with the lowest drug is a high priority.

Although pharmacologic treatment is highly effective, clients often prefer behavioral methods that have more durable effects (Morin et al., 2006) and do not have negative daytime consequences. Cognitive–behavioral therapy for insomnia (CBT-I), a multicomponent behavioral treatment that focuses on thoughts and behaviors that perpetuate chronic insomnia, is efficacious (Chesson et al., 1999; Morin et al., 2006; Riemann & Perlis, 2009), and nurses have successfully delivered it in primary care settings (Espie et al., 2007). Successful treatment of comorbid insomnia is not dependent upon first successfully treating the comorbid medical or psychiatric condition (Morin et al., 2006). Recent studies suggest that the magnitude of the effects of CBT-I are similar in adults and older adults (Morin et al., 2006), in contrast with an earlier Cochrane review that reported only small effects of CBT-I in people over 60 (Montgomery & Dennis, 2003). (See Chapter 6, Insomnia.)

The need for provider training and the time-intensive nature of individual or group counseling associated with CBT-I are potential barriers, especially in primary care settings. However, CBT-I can be successfully used in creative ways to improve efficiency. For example, CBT-I is effective when provided in individual or group format (Rybarczyk et al., 2005), as a home-based self-help intervention (Rybarczyk, Lopez, Benson, Alsten, & Stepanski, 2002), or delivered over the Internet (Ritterband et al., 2009). A service delivery model based on "stepped care" may address the issue of limited time and resources available to deliver CBT-I in primary care settings (Espie, 2009). For example, self-administered CBT-I delivered by booklet, CD/DVD, or Internet may be sufficient for patients with uncomplicated insomnia. Only a few clients may require the highest treatment intensity (referral for one-to-one tailored therapy over numerous sessions delivered by a certified behavioral sleep medicine specialist) (Espie, 2009). Figure 18.2 presents a stepped-care model for CBT-I that may be feasible in the primary care setting.

Although CBT-I holds promise for improving insomnia, there is a need for continued research into its efficacy and feasibility in busy

Table 18.3 ■ *FDA-Approved Hypnotics*

Class/Name	Onset of Action	Duration	Adult Dose (Dose in Elderly)	Cost +	Comments
Benzodiazepines[a]					
Flurazepam (Dalmane)	Intermediate (30–60 minutes)	Long	30 mg. (7.5 mg)	Generic $7.80 Dalmane $57.60	Should not be used in older adults because of very long half-life.
Quazepam (Doral)	Intermediate 20–45 minutes)	Long	15 mg. (7.5 mg)	Doral $131.40	Should not be used in older adults because of very long half-life.
Estazolam (ProSom)	Rapid-Intermediate (15–60 minutes)	Intermediate	1–2 mg. (0.5–1 mg)	Generic $22.20	Because of long half-life, residual CNS effects are likely.
Temazepam (Restoril)	Intermediate-Slow (45–60 minutes)	Intermediate	15–30 mg (7.5 –15 mg)	Generic $19.80	
Triazolam (Halcion)	Rapid (15–30 minutes)	Short	0.125–0.25 mg (0.125 mg)	Generic $12.00 Halcion $46.80	Poor choice because of very short half-life and high incidence of CNS adverse reactions.
Non-benzodiazepines					
Eszopiclone (Lunesta)	Rapid (15–30 minutes)	Intermediate	2–3 mg. (1.2 mg)	$165.30	AE> 10%: headache, unpleasant taste
Zolpidem ER (Ambien CR)	Rapid (30 minutes)	Intermediate	12.5 mg (6.25 mg)	$152.40	AE> 10%: dizziness, headache, somnolence
Zolpidem (Ambien)	Rapid (30 minutes)	Short	10 mg (5 mg)	Generic $135.00 Ambien $160.80	AE>10% dizziness, headache, somnolence
Zaleplon (Sonata)	Rapid (15–30 minutes)	Ultra-short	10–20 mg (5 mg)	Generic $99.30 Sonata $46.80	AE nausea (7%), myalgias (7%)
Melatonin receptor agonist					
Ramelteon (Rozerem)	Rapid (15–30 minutes)	Short	8 mg (8 mg)	$121.20	AE headache (7%), Somnolence (5%), Dizziness (5%), Not a Class C-IV scheduled drug

Source: "Evidence-Based Recommendations for the Assessment and Management of Sleep Disorders in Older Persons," by H. G. Bloom, I. Ahmed, C. A. Alessi, S. Ancoli-Israel, D. J. Buysse, M. H. Kryger, et al., 2009, *Journal of the American Geriatrics Society, 57*, pp. 761–789.
Abbreviations: AE, adverse effects; CNS, central nervous system.
+ 30 doses at lowest recommended hypnotic dose.
[a] All are class C-IV scheduled drugs, and may also be associated with amnesia and complex sleep-related behaviors such as sleepwalking or sleep eating.

primary care settings where clients have complex health care needs. Morin et al., (2006) reported that there has been no complete dismantling of CBT-I to determine the relative efficacy of each component part or definitive investigations to develop and validate treatment algorithms. Some unanswered questions pertinent to the primary care setting are the following: (1) What are best practices to engage primary care clients in online CBT-I? (2) What subset of primary care clients would be best suited to online CBT-I? (3) What is the best CBT-I treatment package? (Vincent & Lewycky, 2009). An NIH State-of-the-Science conference on management of chronic insomnia in adults discussed the need for research in this area in depth. (See http://consensus.nih.gov/2005/2005InsomniaSOS026html.htm.)

Combined hypnotic and behavioral approaches may be helpful, and behavioral strategies can be

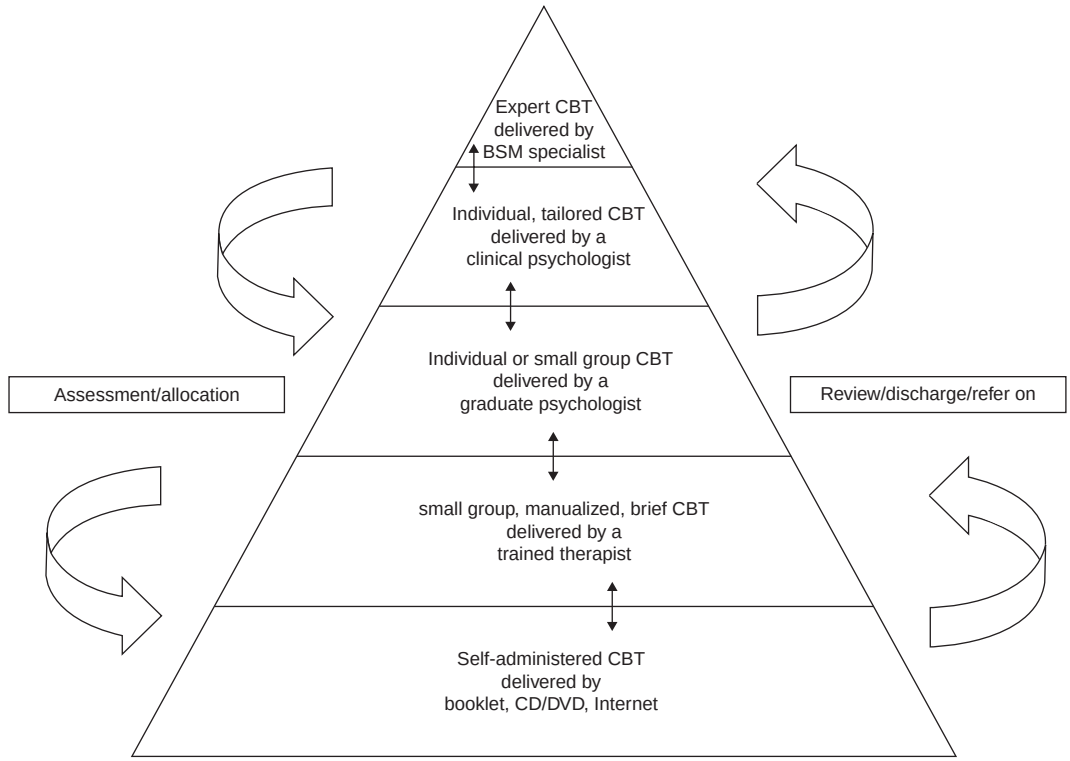

Figure 18.2 ■ An evidence-based stepped care model for CBT (c. 2009) illustrating how patients might be allocated to resources in relation to assessed need, to achieve optimal service provision. Arrows represent self-correcting referral movements. *Source:* "Stepped Care: A Health Technology Solution for Delivering Cognitive Behavioral Therapy as a First Line Insomnia Treatment," by C. A. Espie, 2009, *Sleep, 32,* pp. 1549–1558. Reprinted with permission.

used to promote tapering and discontinuation of hypnotic drugs. Investigators found that 84% of insomniacs who used hypnotics in the primary care setting responded well to non-pharmacologic therapy for insomnia and were able to successfully end their hypnotic use (Espie, Inglis, Tessier, & Harvey, 2001).

Despite the potential benefits of prescription hypnotics and behavioral approaches, adults and older adults often use over-the-counter (OTC) alcohol, antihistamines, and herbal remedies such as valerian or melatonin to manage their insomnia. (See Chapter 6, Insomnia.) Primary care providers must educate clients about the benefits and risks of these strategies. Alcohol is the most frequently used sleep aid in the general population (Kryger, Roth, & Dement, 2005). Because alcohol ingestion before bedtime results in shortened sleep latency, clients mistakenly believe that alcohol improves sleep. However, during the second half of the night, alcohol withdrawal leads to shallow, disrupted sleep (Kryger et al., 2005). Therefore, it is not an effective sleep-promoting agent. Antihistamines are approved by the FDA to be used as "sleep aids" but the risks, especially for older adults, include daytime sedation, impaired driving skills (Pagel, 2007), and cognitive impairment (Basu, Dodge, Stoehr, & Ganguli, 2003). Therefore, patients should be discouraged from using these substances. (See Chapter 6, Insomnia.)

Obstructive Sleep Apnea

Daytime sleepiness and a history of snoring are the most frequent symptoms that should alert the primary care provider to the possibility of obstructive sleep apnea. (See Chapter 7, Sleep-Related Breathing Disorders.) Although obesity is associated with sleep apnea in young

and middle-aged adults, this is not necessarily the case with older adults who may be more likely to report nocturia, sleep-related complaints, and excessive daytime sleepiness (Endeshaw, 2006). Because sleep apnea is a risk factor for hypertension and consequent cardiovascular heart disease (Peppard, Young, Palta, & Skatrud, 2000), glucose intolerance, insulin resistance, and subsequent diabetes (Punjabi et al., 2004; Botros et al., 2009), primary care providers should be particularly vigilant for possible sleep apnea in these clients. There is growing evidence that treatment of sleep apnea may prevent the development or exacerbation of these conditions.

When sleep apnea is suspected, the bed partner should be included during history taking to obtain descriptions of snoring, gasping, or choking during sleep (Bloom et al., 2009). The physical examination should focus on the upper airway to exclude anatomic obstruction (Malhotra et al., 2006; Lam, Ip, Tench, & Ryan, 2005; National Guideline Clearinghouse, 2010). (See Chapter 7, Sleep-Related Breathing Disorders.)

Questionnaires may be used to screen adults and older adults for risk for sleep apnea, but they are not sufficient to diagnosis the condition. The Berlin Questionnaire (Netzer, Stoohs, Netzer, Clark, & Strohl, 1999) is well suited to use in the primary care settings and has been validated in both adult (Sharma et al., 2006) and older adult populations (Chung et al., 2008) and in surgical and primary care clients (Chung et al., 2008; Mitler, Dawson, Henriksen, Sobers, & Bloom, 1988; Netzer et al., 1999). It is available online (see Table 18.1). The Berlin Questionnaire predicted an apnea hypopnea index > 5 (at least mild sleep apnea) with a sensitivity of 86% and specificity of 95%. A Spanish translation of the Berlin Questionnaire is available (Netzer et al., 2003).

The Epworth Sleepiness Scale (ESS), a measure of excessive daytime sleepiness during everyday life, can also be used to screen for this negative consequence of poor sleep. An ESS score greater than 10 is classified as clinically significant daytime sleepiness (Knutson, Rathouz, Yan, Liu, & Lauderdale, 2006). The ESS has been translated into many languages, including Spanish (Chiner, Arriero, Signes-Costa, Marco, & Fuentes, 1999), German (Bloch, Schoch, Zhang, & Russi, 1999), Japanese (Takegami et al., 2009), Norwegian (Beiske, Kjelsberg, Ruud, & Stavem, 2009), and Turkish (Izci et al., 2008). (See Chapter 5, Conducting a Sleep Assessment.) Information on use of the ESS is on the Web site of its author, Dr. Murray Johns.

If sleep apnea is suspected, the provider should refer the client to an accredited sleep center. (See Chapter 5, Conducting a Sleep Assessment and Chapter 7, Sleep-Related Breathing Disorders.) Although there is growing use of portable devices for unattended sleep testing in the home, a recent American Academy of Sleep Medicine task force recommends that portable testing for sleep apnea include, at a minimum, measures of blood oxygenation, respiratory effort, and airflow (see Chapter 7, Sleep-Related Breathing Disorders) and should be utilized only in conjunction with a clinical sleep evaluation conducted by a certified sleep specialist (Collop et al., 2007). Full polysomnography can also be performed in the home, but technical quality has been shown to be inferior to polysomnography conducted in an accredited sleep laboratory, and clients tend to prefer the laboratory approach (Gagnadoux, Pelletier-Fleury, Philippe, Rakotonanahary, & Fleury, 2002). Less is known about patient preference for more limited home monitoring devices for sleep apnea. Chapter 8 provides extensive detail on strategies for assessment and treatment of sleep apnea.

Because sleep apnea is a chronic illness, clients require long-term follow-up and a multidisciplinary approach that includes the referring primary care provider, sleep specialist, nursing personnel, respiratory therapist, sleep technologist, and durable medical equipment provider (Epstein et al., 2009; Institute for Clinical Systems Improvement, 2010). CPAP is the first line treatment for mild, moderate, and severe obstructive sleep apnea. (See Chapter 7, Sleep-Related Breathing Disorders.) After CPAP is initiated by the sleep specialist in the sleep laboratory, the primary care provider has an important role in addressing the challenging problems of adaptation and long-term adherence (Zozula & Rosen, 2001). Adherence to CPAP is a primary limitation to its effective

use. The primary care provider should elicit adherence and assess for problems, such as ill-fitting interfaces (e.g., masks or nasal pillows), nasal congestion, and/or dry skin. The durable medical equipment company who provides the CPAP equipment can also assist the patient with these issues (see Table 18.4). Because CPAP may improve chronic comorbid conditions such as hypertension and diabetes (Barbe et al., 2010; Harsch et al., 2004), the primary care provider should carefully monitor blood pressure and glycemic control and consider the potential need to adjust antihypertensive or diabetes medications accordingly. Clients should be instructed to report the reappearance of snoring or daytime sleepiness because they may signal malfunction of the CPAP machine, inadequate fit of the mask, inadequate CPAP pressure, or nonadherence (Bloom et al., 2009). An ill-fitting CPAP mask may be particularly likely if the clients have gained or lost weight. Follow-up in the sleep laboratory may be indicated for possible re-titration of CPAP.

Because of the significant impact of obstructive sleep apnea on daytime function, including cognition and excessive daytime sleepiness, it is critically important to evaluate clients with this condition (or suspected to be at risk for sleep apnea) for safety and the potential for injury to self or others. This is particularly true for those that operate motor vehicles (especially commercial drivers) or machinery, as sleepiness may contribute to motor vehicle crashes or other causes of injury due to sleepiness. Poor cognitive performance resulting from excessive daytime sleepiness may also result in errors or lapses in memory or attention at home, school, or in the workplace. Often patients are not aware that they are sleepy and may not associate it with a treatable sleep disorder. Nurses in primary care settings can play an important role in educating clients and their families about this safety risk.

Oral appliances or surgery may be indicated depending on the clients' anatomy, dentition, risk factors, and preferences (Epstein et al., 2009). (See Chapter 7, Sleep-Related Breathing Disorders.) Several surgical approaches are available, but some are more effective in reducing snoring than eliminating or sleep apnea itself.

Table 18.4 ■ *Management Tips to Improve Adherence with CPAP Therapy*

Snoring While on CPAP:
- Adjust mask/interface if leaking.
- Ask bed partner whether client is opening mouth (apply chin strap).
- If there is no leaking around mask/interface and snoring continues, it is recommended that the clients have a follow-up visit or phone call to the sleep specialist to reassess pressure level.
- Ask whether client had alcohol before bed.

Opening Mouth:
- Apply chin strap.
- Change interface to full-face mask.

Nasal Congestion or Runny Nose:
- Add or adjust humidifier (cool or heated); consider integrated heated tubing system.
- Use nasal saline spray during the day and at bedtime.
- Use nasal corticosteroid/anticholinergic spray.
- Use antihistamine (oral or nasal spray).
- Change or clean machine filter.

Mask/Interface Leak:
- Adjust mask/interface straps, forehead pads, or nasal cushion.
- Refit mask/interface to different size.
- Change to a different interface or full-face mask.

"Air Hunger"
- Check for mask/interface leak.
- Check for mouth opening (apply chin strap).

"Too Much Air," Can't Exhale:
- Use another type of PAP (flexible, Bi-PAP).
- Begin use of ramp-delay on machine.
- Increase ramp/delay time.
- Change interface.
- Add humidifier (cool or heated).
- Use nasal saline spray during the day and at bedtime.
- Apply chin strap if opening mouth.
- ENT consult for deviated septum or surgical options.
- Dental consult for oral appliance options.

Nosebleeds:
- Add humidifier (cool or heated).
- Use nasal saline spray during the day and at bedtime.
- Use water-soluble nasal saline gel in nares to moisturize.

Claustrophobia:
- Try relaxation skills or desensitizing techniques.
- Instruct to wear CPAP while awake and reading or watching TV to get used to equipment.
- Consider referral for treatment of claustrophobia.

(Continued)

Table 18.4 ■ *Continued*

Removing Positive Airway Pressure without Knowledge:

- Add chin strap to help secure interface to head.
- Safety pin headgear to nightclothes (this is used as a reminder during the night when awakening to keep mask/interface on head).
- Activate disconnect alarm (if available).

Bed Partner Complaints of Cold and Blowing Air:

- Different interface.
- Redirect exhalation port if mask allows.
- Place a barrier (e.g., pillow/blanket) between bed partners.
- Place CPAP on floor.

Complaints of Noise:

- Place CPAP on floor.
- Wear earplugs.
- White noise machine.

Sleepiness Despite Treatment:

- Reassess adequate sleep.
- Reassess returned snoring on CPAP.
- Reassess sleep hygiene before bed.
- Reassess if using CPAP all night.
- Leaking or poorly fitting mask/interface
- Ask whether mask is more than 6 months old.
- Assess environmental noises (e.g., planes, buses, and neighbors).
- Assess bed partner or pet disturbances in bedroom.
- Ask whether napping during the day and for how long.
- Assess bed partner complaints of leg movements.
- Assess bed partner complaints of bruxism (teeth grinding).
- Consider retitration of CPAP.
- Assess for use of alcohol less than 2 hours before bedtime.
- Ask whether opening mouth at night while bleeping with CPAP.

Dental Device Complaints

- Facial and/or tooth pain.
- Appliance not retentive.
- Dental occlusal changes.
- Above complaints should prompt a return to dental practitioner for evaluation and treatment.

Travel and Hospital Visits:

- Remind clients to bring CPAP equipment when traveling or admitted to the hospital.

Source: Health Care Guideline: Diagnosis and Treatment of Obstructive Sleep Apnea by Institute for Clinical Systems Improvement, 2010. Google Scholar. Retrieved from http://www.icsi.org

Because obesity is a risk factor for sleep apnea, weight loss is an important, but underutilized approach to treating sleep apnea. Weight loss may be particularly beneficial in those with mild to moderate sleep apnea. A behavioral approach that includes a low calorie diet and active lifestyle is a low-cost, feasible curative treatment for clients with mild sleep apnea (Tuomilehto et al., 2009) that has other health-related benefits. Clients who lose weight may require a lower level or CPAP or be able to discontinue it, but this should be done only after a titration in a sleep laboratory setting. Patients who gain weight may require higher CPAP pressure.

Movement Disorders

Restless legs syndrome (RLS) presents as a compelling urge to move one's legs due to unpleasant sensations, most often occurs at rest, is relieved with movement, is generally more pronounced during the first half of the night, and rarely involves the arms. PLMD is sometimes referred to as nocturnal myoclonus or periodic leg movements, consists of repeated rhythmic extensions of the big toe and dorsiflexion of the ankle with occasional flexions of the knee and hip, and the client may not be aware of these movements. Assessment for RLS can easily be incorporated into the primary care sleep assessment with the key questions listed in Table 18.5. RLS is more likely to be associated with family history in adults who are under the age of 45 than in clients with a late onset who are more likely to have low serum iron (Allen, 2000). Other risk factors for RLS include fibromyalgia, rheumatoid arthritis, terminal illness, neuropathies, radiculopathies, and diabetes (Hornyak & Trenkwalder, 2004). (See Chapter 8, Sleep-Related Movement Disorders and Parasomnias.) Case Study 18.1 presents an example of a patient presenting in the primary care setting with a sleep disorder.

IMPLEMENTING SLEEP INTERVENTIONS IN ADULT PRIMARY CARE SETTINGS

Evidence-Based Guidelines

There are numerous evidence-based guidelines available to guide nurses and other primary care providers on delivering efficacious sleep

Table 18.5 ■ *Assessment of Restless Legs Syndrome: Questions for the Client*

- Is there an urge to move the legs, and do uncomfortable or unpleasant sensations in the legs accompany or cause this urge?
- Do the unpleasant sensations or the urge to move begin or worsen during periods of rest or inactivity, such as sitting or lying down?
- Does movement such as walking or stretching, partially or totally relieve the unpleasant sensations or the urge to move for at least as long as the activity continues?
- Do the unpleasant sensations or urge to move get worse or only occur in the evening or night?

Source: "Evidence-Based Recommendations for the Assessment and Management of Sleep Disorders in Older Persons," by H. G. Bloom, I. Ahmed, C. A. Alessi, S. Ancoli-Israel, D. J. Buysse, M. H. Kryger, et al., 2009, *Journal of the American Geriatrics Society*, 57, pp. 761–789.

treatments. Examples are provided in Table 18.6. Although some treatments, such as cognitive–behavioral therapy for insomnia, have well-documented efficacy in well-controlled clinical trials, effectiveness studies (studies of the effects of these treatments when implemented to heterogeneous groups of patients in "real world" clinical settings such as primary care) are only just beginning. Even less is known about the cost-effectiveness of treating sleep in the primary care setting, although the costs of the negative consequences of sleep disorders are high and access to sleep specialists is limited for many clients, especially those living in rural settings.

Table 18.6 ■ *Selected Reviews and Practice Guidelines for Sleep Assessment and Interventions*

Sleep Disorders in Older Adults

Bloom, H. G., Ahmed, I., Alessi, C., Ancoli-Israel, S., Buysse, D. J., Kryger, M. H., et al. (2009). Evidence-based recommendations for the assessment and management of sleep disorders in older persons. *Journal of the American Geriatrics Society, 57,* 761–789.

McCurry, S. M., Logsdon, R. G., Teri, L., & Vitiello, M. V. (2007). Evidence-based psychological treatments for insomnia in older adults. *Psychology and Aging, 22,* 18–27.

Montgomery, P., & Dennis, J. (2003). Cognitive behavioural interventions for sleep problems in adults aged 60+. *Cochrane Database of Systemmatic Reviews,* CD003161.

Montgomery, P., & Dennis, J. (2004). A systematic review of non-pharmacological therapies for sleep problems in later life. *Sleep Medicine Reviews, 8,* 47–62.

Insomnia

Chesson, A. L., Anderson, W. M., Litner, M., Davila, D., Harts, K., Johnson S., et al. (1999). Practice parameters for the non-pharmacologic treatment of chronic insomnia. An American Academy of Sleep Medicine report. Standards of Practice Committee of the American Academy of Sleep Medicine. *Sleep, 22,* 1128–1133.

Littner, M., Hirshkowitz, M., Kramer, M., Kapen, S., Anderson, W. M., Bailey, D., et al. (2003). Practice parameters for using polysomnography to evaluate insomnia: An update. *Sleep, 26,* 754–760.

Morin, C. M., Bootzin, R. R., Buysse, D. J., Edinger, J. D., Espie, C. A., & Lichstein, K. L. (2006). Psychological and behavioral treatment of insomnia: Update of the recent evidence (1998–2004). *Sleep, 29,* 1398–1414.

Schutte-Rodin, S., Broch, L., Buysse, D., Dorsey, C., & Sateia, M. (2008). Clinical guideline for the evaluation and management of chronic insomnia in adults. *Journal of Clinical Sleep Medicine, 4,* 487–504.

Restless Legs Syndrome and Periodic Limb Movement Disorder

Littner, M. R., Kushida, C., Anderson, W. M., Bailey, D., Berry, R. B., Hirshkowitz, M., et al. (2004). Practice parameters for the dopaminergic treatment of restless legs syndrome and periodic limb movement disorder. *Sleep, 27,* 557–559.

Chesson, A. L., Jr., Wise, M., Davila, D., Johnson, S., Littner, M., Anderson, W. M., et al. (1999). Practice parameters for the treatment of restless legs syndrome and periodic limb movement disorder. An American Academy of Sleep Medicine Report. Standards of Practice Committee of the American Academy of Sleep Medicine. *Sleep, 22,* 961–968.

Obstructive Sleep Apnea

Epstein, L. J., Kristo, D., Strollo, P. J., Jr., Friedman, N., Malhotra, A., Patil, S. P., et al. (2009). Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. *Journal of Clinical Sleep Medicine, 5,* 263–276.

Barriers to Translation of Sleep Promotion Strategies Into Primary Care

Even when sleep treatments have documented efficacy and effectiveness, the translation of sleep promotion strategies into primary care practice or universal client access to these needed services is not guaranteed. Although the strategies described earlier in this chapter can be implemented by the individual nurse or advanced practice nurse, incorporating these clinical practices may be challenging. Potential barriers include the lack of previously acquired knowledge and skills about sleep and sleep disorders among health care professionals, including nurses; lack of awareness about the importance of sleep among providers and clients; limited time for patient encounters during which many health care concerns must be addressed; limited financial resources (e.g., insurance reimbursement) for specialized sleep treatment; the complex and multidimensional nature of sleep assessment and some treatments (e.g., behavioral treatment for insomnia), and the busy pace of most primary care settings.

Education of Primary Care Providers

Educational efforts to increasing knowledge, awareness, and proficiency regarding sleep and sleep disorders treatment continue to be necessary, as it is well known that educational programs for nurses and other health care providers have very limited content on sleep. Academic detailing is an innovative method of educational outreach frequently provided by pharmaceutical companies. Academic detailing provides an accurate up-to-date synthesis of drug information in a balanced format (Zwar, Wolk, Gordon, & Sanson-Fisher, 2000). The American Academy of Sleep Medicine offers additional approaches to educational outreach through their Web site (see Table 18.2) http://www.aasmnet.org/ProfDev.aspx. Continuing education opportunities are also available face-to-face and online through several academic institutions. (See Chapter 24, Future Directions in Sleep Promotion: Nursing Practice, Research, and Education.) Chapter 23 provides further information on opportunities for advanced practice nurses to obtain specialized sleep training.

Protocol Implementation

While educational efforts are necessary first steps in changing primary care practice related to sleep, they are not sufficient to assure more widespread reach, adoption, implementation, and long-term maintenance at the organizational level. There is a need for development and implementation of organization-wide protocols and systems of care that promote sleep promotion practices. Strategies to address barriers to incorporation of sleep evaluation and sleep disorders treatment at the system/organizational/practice level are likely to be most effective when implemented at the organizational/practice level and when the proposed innovations are perceived to be advantageous and consistent with existing values, can be piloted on a small scale, are less complex, and have consequences that can be easily measured (e.g., decreased cost, improved efficiency, improved patient outcomes) (Rogers, 1995). The advanced practice nurse must utilize the roles of clinical expert, opinion leader, systems analyst, and politician to initiate and maintain practice changes that will engage of senior administrators and fellow health care professionals.

Manual or electronic systematic reminders to prompt health care providers to perform clinical actions (i.e., routine sleep assessment) are effective in changing physician and nurse behavior regarding other types of health care practices (Grimshaw et al., 2001) and may be helpful in improving sleep screening and treatment practices. Research is needed into the feasibility and effectiveness of this strategy. Incorporating cues for sleep assessment into standardized electronic or paper-based templates used for obtaining health histories may also be useful.

Evaluation of Sleep Promotion Practices

Evaluation of outcomes associated with changes in sleep promotion and sleep disorders assessment and treatment is critical to assuring successful adoption, implementation, and maintenance of

sleep promotion strategies, and improving outcomes. At the client level, outcomes include improvements in self-reports of sleep, as well as daytime functioning, mood, quality of life, patient satisfaction, and reductions in morbidity (e.g., hypertension, obesity, diabetes). Patient satisfaction and costs of care are significant considerations. Although addition of some sleep-related services may at first increase the costs of care due to additional time associated with patient care, costs to the health care system may ultimately decrease due to improved patient outcomes.

The performance of primary health care providers in delivering evidence-based sleep promotion strategies should be evaluated on a routine basis. In fact, sleep health will be an important indicator on the U.S. "Healthy People 2020" goals. The use of combinations of audit and feedback, reminders, and local consensus processes may be effective (Grimshaw et al., 2001) in improving provider performance. Case study 18.2 presents an example of such a multifaceted intervention developed with a quality improvement process led by an advanced practice nurse.

SUMMARY

There is a compelling need for widespread access to sleep assessment and treatment among the large population of primary care clients who have sleep disorders—many of which are currently undetected. Primary care providers, especially nurses, are in an ideal position to assess, implement, and evaluate sleep promotion and sleep disorders treatment in primary care clients. The reach, adoption, implementation, and long-term maintenance of sleep promotion and sleep disorders treatment is most likely to be successful if implemented at the practice/organizational level. Nurses, especially advanced practice nurses play a pivotal role in implementing and evaluating policies and procedures to assure the translation and uptake of these important services.

CASE STUDY 18.1 The Patient with Obstructive Sleep Apnea in the Primary Care Setting

Identifying information. WC is a 65-year-old Caucasian male who presents to his primary health care provider (PHP) for a routine cholesterol and blood pressure check. WC has been working to manage his modifiable cardiovascular risk factors since the age of 35 with diet and exercise. WC is careful with his food choices, exercises vigorously at least three times a week, and has managed to decrease his BMI from the obese category (30.5) to slightly overweight (26.4). His wife LC comes with WC to this appointment because she is concerned about WC's sleep.

Presenting complaint and sleep information. LC, reports that WC snores, and sometimes sounds like he stops breathing during the night. He wakes repeatedly sometimes gasping for air, and he frequently has to get up to urinate. She says that, in the past, when she asked him to turn onto his side so that the snoring would subside, he slept through the night, but now, he snores while lying on his back and his side. The PHP turns to WC and asks whether he thinks he sleeps well. WC and LC laugh and say, except during the night, he is a great sleeper, he can fall asleep anywhere, anytime. The PHP asks WC how he feels in the morning when he wakes up. WC says he feels tired and often wakes with a headache. When WC completes the Epworth Sleepiness Scale, he has a score of "12." The PHP then performs a physical examination and takes WC's medical history, including sleep and daily functioning habits for him and his family. The PHP also checks WC's tonsils, uvula, and soft palate for enlarged tissues.

Referral. Following the examination, the PHP suspects sleep apnea and refers WC to an accredited sleep specialist to determine the cause of his symptoms. The sleep specialist diagnoses obstructive sleep apnea based on a split night sleep study. The sleep specialist prescribes the most common treatment for sleep apnea, continuous positive airway pressure (CPAP). WC has an insurance plan that covers his CPAP, and WC is assigned a case manager to help him appropriately handle his disorder.

(*Continued*)

CASE STUDY 18.1 *Continued*

Follow-Up. In WC's case, although he is not significantly overweight, his advancing age places him at greater risk for upper airway obstruction that restricts the throat from being open enough to allow adequate oxygenation. The PHP explains that the sleep apnea is, most likely, the root cause of WC's early morning fatigue and daytime sleepiness. Also, the PHP explains that there are some changes in his daily activities that can be made to help his disorder, for example, avoid alcohol, smoking, and medications that may make him drowsy. The PHP ensures that the durable medical equipment company contracted for the CPAP machine provides WC and LC with education regarding machine set-up.

A week after beginning the treatment, WC calls his PHP. He says he cannot use the CPAP machine because he has a stuffy nose and headaches. The PHP tells WC that these are likely side effects of the CPAP therapy. She also assesses for any other side effects such as facial skin irritation, bloating, or sore eyes. She advises WC to contact his sleep specialist, who recommends a nasal spray for his stuffy nose and a decongestant for his headaches. In a follow-up phone call, 1 month later, LC tells the PHP that WC is now successfully using the CPAP machine every night. She says, "WC is a new man with more energy." By following up with WC, the PHP ensures that he is compliant with his CPAP therapy. These actions collectively promote quality, cost-effective interventions, and outcomes.

You administer the Epworth Sleepiness Scale to an older adult who complains of disturbed nighttime sleep. The clients scores "14" and indicates he has occasionally fallen asleep when driving. When you ask how he got to today's appointment. He says he drove in by himself. What is your first action?

1. What groups are most at risk for obstructive sleep apnea?
2. What age-related disorders affect disrupted sleep?
3. How can disrupted sleep be identified in the primary care setting?
4. Is sleep hygiene a successful evidence based intervention?

CASE STUDY 18.2 Quality Improvement Project

You are working as an advanced practice nurse in primary care setting and have been charged with leading quality improvement efforts in your clinic. Your clinic uses the FOCUS-PDCA quality improvement model.

F-Find the opportunity. During the first meeting of the quality improvement team, members identified improved care of patients with obstructive sleep apnea as an important issue and the project that had the best potential to impact quality client care. The team identified goals and outcomes based on the published guideline from National Guideline Clearinghouse at www .guideline.gov.

The long-term goal of the project was to ensure that 100% of clients with sleep apnea were identified and received:

- Referral to a sleep specialist.
- Appropriate follow-up.
  - Obtain records from referral physician and assess patient's adherence to recommendations of management.
  - Patient education.
    - What to do if symptoms worsen?
    - Follow-up appointments.
  - Evaluate for weight loss program if obesity is a contributing factor.

Outcomes identified to determine success were

- improved rates of screening and identification patients with sleep apnea.

- improved diagnosis and referral for management and treatment.
- improved quality of life for patients with sleep apnea.
  - return of normal sleep patterns.
  - decreased daytime sleepiness or fatigue.
  - prevention of comorbidities associated with untreated sleep apnea: (hypertension, cardiovascular disease, congestive heart failure, stroke, heart attack).

The team determined that evidence must be present in the medical record to demonstrate that the client had been assessed for sleep disturbances and, if indicated, appropriate referrals and follow-up were implemented.

O-Organize the team. The team discussed who the key players were to ensure that these changes could be implemented and invited them to participate in the quality improvement effort.

C-Clarify Current Knowledge of the Practice Issue. Baseline data analysis in all areas, in particular, documentation of sleep assessment was very low, with only 10% of clients' records having documentation indicating that their sleep was assessed. The team decided to focus their first improvement efforts on improved assessment.

U-Uncover the sources of variation. Baseline data indicated that the clinic was far from meeting the target goals, the team identified factors that influenced variability.

- Decreased clinician knowledge
- Lack of system reminders
- Outdated forms that did not reflect current evidence-based care regarding sleep assessment

Identification of these factors provided the committee direction for prioritizing and implementing strategies to improve outcomes.

S-Select the strategies for improvement. The team determined that the first step was to develop and implement education strategies for all practitioners (nurses and physicians), clarify the strongest evidence regarding screening for obstructive sleep apnea in the primary care setting and deliver this information to all concerned providers. In addition, new charting forms were developed and approved so that they could be introduced during the educational forums. Team members suggested that pocket cards that include the outcome indicators could be distributed during the educational forums. The team identified all education forums available where this information could be disseminated, as well as new educational forums that were needed. These forums included monthly staff meetings, updating content in orientation, annual skill competencies, Nursing Practice Grand Rounds, Medicine Grand Rounds, electronic reminders, and unit posters.

P-Plan. The team developed a curriculum (teaching plan) that identified all learning objectives, educational methods to be used, forums, and an evaluation tool. They developed a timeline for provider education.

D-Do. All in-services were completed.

C-Check. Pretest/post-test evaluation tools were summarized. The team concluded that the educational effort was a success and gains had been made.

A-Act. The new methods of documentation were implemented and repeated audits were planned in 3 months to determine the success of the project.

1. Is prevention of comorbidities a feasible outcome?
2. If you were on the team, could you make prevention of comorbidities a measurable outcome?
3. Who would be essential members of the QI team in your practice setting?
4. What educational forums can you identify in your practice setting to communicate information about best practices in screening for obstructive sleep apnea?
5. Do you think computerized reminders would be helpful to remind clinicians to screen clients for obstructive sleep apnea?

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Sleep in Psychiatric-Mental Health Settings

19

Geoffry Phillips McEnany

Approximately 26.2% of Americans over the age of 18 live with some form of psychiatric illness in any given year (Department of Health and Human Services, 2000). One in 10 children and adolescents suffer from mental illness severe enough to cause some level of impairment (Source: http://www.surgeongeneral.gov/topics/cmh/cmhreport.pdf). These impressive statistics have serious implications for the sleep of people diagnosed with psychiatric illnesses across the lifespan, given the bidirectional relationship between sleep and psychiatric disease. Sleep pattern disturbance is ubiquitous across psychiatric disorders (see Chapter 12, Sleep and Psychiatric Disorders) and the sheer number of people diagnosed with these illnesses has weighty implications for nursing practice across all specialties and at all levels of practice. Appropriate identification of and intervention in sleep disturbance across the various psychiatric diagnostic categories has broad implications for public health, patterns of disease management, containment of health care related costs, and quality of life of the people who live with these illnesses.

In psychiatric nursing practice, recognition of sleep dysregulation associated with the various mental illnesses has implications that transect all levels of the systems involved in the general health of the individual receiving treatment. At the interpersonal and clinically focused level, psychiatric and mental health nurses provide a broad range of services to patients in inpatient hospitals, outpatient practices, and partial hospitalizations. Nursing practice across all settings includes assessment, disease management, risk reduction, and relapse prevention.

At the health care organizational level, there are a number of salient issues related to sleep that need to be addressed to assure positive clinical outcomes in psychiatric care. Probably, foremost is the culture shift that is inherent with heightening the level of awareness of sleep and its relationship to psychiatric disease identification and management. The NIH State-of-the-Science Conference Statement on manifestations and management of chronic insomnia in adults (2005) identified sleep-related clinical education of health care professionals as a main priority. The need for increased clinical attention to sleep spans the scope of the organizational system. From an organizational perspective, inclusion of clinical sleep education as a fiscal priority in strategic planning will serve to improve the quality of clinical services with enhanced and updated perspectives on psychiatric illness management. Monitors of quality management to scrutinize the impact of programmatic interventions aimed at educating clinicians about assessment and management of sleep disorders will provide organizations with measureable benchmarks to evaluate the quality of their outcomes. The outcomes of educational efforts are likely to be reflected in improved measures of clinical outcomes and associated cost containment.

The discrepancy between the current state of knowledge in sleep science and standard psychiatric practice provides a dangerous window for clinical liability. The evidence between sleep dysregulation and risk for disease onset has been clearly established. Inattention to these findings due to lack of knowledge may lead to negligent care. Clearly, this is *no time to slumber* for psychiatric and mental health clinicians across the disciplines. For nursing, the need for significant retooling of practice to include state-of-the-science clinical knowledge on sleep seems imperative (Phillips McEnany, 2006). Strategic moves within health care organizations related to sleep education to transform

and update practice only serve to improve the safety and quality of clinical services provided to those with psychiatric illnesses.

Clinical practice is significantly influenced by policy and economics. Insomnia and sleep dysregulation related to medical and psychiatric illnesses are pervasive and costly (Daley, Morin, LeBlanc, Grégoire, & Savard, 2009), and the pharmacoeconomic burden related to the treatment of these sleep disturbances is high. Recent estimates of the direct and indirect costs of insomnia have been estimated between $92.5 billion and $107.5 billion (Reeder, Franklin, & Bramley, 2007). The current juxtaposition of emerging practice guidelines related to comprehensive treatment of sleep dysregulation in psychiatric and medical diseases (Morganthaler et al., 2006) poses a healthy challenge to the current reality of clinical practice with escalating costs of insomnia treatment in psychiatric care, often with increasing pharmacological utilization (Asche, Joish, Camacho, & Drake, 2010). In many circumstances, the guidelines for insomnia and other sleep disorders are not evident in psychiatric practice. CBT-I (cognitive behavioral therapy for insomnia) and other evidence-based approaches to sleep disorders are likely to demonstrate significant cost reductions in care (Edinger & Carney, 2008). These data are in keeping with prescription medication data from the third National Health and Nutrition Examination Survey, which shows significant increases in psychotropic use among Americans in recent years, particularly with antidepressants, some of which are commonly prescribed as off label hypnotics (Paulose-Ram, Safran, Jonas, Gu, & Orwig, 2007). Many hypnotics approved by the Food and Drug Administration are benzodiazepine receptor agonists (see Chapter 12, Sleep and Psychiatric Disorders), but interestingly, Medicare Part D does not cover benzodiazepines. Consequently, patients covered under this plan must resort to using an off-label hypnotic medications or pay out-of-pocket for benzodiazepines. Cost effectiveness analysis drives the formulary decision making process (Wang, Salmon, & Walton, 2004), and nurses are becoming increasingly aware of these economic relationships as they participate influentially on pharmacy and therapeutics committees where decisions are made based on pharmacoeconomic data. At the level of clinical practice, nurses who prescribe face an escalation of the need for prior authorizations by insurers for select medications routinely prescribed for insomnia, adding to the time demands associated with pharmacotherapies for insomnia treatment. This imposed impediment frequently opposes evidence, which shows that the hypnotics that often require prior authorization are cost-effective measures (Snedecor et al., 2009).

The trends in policy and economics briefly discussed here have direct relevance to psychiatric nursing practice and positions nurses in an odd set of circumstances. Evidence-based practice guidelines are required to assure quality practice outcomes, but policy at the level of the insurers creates some barriers. In addition to medication issues related here, there is a trend toward denial of payment for CBT-I. Therefore, cost-effectiveness evaluations of CBT-I are needed, although the clinical effectiveness of this therapy is well established. Empirical support of the cost savings associated with a comprehensive approach to the treatment of sleep disorders will positively affect the patterns of disease management, recidivism, and cost containment, elements that are critical to the survival of a sustainable mental health care system.

ROLE OF THE NURSE IN PSYCHIATRIC TREATMENT SETTINGS

Inpatient Treatment Settings

The role of the nurse in psychiatric inpatient, crisis management, and detoxification units is very different from the role of the nurse in outpatient or community-based services. Patients in inpatient settings are acutely ill, often have life-threatening symptoms, and have the potential for self-harm or severe impairment in self-care capacity. In the inpatient settings, assessments are urgently conducted and intervention reflects the acuity of the presenting

illness. Prompt symptom containment is vital to stabilization of the acute condition. Abbreviated hospital stays are common in the current health care economic climate. This trend results in greater pressure for efficient and measureable symptom control in order to transition care to less expensive outpatient-based services. Nurses work under these pressurized conditions and prioritize the care to meet the most critical needs.

Although the clinical experience of the nurse almost universally can testify to the presence of severe sleep disturbances among patients on inpatient units, the literature to corroborate these clinical observations is sparse. Dogan and Ertekin (2004) examined sleep patterns in patients admitted for treatment of a psychiatric disorder compared with those hospitalized with medical conditions. Their results showed that those in the psychiatric cohort had significantly more disturbed sleep. Women had significantly poorer sleep than men. Collier, Skitt, and Cutts (2003) explored the subjective experience of patients in an inpatient psychiatric setting. While their study exposed a number of themes, one important finding was that patients often do not discuss their sleep-related complaints with clinicians. However, even when sleep pattern disturbances are discussed, they are often dealt with inconsistently by nursing staff. Green, Hicks, and Wilson (2008) reported that patients with chronic insomnia believe that clinicians do not understand their insomnia, and patients often want better information about insomnia and its management than the clinicians provide.

Medications are a mainstay of inpatient treatment and these interventions are used in the service of acute stabilization of symptoms, including sleep disturbance. Depending on the presenting condition, standard treatments for insomnia may be used. Alternatively, medications may be selected according to their capacity to target the greatest number of symptoms in the most efficient way. For example, for a person admitted for treatment of a psychotic condition, sedating antipsychotics are used in lieu of an antipsychotic and a hypnotic. Rapid titration of medications is common in inpatient settings (Feifel, 2008), and there is concurrent risk for more acute side effects of medications. However, given the availability of nurses around the clock, side effects are identified and early intervention mitigates their intensity. Effective management of side effects is an important factor to consider because the presence of side effects is a predictor of poor adherence (Goethe, Wooley, Cardoni, Woznicki, & Piez, 2007).

In addition to aggressive pharmacotherapy, behavioral interventions aim to contain acute symptoms. A mainstay of nursing care is to help the patient regain self-care capacity in those areas impacted by the illness. Nursing care focuses on the areas of self-care described by Dorothea Orem and Taylor (1986) in her self-care theory. According to the theory, nursing care focuses on the areas of self-care deficit that are caused by the illness. These areas include nutrition, elimination, hygiene, rest/activity, and solitude/socialization. Nursing intervention specific to sleep and wakefulness focuses on reregulating patterns of activity and sleep through the use of structured activity. Sleep hygiene becomes a critical dimension of this approach. As the acuity of the patient's symptoms abate, teaching becomes a critical focus in the areas of self-care. Sleep is an especially important focus, given its relationship to symptom control.

Ideally, evidence-based protocols for regulation of sleep and wakefulness would be utilized to guide sleep interventions from admission to discharge. Given the fact that many of the psychotropic medications carry a significant risk for weight gain and risk for obstructive sleep apnea syndrome (see Chapter 7, Sleep-Related Breathing Disorders) and restless legs/periodic limb movements (see Chapter 8, Sleep-Related Movement Disorders and Parasomnias), assessments aimed at evaluating the presence of related symptoms is important and can become the foundation for referral for further work up. Nurses on the night shift are in a prime position to document patterns of snoring and movements as patients in inpatient units are routinely checked at regular intervals throughout the night.

Duration of the hospital stay and the readiness of the patient to learn become determining factors in the approaches used in discharge

teaching. Psychosis, severe depression, substance withdrawals, mania, severe forms of obsessive–compulsive disorder or panic may impair cognitive capacities—all of which will impact the approach used to teaching. Many patients leave the hospital with significant improvement but less than complete symptom control. When available, family members are included in the teaching, especially in light of conditions that impair learning readiness in the patient. Patients with severe and persistent forms of the illness may be discharged to other facilities offering less acute levels of care. In these circumstances, communication of the sleep-related plan of care to the receiving facility becomes critical.

Structured plans of intervention could be communicated with the outpatient clinician who will assume the care of the patient after discharge. However, the extent to which this can happen successfully depends in part on the knowledge base of the nurses/clinicians involved in the trajectory of the patient's care. Table 19.1 offers an example of an inpatient nursing care plan to address sleep-related disturbances.

Outpatient Treatment Settings

Treatments offered in outpatient settings include individual, family, and group therapies. Psychopharmacologic assessment and management are provided at the advanced practice levels of nursing care delivery. Treatment in the outpatient setting may be episodic in the event of a single episode of an illness such as major depression or ongoing in light of chronic psychiatric illnesses such as schizophrenia.

Table 19.1 ■ *Inpatient Care Plan for Sleep Disturbance*

On admission:

- Assess the presenting patterns of sleep/wakefulness including insomnia, hypersomnia, and excessive daytime sleepiness.
- Assess for the presence of possible comorbid sleep disorders, especially risk for obstructive sleep apnea and restless legs syndrome/periodic limb movement disorder.
- Evaluate the side effects of medications that contribute to patterns of sleep disturbance and daytime sleepiness.
- Utilize instrumentation aimed at objective evaluations of the presenting symptoms on admission will allow for tracking of the symptoms on admission, throughout the hospitalization and at discharge. (See Chapter 5, Sleep Assessment.) When possible, engage the patient in the use of sleep-wake logs.

During the hospitalization:

- Based on assessment data, implement interventions aimed at correction of self-care deficits in the area of sleep and wakefulness.
- Communicate assessment data to collaborating nurse practitioners or physicians for further work up of possible sleep disorders is crucial.
- Utilize sleep-wake logs to correct disturbances in sleep hygiene and use the data from the logs to begin teaching about negative influences to sleep and wakefulness. Addressing the impact of medications on sleep-wake patterns is crucial.
- Address the connection between sleep regulation and symptom course, as well as the impact of ongoing sleep dysregulation on other indices of health.

At discharge from the hospital:

- Provide synopsis of sleep-related plan to be included in the discharge summary.
- Develop recommendations for follow-up care including the use of cognitive behavioral therapy for insomnia, if appropriate.
- Provide written materials on sleep hygiene as well as copies of sleep-wake logs for the patient to use post discharge as a means of symptom monitoring.
- Discuss the importance of pattern recognition related to sleep disturbance as an index of symptom control during acute treatment and an indicator of relapse during continuation treatment is essential.
- Teach about the impact of medications on sleep and wakefulness.
- Reduce risks related to substance use in the service of maintaining wakefulness (e.g., use of caffeine, energy drinks) or sleep induction and maintenance (e.g., alcohol, marijuana, other herbals such as kava). Use of these substances provide a benchmark of difficulty and can be used as indices of the need for more appropriate intervention to address the underlying problem.

Sarsour, Morin, Foley, Kalsekar, and Walsh (2010) report that the odds of severe insomnia associated with a psychiatric diagnosis is high. Consequently, patients with psychiatric illnesses often come to outpatient care with insomnia that is severe and chronic. (See Chapter 12, Sleep and Psychiatric Disorders.) Insomnia associated with psychiatric illnesses is complicated by a number of factors including lifestyle irregularity imposed by the symptoms of the disease, fearful anticipation that sleep will not normalize in recovery as well as partial remission of the illness itself. de Niet, Tiemens, Lendemeijer, and Hutschemaekers (2008) point out an important factor in dealing with insomnia in psychiatric disease. Often, patients under-report the insomnia symptoms when compared with objective measures of sleep. The dissonance may be perpetuated by the fact that when there is some (yet incomplete) relief of insomnia symptoms, patients are often so grateful to see an improvement in their sleep patterns, that they accept the partial remission as adequate. In the absence of thorough assessment of this phenomenon, the partial control of symptoms may be missed and the lack of clinical assessment can become a perpetuating factor in the insomnia. Difficulty resuming sleep in light of a psychiatric illness is not uncommon but this difficulty provides a salient index of incomplete recovery (Ohayon, 2009).

Outpatient settings have advantages over their inpatient counterparts inasmuch as they afford treatment over the course of a year or more. Nurses working in these settings are ideally situated to fully assess patterns of sleep and wakefulness and devise a treatment plan to address the sleep-related needs over time. Psychiatric/Mental Health Clinical Nurse Specialists and Nurse Practitioners provide a wide range of services in outpatient settings that include individual, group, and family therapies. In most states, these nurses have prescriptive authority, which allows them the opportunity to provide comprehensive service to patients. Psychiatric and mental health advanced practice nurses are routinely trained in cognitive behavioral therapy, given the evidence base in support of its effectiveness across many of the psychiatric disorders (Olatunji & Hollon, 2010). Because of their preparation, these nurses are very well positioned to offer treatment for insomnia but need additional training in the application of CBT-I. Examples of training opportunities are provided in Chapter 23, The Role of Advanced Practice Nurses (APNs) in Specialized Sleep Practice.

Assessment of sleep in the psychiatric outpatient setting needs to follow standard approaches to sleep (see Chapter 5, Conducting a Sleep Assessment), which includes physical and mental status examinations, a sleep health questionnaire, sleep-wake logs, and other objective measures of sleep and wakefulness. Because of the impressive impact of psychiatric illness on sleep, other assessment measures would provide specific data to the assessment of the patient with psychiatric disease. Table 19.2 details psychometric measures to augment standard sleep assessment in the psychiatric patient. Critical to the ongoing care of the person who is psychiatrically ill is ongoing symptom management and the instruments cited will provide objective data to guide the assessment of progress as well as indicate harbingers of relapse.

All of the elements of CBT-I, for example, sleep hygiene, stimulus control, sleep restriction therapy, relaxation techniques, and cognitive behavioral therapy, may be *selectively* applied. While CBT-I has been widely documented to be effective in primary insomnia, it lacks a broad evidence base for its use in psychiatric illness. However, standard cognitive behavioral therapy (not applied to insomnia) is held as a gold standard for the treatment of anxiety disorders and depression (Hynninen, Bjerke, Pallesen, Bakke, & Nordhus, 2010) and has been widely applied to treatment of substance abuse disorders (Easton et al., 2007). As discussed in Chapter 12 (Sleep and Psychiatric Disorders), sleep regulation is directly linked to recovery. However, in conditions such as the bipolar spectrum disorders, elements of CBT-I may have negative effects. Sleep restriction in a person with bipolar illness potentially can trigger mania. No systematic evaluation of sleep restriction therapy in bipolar illness has been conducted, and this may be related to the associated risk of

Table 19.2 ■ *Common Psychometric Measures to Supplement Sleep Evaluation*

Depression:

- Beck Depression Inventory [BDI] (http://www.musc.edu/dfm/RCMAR/Beck.html)
- Center for Epidemiologic Studies Depression Scale [CESD] (http://apm.sagepub.com/content/1/3/385)
- Hamilton Rating Scale for Depression [HAM-D] (http://healthnet.umassmed.edu/mhealth/HAMD.pdf)

Bipolar Disorder:

- Mood Disorders Questionnaire [MDQ] (http://www.dbsalliance.org/pdfs/MDQ.pdf)
- Young Mania Rating Scale (http://www.atlantapsychiatry.com/forms/ymrs.pdf)
- Mood Chart (http://www.manicdepressive.org/moodchart.html)

Schizophrenia:

- Positive and Negative Symptom Scale [PANSS] (http://www.bli.uzh.ch/BLI/PDF/panss.pdf)
- The Calgary Depression Scale for Schizophrenia (http://www.ucalgary.ca/cdss/)
- Brief Psychiatric Rating Scale (http://www.public-health.uiowa.edu/icmha/outreach/documents/BPRS_expanded.PDF)

Post Traumatic Stress Disorder:

- Clinician Administered PTSD Scale (http://portal.wpspublish.com/portal/page?_pageid=53,70500&_dad=portal&_schema=PORTAL)
- Clinician Administered PTSD Scale for Children and Adolescents (http://portal.wpspublish.com/portal/page?_pageid=53,70504&_dad=portal&_schema=PORTAL)
- Post Traumatic Stress Disorder Scale (http://www.ptsd.va.gov/PTSD//professional/pages/assessments/caps.asp)

Anxiety Disorders:

- Panic and Somatization Scale (http://www.kaaj.com/psych/scales/pansoma.html)
- Beck Anxiety Inventory (http://www2.massgeneral.org/schoolpsychiatry/screening_anxiety.asp#BAI)
- Yale-Brown Obsessive Compulsive Scale [Y-BOCS] (http://www.ocdrecoverycenters.com/ocd/ybocs.pdf)

Alcoholism:

- CAGE Alcohol Screening (http://counsellingresource.com/quizzes/alcohol-cage/index.html)

Rapid Alcohol Problem Screen [RAPS4] (http://alcoholism.about.com/od/tests/a/raps.htm

triggering relapse in those diagnosed with bipolar disorder (Smith, Huang, & Manber, 2005). However, there is significant evidence that supports the use of cognitive behavioral therapy in the treatment of insomnia in patients with some psychiatric illnesses. Edinger et al. (2009) report that CBT-I is effective in both primary and secondary insomnia related to *nonpsychotic* conditions. No studies are currently available to determine the effects of CBT-I in psychotic illnesses. The reason for this gap in the literature is probably related to the fact that psychotic disorders are classified as thought disorders and are often characterized by cognitive difficulties.

For persons with psychotic disorders, sleep difficulties are treated somewhat differently, using some elements of the CBT-I repertoire. As discussed in Chapter 5 (Conducting a Sleep Assessment), sleep in schizophrenia is characterized by poor sleep efficiency, reduced total sleep time, and protracted sleep latencies, and these sleep characteristics may reflect the pathophysiology of the disorder. Sleep disturbances in those with schizophrenia are often addressed with sedating antipsychotic medications such as quetiapine (Philip, Mello, Carpenter, Tyrka, & Price, 2008). Second generation antipsychotic medications have the capacity to address both sleep disturbance and the psychotic symptoms common to the disease. However, the use of these medications does not eliminate the need for behavioral measures. Sutton (2004) discusses the importance of helping the patient recognize early warning symptoms of relapse, which he terms as *relapse signatures*. This risk-reduction strategy involves helping the patient identify changes in sleep as an early warning sign for relapse, and to use this symptom

as an indicator of the need for intervention and help. Schizophrenia and other psychotic disorders are characterized by periods of stability and relapse. Ongoing close monitoring will reduce recidivism and associated disability. Additional behavioral measures that can be used in the treatment of sleep disturbances in people with psychotic disorders are sleep hygiene, relaxation techniques, and stimulus control.

Table 19.3 outlines a standard treatment plan to address sleep disturbances in the outpatient setting. Pharmacotherapy plays an important role in sleep across psychiatric illnesses; refer to Chapter 12 (Sleep and Psychiatric Disorders) for details related to the treatment of the psychiatric illnesses as well as FDA approved and off-label medications to treat associated insomnia. The role of the nurse in the outpatient treatment setting involves standardized approaches to assessment of sleep in conjunction with the presenting symptoms of the psychiatric disorder, recognizing the shared bidirectional relationship. The nature of outpatient work allows the nurse the opportunity for the development of a long-term relationship with the patient. Though the psychiatric nurse will be involved in a variety of

Table 19.3 ■ *Outpatient Treatment Plan for Sleep Disturbance and Comorbid Psychiatric Illness*

Admission:

- Gather information from collaborators in the patient's care, including information from inpatient hospitalizations, previous psychotherapists/psychopharmacologists, and primary care providers.
- Provide structured sleep assessment as a dimension of the broader outpatient admission evaluation. (See Chapter 5, Conducting a Sleep Assessment.)
- Utilize psychometric measures to clearly identify the target symptoms at the time of admission and to develop specific interventions aimed at their abatement. Repeated use of these measures provides benchmarks of progress across the treatment period.
- Develop a treatment plan based on assessment data (e.g., sleep-focused plan, psychotherapeutic plan, pharmacologic strategies).

During Initial Treatment:

- Initiate ongoing use of sleep-wake logs.
- Provide information related to sleep hygiene, stimulus control and the use of relaxation techniques.
- Introduce the elements of CBT-I and discuss the role of CBT-I in the larger psychotherapeutic plan.
- Assess the appropriateness of utilizing sleep restriction, given the associated risks for patients with bipolar disorder or schizoaffective disorder, bipolar subtype.
- Discuss formal CBT-I sessions as a compartmentalized dimension of the overall psychotherapy treatment plan, though ongoing sleep monitoring will be continued throughout treatment, given the potential presence of sleep-associated *relapse signatures* and their relationship to potential for relapse during treatment.
- Conduct CBT-I sessions (different models for the CBT-I have been developed by Edinger and Carney (2008) and Perlis, Jungquist, Smith and Posner (2005)).

During Continuation Treatment:

- Reinforce CBT-I skills.
- Continue to address risk reduction and relapse prevention from a sleep-focused perspective.
- Monitor medications in an ongoing manner with a focus on potential for iatrogenic impacts on sleep quality.
- Manage symptoms with various therapies used within the treatment plan.

At Discharge:

- Reinforce relapse prevention and risk reduction strategies.
- Discuss the importance of early symptom identification and the associated need for early intervention if needed.
- Review target symptoms of the disorder as they presented, and use these symptoms as teaching points for relapse prevention.

Reinforce the importance of sleep hygiene, stimulus control, use of relaxation techniques and cognitive strategies in maintaining quality sleep.

Abbreviation: CBT-I, cognitive behavioral therapy for insomnia.

ongoing patient teaching efforts (medication, relapse prevention, risk reduction), provision of a wide range of mental health services is central to the role of nurses in this specialty. Acquiring knowledge and skills specific to sleep provides a new and essential dimension to psychiatric nursing practice.

Community-Based Settings

The notion of nurses engaging different factions of the community in sleep-related work is novel and to date, there are a few studies that have examined sleep disruption from a broader community perspective. The few studies that are in the literature mainly focus on elders (Fragoso & Gill, 2007; Williams, 2009). However, one article describes a programmatic approach to address the learning needs of owners, operators, and direct care providers in adult family homes for residents diagnosed with dementia (McCurry, LaFazia, Pike, Logsdon, & Teri, 2009). The innovative program, *Sleep Education Program* takes a different direction with community-based education and provides an exemplar for meeting community-based needs for sleep education.

The needs for public education in the area of sleep are enormous. Among those with psychiatric disease, the learning needs are even significantly greater, though no data exist on the magnitude of the problem in this group. One does not have to look far within the health care disciplines to see the vastness of sleep-related learning needs among health care providers. It is safe to assume that these learning needs are amplified in the general public. Among those with psychiatric illnesses, the vulnerabilities for sleep disorders have been well documented and discussed in Chapter 12 (Sleep and Psychiatric Disorders). The iatrogenic risk for weight gain related to many of the drugs used to treat the diseases is great and is associated with risk for obstructive sleep apnea, restless legs syndrome, or periodic limb movement disorder. These sleep disorders then degrade sleep quality and provide the *perfect storm* for treatment-related sleep disturbances and subsequent risk for poor control of the underlying psychiatric illness. The implications for public health are immense and underscore the need for public education on sleep.

Creative approaches to addressing learning needs of the community bifurcate into program development to assess the breadth of the needs within a given community and then to create programs to meet these needs. Sources of collaboration are broad-based. Faculty from the Department of Nursing at the University of Massachusetts Lowell have engaged the local adult education to offer free classes to members of the community focused on sleep, depression, and post-traumatic stress disorder. The groups have been overwhelmingly well attended by members of the community. The faculty members have been approached to offer similar programs to the local community health boards and organizations, through local churches, temples, and community health centers across the city. These casual invitations provide great opportunities to begin community assessment and to formulate programs designed to target community members who are in need of information related to sleep and health/disease. Nursing faculty and students provide the education, and these experiences can easily be incorporated into community health clinical rotations. Clearly, the opportunities for alternative approaches to student education impact the education of future health care professional. Given the great needs for public and professional sleep education, nurses can play a powerful role in shaping public and professional awareness of sleep and health-related concerns.

Addressing the learning needs of both nursing students and practicing nurses will require innovative approaches as well. While the work by Lee, Landis, Chasens and colleagues (2004) proposes appropriate curriculum for both undergraduate and graduate nursing students in the area of sleep and chronobiology, similar papers need to be written to advise the community of nurse educators about options for meeting the needs of practicing nurses as well. Preconference workshops, online learning, continuing education offerings all provide venues for beginning the process of meeting these very broad learning needs.

CASE STUDY I'm Afraid that I'll Never Sleep Normally Again in My Life

Jana is a 36-year-old woman who presents at your practice on the referral of her primary care provider for treatment of protracted postpartum depression, severe anxiety, and insomnia characterized by difficulty falling asleep and staying asleep. She has had these symptoms since the birth of her second child 5 months ago. She notes that she has "tried everything to help me with sleep but nothing works . . . clinicians just keep giving me more drugs and I'm afraid that I'm going to get addicted . . . " She has seen four clinicians for sleep-related complaints over the last 5 months and usually leaves their practices feeling frustrated that "nobody can help me . . . I am really discouraged and having a hard time taking care of my kids."

Objectively, she has seen four psychiatric clinicians in the last 5 months. She reports that she has in the last 5 months been given zolpidem 10 mg, olanzapine 50–100 mg, clonazepam 1 mg, trazodone 100–200 mg, diphenhydramine 50 mg, and mirtazapine 15 mg, all aimed at enhancing sleep, though the lattermost medication was prescribed for depression and sleep. Her current regimen for sleep is zolpidem 10 mg and clonazepam 2 mg before bedtime, which she says "helps me to get to sleep but 3 hours later I am awake and then I get terrified with not being able to get back to sleep." She reports that she took the other medications for "just a short time because I couldn't stand the side effects. I gained 10 pounds with mirtazapine, felt really drugged all day with the olanzapine, and had severe dry mouth with the diphenhydramine." She fears that her sleep deprivation will yield sleepiness during the day will prevent her from caring for her children.

On exam, she is engaged and interactive. She appears severely tired and sleepy. Her affect and mood are extraordinarily anxious and depressed. She denies suicidal thinking and has no evidence of psychosis. Her cognitive functions are intact though she notes that "my memory stinks since I can't sleep." She has no intention of neglecting or harming her children. In fact, her greatest concern is that she'll fall asleep and "something may happen to them." Her self-care is satisfactory though her appetite is poor and she has lost 10 pounds in the last 3 months.

Your evaluation consists of sleep-wake logs, a sleep questionnaire, and psychometric measures of anxiety and depression. The data show severe anxiety and moderate depression. Her sleep hygiene is poor and she notes that as the nighttime approaches, she begins to get more anxious about not sleeping. She has a number of perceptual distortions around sleep, particularly focused on worries that her sleep patterns have been permanently damaged and that she will live the rest of her life in this condition.

Your plan is comprehensive. You have her return to her primary care provider for a full physical exam, which is negative, save the weight loss. All labs are normal. The full psychiatric evaluation shows that she has had two episodes of depression in the past after the births of her other children. She notes that she had taken amitriptyline 100 mg in the past, which she said was helpful but she had difficulty with urination, particularly urinary hesitancy. She had been started on 50 mg for 5 days, then increased to 100 mg a day. The urinary difficulties occurred 2 days after the dose was raised to 100 mg.

Looking at options, you suggest that she remain on the sleeping medications and return to the use of the amitriptyline at a low dose (25 mg). This medication taken at bedtime helps with sleep, given its soporific effects. After a week, you raise the dose to 50 mg and within a week, her anxiety is less and she is feeling less depressed "and sleeping better but it's still not great." Given her improvement, CBT-I is started and she actively engages it. She sees how her patterns of perception and thinking about sleep perpetuate the sleep disturbance and how sleep hygiene, stimulus control, relaxation exercise, and the cognitive work is helpful. After two CBT-I sessions, a taper of the clonazepam is slowly started. A slight return of middle of the night awakening is addressed with a dose increase of the amitriptyline to 75 mg, and this dose adequately controls the associated anxiety, without anticholinergic side effects. Continuing the CBT-I, she is able to taper off the dose of the clonazepam and zolpidem. The plan is to continue the amitriptyline for 9–12 months for the treatment of depression and to continue psychotherapy aimed at enhancing her coping skills.

(*Continued*)

CASE STUDY *Continued*

Critical Thinking Questions

1. What do you think was the rationale for continuing the sleep medications even after CBT-I was started?
2. What were the main themes in her thinking that were targeted for improvement with CBT-I and why?
3. What are the principles on which CBT-I is based for this woman?
4. Outline a teaching plan for this woman's care and discuss how you would monitor her progress across the treatment period.

CONCLUSION

Across all specialties and levels of nursing practice, nurses deal directly with patients who live with psychiatric illness. Sleep disorders are common within this population of patients and are a source of significant morbidity and mortality. Accurate symptom identification, thorough assessment, and evidence-based intervention strategies related to sleep in those with psychiatric illness will have a powerful impact on public health. In order to facilitate increasing awareness of sleep in psychiatric nursing practice, a multifaceted approach needs to be developed that involves professional nursing education as well as health care and community organizations. Broad-based education is an essential key to facilitating the culture shift within these groups to embrace sleep-related science to the benefit of public health.

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Sleep in Adult Acute and Critical Care Settings

20

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Poor sleep is very common among adult patients who are hospitalized in acute and critical care settings and is characterized by partial to complete sleep deprivation and alterations in circadian rhythms of activity/rest and sleep. Primary sleep disorders, such as insomnia, sleep-disordered breathing, and parasomnias are also prevalent. Factors that contribute to sleep disorders during hospitalization are multifactorial and include clinical and demographic characteristics of patients, nature of the illness and treatment, and the hospital environment. Although research on the consequences of sleep disorders among acute care patients is limited, it is likely that sleep has profound effects on important physiological, behavioral, and functional status outcomes. The purpose of this chapter is to discuss the characteristics of sleep, factors associated with sleep, and evidence-based strategies to promote sleep in acute and critical care settings. Implications for nursing practice and research are discussed.

CHARACTERISTICS OF SLEEP IN ACUTE AND CRITICAL CARE PATIENTS

Consistent with the perspective that sleep is a multidimensional biobehavioral phenomenon, researchers have employed self-report, behavioral, and polysomnographic measures to study sleep in acute and critical care settings. Despite the wide variety in patient populations studied, the consistency in findings is remarkable: Many patients have severe sleep disturbance and report that it is stressful. Acutely ill patients report that their sleep is shorter in duration, associated with more frequent awakenings, and of poorer quality than sleep obtained at home (Freedman, Gazendam, Levan, Pack, & Schwab, 2001; Nicolas et al., 2008; Topf, Bookman, & Arand, 1996; Topf & Davis, 1993; Tranmer, Minard, Fox, & Rebelo, 2003). Earlier bedtimes, earlier morning awakenings, and nightmares are common (Frisk & Nordstrom, 2003; Meyer et al., 1994).

Objective characteristics of sleep disturbance in acutely ill patients include severe sleep fragmentation, electrophysiological arousals, and sleep-stage deprivation, including increases in Stage N1 sleep, decreases in Stages N2, N3, and REM sleep (Freedman et al., 2001; Friese, Diaz-Arrastia, McBride, Frankel, & Gentilello, 2007; Gabor, Cooper, & Hanly, 2001), and changes in circadian rhythms (Olofsson, Alling, Lundberg, & Malmros, 2004). Although some patients do not sleep at all, there is evidence that others have sufficient sleep time over the 24-hour day, but its quality is poor (Freedman et al., 2001). Sleep occurs throughout the 24-hour day, with as much as 50% during daytime hours (Redeker, Mason, Wykpisz, & Glica, 1996). This suggests the importance of monitoring sleep throughout the 24-hour day.

FACTORS ASSOCIATED WITH SLEEP DISTURBANCE IN ACUTE CARE PATIENTS

As depicted in the organizing framework, adapted from Redeker and Hedges (2002) (see Figure 20.1), sleep disturbance is a multifactorial problem in acutely ill patients. Contributing factors are also multifactorial and include prehospitalization characteristics, the nature

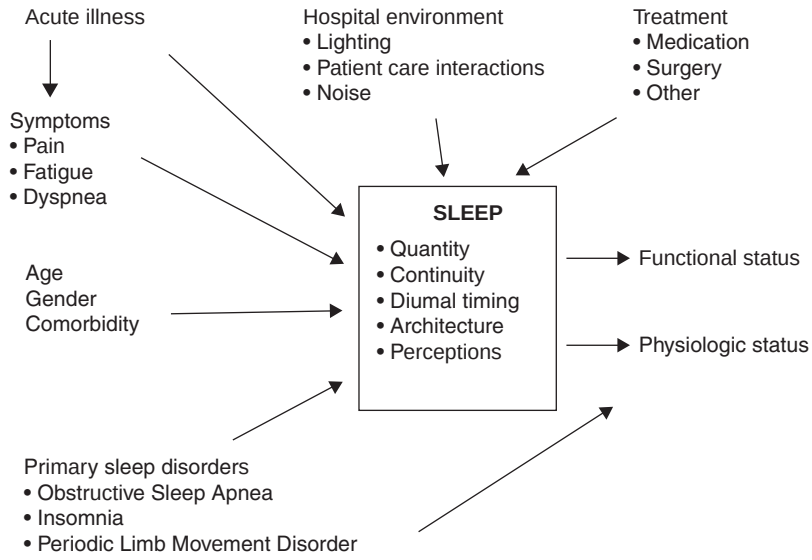

Figure 20.1 ■ Correlates and consequences of sleep in acutely ill patients. *Source:* Adapted from "Sleep During Hospitalization and Recovery After Cardiac Surgery," by N. S. Redeker and C. Hedges, 2002, *Journal of Cardiovascular Nursing, 17*(1), pp. 5–68.

of the index illness, hospital environment, and treatment (Redeker, 2000). Numerous studies examined the individual relationships between these factors and sleep patterns, but multifactorial studies are sparse.

Prehospitalization Factors

Prehospitalization factors that may contribute to poor sleep during acute care hospitalization include demographic, clinical, and disease-related characteristics (e.g., chronic health conditions, health status). Pre-existing sleep disorders (e.g., sleep apnea, insomnia, periodic limb movement disorder) are common and may contribute to poor sleep and complicate recovery.

Demographic Factor

Aging is a potentially important influence on sleep during acute care hospitalization. Although there are age-related changes in normal sleep (see Chapter 2, Developmental Aspects of Normal Sleep), most experts believe that sleep disturbance is a result of poor health in older individuals. Therefore, the effects on sleep during hospitalization may be secondary to chronic illness or primary sleep disturbances. On the other hand, the large number of older patients in acute care settings suggests the importance of understanding their needs relative to sleep. It is possible that normal developmental features (e.g., less robust circadian rhythms) may predispose older adults to poor sleep in the hospital. Likewise, there are gender differences in sleep and the gender prevalence of some sleep disorders (e.g., men in some age groups have higher levels of sleep apnea, but women may have more insomnia). (See Chapter 3, Gender and Sleep.)

Health Status and Comorbidity

Health status may also have an important influence on sleep during acute care hospitalization. In studies of medically treated patients with ischemic heart disease (Redeker, Tamburri, & Howland, 1998) and cardiac surgery patients (Redeker, Ruggiero, & Hedges, 2004a,b), age, New York Heart Association Functional Classification (a functional status measure), gender, and prehospitalization sleep patterns predicted

sleep during hospitalization. In another study, comorbidity was the best predictor of insomnia during hospitalization (OR 7.9, SE 0.85, $p = .01$) (Isaia et al., 2010). These findings suggest the multifactorial contributions of preoperative patient characteristics to sleep during hospitalization.

Sleep Disorders

Sleep disorders (e.g., insomnia, sleep apnea, movement disorders) are common in the general population and may contribute to poor sleep during hospitalization. (See Chapter 6, Insomnia.) Although prehospitalization sleep disturbance predicted poor sleep after cardiac surgery (Redeker et al., 2004), the contributions of specific sleep disorders have been understudied.

Obstructive sleep apnea (OSA) is prevalent in patients with cardiovascular disorders, diabetes, and obesity. (See Chapter 7, Sleep-Related Breathing Disorders.) Unfortunately, OSA is underdiagnosed, and many more patients likely have the diagnosis than have been identified. These patients are at high risk for the negative consequences of OSA, including airway collapse, hypoxemia, sleep fragmentation and sympathetic activation and therefore prone to complications associated with the use of opioids, sedative medications, and anesthetics that contribute to respiratory depression.

Sleep-disordered breathing may worsen during acute care hospitalization. For example, central sleep apnea/Cheyne-Stokes breathing often worsens in patients with decompensated heart failure and in patients with arrhythmias and myocardial ischemia (Tsukamoto & Ohara, 2006).

Many chronic medical conditions, such as heart disease, diabetes, chronic obstructive pulmonary disorders, renal disease, and Parkinson's disease, and psychiatric disorders are also closely associated with the development of insomnia. (See Chapter 11, Sleep in Medical Disorders; Chapter 12, Sleep and Psychiatric Disorders; and Chapter 6, Insomnia.) Hospitalization may exacerbate insomnia in these patients, but acute illness and hospitalization may be a precipitating factor for the development of chronic insomnia.

Acute "Index" Illness or Injury

Virtually all medical disorders and injuries can result in sleep disturbance during acute care hospitalization. The direct effects of illness or injury on the brain itself (e.g., head injury, stroke, ischemia, sepsis, altered electrolytes) or illness-related symptoms (e.g., pain, anxiety) may contribute to poor sleep. For example, patients with acute and chronic head injury (Valente et al., 2002), adult respiratory distress disorder (Lee et al., 2009), or acute heart failure report profound sleep disturbance. Hypersomnolence often occurs in hospitalized patients with infectious disorders such as viral and bacterial infections (Parish, 2009). (See Chapter 11, Sleep in Medical Disorders.) Increased sleep disturbance associated with the exacerbation of medical conditions (e.g., heart failure, chronic obstructive pulmonary disease, asthma, and renal disease) may be a prodromal sign (like dyspnea in cardiopulmonary disorders) that signals the need for acute intervention.

Treatment

Many treatments, including surgery, anesthesia, medications, and mechanical ventilation, among others, have unintended negative consequences on sleep.

Anesthesia and Surgery

Anesthesia and surgery have negative effects on sleep architecture and often result in sleep deprivation and fragmentation (Kaw et al., 2006; Moos & Cuddeford, 2006; Orr & Stahl, 1977) but may also alter circadian rhythms of sleep-wake and other physiological processes. However, it is also difficult to discern the separate contributions of each treatment because they co-occur.

Hospitalized cardiac surgical patients have frequently been studied because of evidence of persistent sleep disturbance. They generally have poor self-reported and objective nocturnal sleep quality, short duration, high degrees of fragmentation, poor sleep efficiency, and large amounts of daytime sleep, especially during the first few postoperative days (Edell-Gustafson & Hetta, 1999; Edell-Gustafson, Hetta, & Aren,

1999; Hedner, Caidahl, Sjoland, Karlsson, & Herlitz, 2002; Johns, Large, Masterson, & Dudley, 1974; Orr & Stahl, 1977; Redeker & Hedges, 2002, 2006; Redeker et al., 1996; Redeker et al., 2004a,b; Simpson, Lee, & Cameron, 1996). As much as half of daily sleep occurs during the day (Edell-Gustafson et al., 1999; Hedner et al., 2002; Knapp-Spooner & Yarcheski, 1992; Redeker et al., 1996).

Evidence of dramatic improvements in sleep consolidation and increasing organization of circadian patterning within the first few postoperative days during hospitalization in cardiac surgery patients with uncomplicated recovery (Redeker, Mason, Wykpisz, & Glica, 1995; Redeker, Mason, Wykpisz, Glica, & Miner, 1994) suggests that sleep improvements are not solely due to the hospital environment, but profoundly influenced by surgery and anesthesia. In the case of cardiac surgery patients, the cardiopulmonary bypass pump may also have an influence. There is a need for systematic study of other groups of medical and surgical patients. Although there have been isolated studies, the majority have included very small samples.

Innovations in surgical treatment, anesthesia, and related care for cardiac and other surgical patients, such as shorter surgical times and newer anesthetic agents, may have an influence on sleep. However, the sleep-related consequences of such changes are understudied. Changes in use of these procedures make it difficult to compare studies done in different institutions or at different times. As treatments improve, there is a need to study their impact on sleep, as well as related physiological and functional consequences. For example, patients with "off-pump" cardiac surgery had better objective sleep continuity than those with traditional "on-pump" procedures (Hedges & Redeker, 2008).

Anesthesia is especially problematic for patients who have sleep apnea. It may lead to collapse of the upper airway and has deleterious effects on sleep architecture, such as decreases in REM sleep (Ead, 2009; Hwang et al., 2008; Kaw et al., 2006; Moos & Cuddeford, 2006; Poirier et al., 2009). Cardiac surgical patients who had sleep apnea had longer intensive care unit stays, higher rates of infection, and encephalopathy (Kaw et al., 2006), while a group of elective surgery patients with mixed surgical procedures sleep apnea had more complications, including bleeding and cardiovascular, respiratory, and gastrointestinal disorders (Hwang et al., 2008).

REM sleep rebound after REM suppression during the early postoperative period may worsen respiratory complications in patients who continue to receive narcotics or other respiratory depressant medications, especially in patients with OSA. Edema of the airway and hypertension may also complicate postoperative recovery.

Medications

Virtually all medications can negatively influence sleep, especially those with effects on the central nervous system (see Table 20.1). Acute withdrawal of substances, such as benzodiazepines, opioids, nicotine, caffeine, and alcohol contributes to sleep disturbance, and some medications (e.g., sedatives, opioids) may worsen pre-existing sleep disorders, such as sleep apnea or periodic limb movement disorder (Weinhouse, 2008). Although sedative agents may promote sleep, sedation is not sleep and does not have its restorative benefits (Weinhouse & Watson, 2009).

Mechanical Ventilation

Mechanical ventilation is another significant cause of sleep deprivation (Parthasarathy & Tobin, 2002). In one study, 60% of patients who receive mechanical ventilation reported poor sleep, and 30% reported associated panic or anxiety, but survivors did not recall poor sleep during this time period (Hardin, 2009). It is likely that the poor sleep of mechanically ventilated patients is not due to the ventilator alone, but due to acute illness, the hospital environment, and other treatments. However, altering ventilator modes may have an impact on sleep.

Symptoms

Pain, dyspnea, and anxiety are prevalent in acutely ill patients. Although numerous studies separately described pain and sleep as concerns of acutely ill patients, there has been surprisingly

Table 20.1 ■ *The Effects of Common Medications on Sleep in Hospitalized Patients*

Medications (Examples)	Sleep Alterations	Adverse Effects
Analgesics		
■ Opioids (M.S., Codeine, Demerol)	↓TST, REM, SWS; ↑W	Sedating properties may worsen SDB
■ NSAIDs (Ibuprofen)	↓TST, SE	
Antidepressants		
■ Tricyclics (Amitriptyline, doxepine	↓W, REM; ↑TST	Drowsiness; CNS depression enhanced by alcohol intake
■ SSRIs (Paroxetine, fluoxetine	↓TST, SE, REM; ↑W	Insomnia, agitation; extensive drug interactions
Antiepileptics		
■ Phenytoin	↓SL; ↑SWS, TST	CNS effects; extensive drug interactions
■ Phenobarbital	↓W, SL, REM; ↑TST	Sedating effects common
■ Carbamazepine	↓LS, REM; ↑SWS	Drowsiness, fatigue
■ Gabapentin	↓W; ↑TST, REM, SWS	Dream disturbances, emotional lability, dizziness, drowsiness
Anti-Parkinsonian drugs		
■ Levadopa	↓SWS; nightmares	Disturbing dreams, mood changes, malaise
■ Methyldopa		Drowsiness, nightmares
Antipsychotics		
■ Haloperidol	↓W, SL; ↑SE	Insomnia, restlessness
Cardiovascular agents		
■ β antagonist (propanolol, metoprolol)	↑W, SL; ↑REM	Drowsiness, fatigue
■ Calcium channel blockers (Nifedipine, verapamil)	NA	Drowsiness, weakness Fatigue, syncope
■ ACE Inhibitors (lisinopril)	No known sleep effects	Insomnia, weakness, nocturia
■ Diuretics (HCTZ, furosemide)	NA	
Corticosteroids		
■ Prednisone; cortisone	↓REM, SWS; ↑W	Insomnia, restlessness, behavior changes
H2 Antagonists		
■ Cimetidine	↑SWS	Hallucinations, somnolence
Mood Stabilizers		
■ Lithium	↑TST, SWS; ↓REM	Drowsiness, lethargy
Respiratory		
■ Theophylline	↓TST, SE, REM, SWS; ↑W	Agitation, insomnia
Sedatives/Hypnotics		
■ Benzodiazepines (Midazolam)	↓W, REM, SWS, SL; ↑TST	Delirium, dreaming, insomnia, nightmares
■ Propofol	↓W, SL; ↑TST	CNS depressant; hypnotic

Source: "Sleep in Hospitalized Medical Patients, Part 2: Behavioral and Pharmacologic Management of Sleep Disturbance," by J. S. Young, J. A. Bourgeois, D. M. Hilty, and K. A. Hardin, 2008, *Journal of Hospital Medicine, 4*, pp. 50–59. "Pharmacology I: Effects on Sleep of Commonly Used ICU Medications," by G. L. Weinhouse, 2008, *Critical Care Clinics, 24*(3), pp. 477–491.
Abbreviations: NA, not available; REM, rapid eye movement sleep; SDB, sleep-disordered breathing; SE, sleep efficiency; SL, sleep latency; SWS, slow-wave sleep; W, wakefulness; TST, total sleep time.

little study of the associations between these symptoms and sleep in the acute care setting. Poor sleep may be either a cause or a consequence of pain and anxiety. These symptoms may also cluster together or in combination result from the underlying disorder.

Environmental Influences on Sleep in Acute Care

The influence of the acute care environment on patients' sleep has been of interest to clinical nurses and nurse scientists for more than 30 years.

Studies have focused on noise, lighting, and interruptions of sleep due to patient care activities.

Noise

Health care professionals, patients, and researchers often attribute poor sleep to noise. Peak sound levels throughout U.S. hospitals often exceed the recommended 35–45 dB (A) (Christensen, 2005; Environmental Protection Agency, 2009) and may be as high as 80 dB (A) throughout the 24-hour period (Aaron et al., 1996; Christensen, 2005; Kahn et al., 1998; MacKensie & Galbrun, 2007). These levels are comparable to the noise of a busy highway and exceed the federal regulations for the threshold at which a worker must wear earplugs (Joseph & Ulrich, 2007). Unfortunately, noise levels in acute care hospitals are getting higher (Busch-Vishniac et al., 2005).

Overall noise levels, but not peaks in sound levels, were associated with electrophysiological arousals in normal subjects exposed to recorded ICU sounds (Stanchina, Abu-Hijleh, Chaudhry, Carlisle, & Millman, 2005). Reverberation, "the persistence of sound that results from reflections of the sound after the source has stopped" (Joseph & Ulrich, 2007) associated with hard surfaces in construction materials, may cause noise to propagate down hallways and corridors. In noisy hospital units, staff may raise their voices to be heard over the background noise and further add to the sound levels.

Patients consistently identify noise from alarms, call bells, banging of equipment, doors banging, loud voices, talking at the nurses' station, and noises from other patients, as contributors to sleep disruption (Freedman et al., 2001; Freedman, Kotzer, & Schwab, 1999; Nicolas et al., 2008; Simpson & Lee, 1996; Southwell & Wistow, 1995). Healthy females exposed to taped CCU sounds had more prolonged sleep latency, less total sleep time, and poorer sleep quality than a control group (Topf et al., 1996; Topf & Davis, 1993).

An important feature of "noise" is the degree to which it is annoying. For example, recorded acute care staff conversation was more likely than other noise sources to cause EEG arousals in healthy subjects, and conversations and alarms were the most disruptive among a variety of other sources of noises (Gabor et al., 2003). Patients' reports that staff talking was the most highly annoying source of noise (Redeker & Olsen, 2001, unpublished data) and the report that patients were more likely than nurses to report that the nurses' talking was a source of noise (Southwell & Wistow, 1995) indicate the need to reduce unnecessary staff talking during nocturnal hours.

Polysomnographic studies demonstrated that noise is only one factor that affects sleep in the acute care setting. For example, environmental noise explained only 11%–20% of electrophysiological arousals and 17% of awakenings in critically ill patients (Freedman et al., 2001; Gabor et al., 2003). Therefore, other factors are likely to play an important role.

Lighting

Light serves as an environmental cue or "zeitgeber" that helps to regulate the circadian cycle. Continual 24-hour exposure to bright lights in the ICU adversely affects the circadian cycle and sleep. Conversely, exposure to light levels that resemble normal diurnal variations may improve circadian rhythms and sleep. It is possible to regulate diurnal rhythms of light in the hospital through design modifications and implementation of guidelines (Meyer et al., 1994; Walder, Francioli, Meyer, Lancon, & Romand, 2000). The extent to which these changes improve sleep requires further study.

Nocturnal Care Disruptions

The continuous nature of patient care in acute care settings, especially intensive care units, is an important source of sleep disruption. Patient care activities that disrupt sleep include monitoring, assessment, and therapeutic and personal care activities (Kahn et al., 1998; Monsen & Edell-Gustaffson, 2005; Patel, Chipman, Carlin, & Shade, 2008; Tamburri, DiBrienza, Zozula, & Redeker, 2004; Topf et al., 1996). Review of the nursing records of ICU patients revealed an average of 42 patient care interactions over a

12-hour night shift (7 PM–7 AM), with the most frequent at midnight (Tamburri et al., 2004). The fact that only 6% of the 147 nights included 2–3 hours of uninterrupted time suggests that few patients had the opportunity for complete sleep cycles (~90 minutes). Another team obtained similar results in a study (Celik, Oztekin, Akyolcu, & Issever, 2005) conducted in Turkey. While many patient care activities are necessary due to patient acuity, the common practice of bathing patients in the middle of the night (Tamburri et al., 2004) suggests that some activities may be scheduled according to routine and ritualistic practice rather than evidence and suggests the need to reorganize care to allow sufficient time to sleep.

OUTCOMES OF SLEEP DISTURBANCE

Because the factors that contribute to sleep and its outcomes are multifactorial, it is often difficult to quantify the direct effects of sleep disturbance. Outcomes of interest include functional performance (e.g., ability to perform activities of daily living and physical activity, cognition, memory, and learning); physiological status/recovery; and patient satisfaction. Cost of care is another important outcome, but it is understudied.

Functional Outcomes

As suggested by the growing literature on sleep in the general population and in clinical and laboratory studies, sleep disturbance has a profound impact on functional performance (e.g., ability to perform activities of daily living and physical activity, cognition, memory, and learning). Patients must be able to function sufficiently to perform self-management activities at hospital discharge. Circadian rhythms of activity-rest and sleep patterns were associated with self-reported functional outcomes at the time of hospital discharge among two groups of cardiac surgical patients (Redeker et al., 1994; Redeker et al., 2004a,b), and organized sleep patterns during the acute care period were predictors of disability and survival in patients with head injury (Evans & Bartlett, 1995; Valente et al., 2002). However, in another study (Hedges, 2005), no association was found between sleep and postoperative cognitive function.

Delirium is common among acutely ill patients, especially older adults. Based on observations in other settings, sleep deprivation may contribute to delirium in hospitalized patients, and many have speculated that adequate sleep may reduce the risk (Figueroa-Ramos, Arroyo-Novoa, Lee, Padilla, & Puntillo, 2009; Weinhouse et al., 2009). However, it is possible that the relationship between sleep and delirium is bidirectional or that both sleep and delirium are secondary to the same underlying brain dysfunction (Weinhouse et al., 2009). To date, scientific evidence of the relationships between delirium and sleep are lacking. Nevertheless, sleep and delirium seem to go hand-in-hand and measures focused on reducing the likelihood of sleep deprivation may reduce the incidence of delirium.

Physiological Outcomes

Research that explicitly addresses physiological outcomes during acute illness is sparse, but recent growth in understanding of the pathophysiological effects of sleep deprivation, and sleep-disordered breathing in other settings suggest that sleep disorders in the acute care setting may be closely tied to pathophysiological outcomes (Sareli & Schwab, 2008). These are likely to include alterations in immune function and the HPA-axis, inflammatory processes, activation of the neuroendocrine system with elevated catecholamines, derangement of metabolic processes, such as thyroid levels, and glucose and insulin regulation. Alterations in secretion of growth hormone associated with decrease slow-wave sleep may contribute to poor wound healing. These pathophysiological effects are likely to exacerbate the index illness, complicate treatment, and slow recovery.

Patients with OSA (see Chapter 7, Sleep-Related Breathing Disorders) are at high risk for hypoxia, autonomic arousal, cardiac dysrhythmias, sudden death (Chan & Wilcox, 2010; Hersi, 2010; Hoffstein & Mateika, 2009; Pedrosa et al., 2010; Selim, Won, & Yaggi, 2010), myocardial infarction, and stroke. Central apnea is also associated with hypoxia and arrhythmias

and is often found in patients with systolic heart failure. These events may be the precipitating cause of hospitalization, but may also be complications of sleep-disordered breathing in people who are admitted to the hospital for other reasons. Because sleep-disordered breathing is often undiagnosed, nurses must be vigilant for signs of these conditions.

SLEEP PROMOTION IN ACUTE AND CRITICAL CARE SETTINGS

Strategies designed to promote sleep in the acute care setting focus on risk factors, as identified in the model (see Figure 20.1). To be most effective, sleep promoting practices should be incorporated into protocols, procedures, and guidelines and promoted at the unit and institutional levels (Fontaine, Briggs, & Pope-Smith, 2001). Like other evidence-based practice changes, these patient-centered strategies will be most effective if there is support from bedside nurses and other clinicians, but they should also be championed by leaders at the unit and institutional levels. Broad categories of sleep promotion strategies include assessment and identification of risk factors; preventing and managing sleep deprivation; and preventing and managing the potential negative consequences of sleep-disordered breathing. Table 20.2 summarizes strategies for

Table 20.2 ■ *Interventions to Promote Sleep During Hospitalization*

Assessment	Interventions	Evidence
Goal: Reduce Effects of Environmental Stimuli		
Monitor noise, lighting, frequency/timing of patient care interactions; patients, perceptions of environmental stimuli and potential stress	Decrease noise; low light at night; normal lighting during the day Cluster patient care interactions; "Quiet Time" ear plugs/eye masks Massage, music, white noise Provide a structured bedtime routine "PM Care"	Gardner et al. (2009), Hu et al. (2010), Richards (1998), Richardson et al. (2007), Olson et al. (2001)
Goal: Modify Illness and Treatment Related Effects on Sleep		
Assess changes in sleep patterns associated with medications, surgery, and other treatments Treat pain, dyspnea, and other symptoms Assess for signs of delirium	Review drug interactions affecting sleep phases; evaluate sleep effectiveness on all hospitalized patients. Hypnotic medications Prescribe/administer analgesics; Anti-anxiety drugs Behavioral treatments for pain	Bourne and Mills (2004), Hardin (2009), Weinhouse and Watson (2009)
Goal: Prevent and Manage the Negative Complications of Sleep-Disordered Breathing		
Assess for oxygenation, snoring, witnessed apnea, dysrhythmias, especially in patients who have undergone anesthesia or used sedative medications or opioid analgesics	Use minimal sedation necessary Use nonsteroidal pain medication rather than opioids as possible Position patients on side with head of bed elevations minimally at 30 degrees unless contraindicated Apply patient's own home CPAP system if patient not mechanically ventilated. If on mechanical ventilation, consult with care provider for support. Apply CPAP following extubation. Monitor ECG rhythms carefully during episodes of hypoxia, apneas or hypopneas, and snoring. Cardiac monitoring in multiple leads is advantageous. Monitor and document any respiratory abnormalities occurring with ventricular or atrial ectopy and bradyarrhythmias. Postoperatively: Monitor SpO2 continuously for patients at high risk for desaturation, including those with prior SDB. Monitor critically ill patients on mechanical ventilation to prevent over ventilation.	Gross et al. (2006), Young et al., 2008. Kaw et al. (2006), Weinhouse and Watson (2009). Drew et al., 2004; Koshino et al. (2008); Ryan, Juvet, Leung, and Bradley (2008)

Abbreviations: CPAP, continuous positive airway pressure; SDB, sleep-disordered breathing.

monitoring and intervention for sleep disturbances in hospitalized adults.

ASSESSING SLEEP

Admission assessment should include prehospitalization factors that put patients at risk for sleep disturbance (see Table 20.3). It is particularly important that everyone on the health care team be aware of the presence of sleep-disordered breathing so that precautions can be taken to assure appropriate support of the airway during anesthesia or use of sedating medications. Because there are many individuals who have occult sleep apnea, the nurse should pay particular attention to patients who may be at high risk for this disorder. (See Chapter 7, Sleep-Related Breathing Disorders.)

Sleep should be monitored on a daily basis during hospitalization. This should include the duration and patterning of sleep throughout the day, patients' perceptions about sleep, and potential outcomes (e.g., delirium, cognitive impairment, fatigue, sleepiness). Contributing factors, such as medications and other treatments and environmental stimuli are important, and should be modified as possible to promote sleep (see below).

Preventing Sleep Deprivation

Strategies for reducing sleep deprivation focus on modifying environmental stimuli, such as lighting, noise, and the patterning of patient care interactions. Several investigators have evaluated strategies that promote relaxation and/or reduce physiological arousal, such as music or massage, with mixed success. Hypnotic medications are also useful (see Chapter 6, Insomnia), but should be used cautiously due to the potential for drug interactions and negative daytime effects, especially in older adults. Improvements in pain, anxiety, and other symptoms may lead to improvements in sleep duration and continuity, but this has been understudied. Table 20.3 provides an overview of recommended sleep-promoting interventions.

Strategies to reduce the effects of environmental stimuli focus on reducing or eliminating them (e.g., turning off lights, reducing noise, changing the design of hospital units, providing structured time without excessive stimuli), blocking input of environmental stimuli at the patient level (e.g., ear plugs, eye masks, white noise), or modifying patients' response to the physiological arousal associated with the stimuli (e.g., massage, music).

Table 20.3 ■ *Assessment of Sleep in Acute Care Patients*

Prehospitalization Risk Factors

- Gender
- Age
- Comorbid medical or psychiatric conditions
- Traumatic events, mood state
- Pain
- History of sleep disorders
  - Insomnia
  - Sleep apnea
  - Periodic limb movement disorder
  - Restless legs syndrome
  - Parasomnias
- Risk for sleep apnea (obesity, neck size >17 in men, >16 in women, snoring, witnessed apneas, choking, gasping, and daytime sleepiness)
- Treatment for sleep disorders
- Habitual sleep patterns
  - Bedtime
  - Retiring time
  - Wake time
  - Time to fall asleep
  - Frequency and cause of awakenings
  - Bedtime routine
  - Perception of restorative/nonrestorative sleep
  - Shift work or travel across time zones
- Use of alcohol, cocaine, methamphetamines, marijuana, and opioids
- Prescribed and over-the-counter medications
- Alternative/complementary therapies

Sleep During Hospitalization

- Time and duration of sleep periods
- Patterning of sleep throughout the day
- Perception of factors that influence sleep (e.g., comfort, pain, noise)
- Perception that sleep is restorative/nonrestorative
- Consequences: delirium, cognition, fatigue, and anxiety
- Signs of delirium: restlessness and cognitive changes
- Signs of sleep apnea: snoring, apneas, gasping, choking, and hypoxia, electrocardiographic abnormalities

Reduction in Noise, Lighting, or Patient Care Activities

Systematic approaches to improving staff behaviors regarding noise are successful in reducing overall noise, peak levels, and patient disturbance relative to noise (Cmiel, Karr, Gasser, Oliphant, & Neveau, 2004; Hardin, 2009; Kahn et al., 1998; Monsen & Edell-Gustaffson, 2005), but the effects on sleep are not completely known. Only one study (Cmiel et al., 2004) measured sleep after the intervention, but there was no measure of sleep at baseline. Therefore, the impact of the intervention is impossible to evaluate. An educational intervention to reduce noise and light stimuli decreased sound and light levels (Schnelle, Alessi, Al-Samarrai, Fricker, & Ouslander, 1999) in a nursing home, but there were no improvements in observed or actigraphic-recorded sleep. Environmental changes alone may not be sufficient to improve sleep, but should be supplemented with behavioral interventions. This study may have implications for the acute care setting, but further research is needed.

Hospitals have also experimented with using sound meters in the nursing station to alert staff when the noise level becomes too high. These may result in noise reduction in the nursing station as a result of reminding staff about noise levels, but may not be sensitive to the noise in patients' rooms where the noise is most likely to have an impact on sleep.

Designing patient-centered health care environments is an important priority in many hospitals that may contribute to improved sleep. Current recommendations include installation of sound-absorbing ceiling tiles, softer floor surfaces that eliminate reverberation, inpatient units with only single-bed rooms, and noise reduction through replacement of overhead pagers and other noisy communication devices with wireless systems (Joseph & Ulrich, 2007; Taylor-Ford, Catlin, LaPlante, & Weinke, 2008). Systematic research of the impact of improvements in unit design on sleep is lacking.

Blocking Environmental Stimuli at the Patient Level

The use of earplugs to block auditory stimuli holds promise as a means of improving sleep. Investigators noted beneficial effects on REM sleep in studies in which healthy subjects were exposed to recorded critical care unit noise (Hu, Jiang, Zeng, Chen, & Zhang, 2010; Wallace, Robins, Alvord, & Walker, 1999). Hu and colleagues found increases in melatonin (Hu et al., 2010), an important marker of circadian rhythmicity. The use of ear plugs and eye-masks improved self-reported sleep in postoperative cardiac surgical patients (Richardson, Crow, Coghill, & Turnock, 2007), but some patients reported discomfort. The investigators did not consider the separate effects of either eye masks or earplugs. Future randomized clinical trials are needed to evaluate the effects of ear plugs and/or eye-masks—low-cost and patient-centered interventions.

White noise may be effective in masking the variability and specific sources of sound. Among cardiac surgical patients, exposure to recorded ocean sounds, used as white noise to mask noxious sounds, improved self-reported sleep depth, awakenings, and return to sleep in the experimental group (Williamson, 1992). In a more recent study (Stanchina et al., 2005), white noise attenuated electrophysiological arousal during sleep in four healthy subjects exposed to recorded noise from an intensive care unit.

Clustering Patient Care Activities and Scheduling "Quiet Time"

Providing "quiet time" or clustering daily activities and reducing environmental stimuli may be useful in promoting sleep by allowing undisturbed time for it (Edwards & Schuring, 1993; Gardner, Collins, Osborne, Henderson, & Eastwood, 2009; Olson, Borel, Laskowitz, Moore, & McConnell, 2001). "Quiet time" with reduction of light and noise during the hours of 2–4 PM and 2–4 AM (times that correspond to the circadian propensity for sleep) in the neuro-critical care setting led to increase in the proportions of patients who were observed to be asleep during these time periods (Olson et al., 2001). Similarly, rest periods between 2 and 4 PM in an orthopedic unit led to improved sleep. There were also associations between the proportion of patients who were asleep and the decibel level, and patient, family, and health care provider satisfaction with the intervention (Gardner et al., 2009). Although these studies were limited by

their quasi-experimental designs and measurement only of observed sleep, they suggest promise as means to address multiple environmental stimuli (noise, lighting, patient care interactions, and visitors) and improve sleep.

Reducing Patients' Responses to Environmental Stimuli

Music, massage, visualization, and imagery may provide distraction, promote relaxation, and thereby reduce the stress response and promote sleep. For example, cardiac surgery patients who participated in two daily sessions of a music video with soft instrumental music for 2 days after surgery reported better sleep quality on the third postoperative morning than the control group who had only a rest period. There was a nonsignificant trend for the third group, exposed to audio-recorded music, to have improved sleep (Zimmerman, Nieveen, Barnason, & Schmaderer, 1996). The exact mechanism through which music improves sleep quality needs further study as it is not known whether music type (style, tempo, and volume), personal choice, or even method of delivery influences sleep. For example, music delivered via noise-reducing headsets may parallel the impact of a "white noise" intervention, while ambient background music would have quite a different effect. Back massage, compared with use of a muscle relaxation and mental imagery tape and routine coronary unit care (Richards, 1998), resulted in promising, but not statistically significant, trends toward improved sleep efficiency in critically ill men. Despite the promising effects of these studies, there have been few subsequent studies to validate or extend these findings.

Multimodal sleep-promoting interventions may also be efficacious. For example, older adults admitted to a medical surgical unit received a "PM Care" intervention consisting of a warm drink, a back rub, aromatherapy, a warm blanket, relaxing music, and noise reduction. Thirty (75%) responded that this intervention improved their sleep (Robinson, Weitzel, & Henderson, 2005). In another study in which the nursing staff administered a back rub, warm drink, and relaxation tapes administered to older patients admitted to a general medical unit (McDowell, Mion, Lydon, & Inouye, 1998), sleep quality was higher in participants who received more of the components of the intervention, the warm drink, or the back rub. Adherence to all or part of the protocol occurred on 74% of patient days. Adherence was highest for the backrub (52%), warm drink (49%), and relaxation tapes (36%). Patients most frequently refused the relaxation tapes (48%) and least frequently refused the back rub (30%). Rates of hypnotic use declined with the protocol, but chronic hypnotic users were more likely than others to decline to participate in the study. Taken together, these findings suggest that multimodal strategies may have beneficial effects on sleep. However, randomized controlled trials are needed.

Modifying Illness- and Treatment-Related Influences on Sleep

Modifying treatment, where necessary and safe, may result in improvements in sleep. A good example of this approach is modification of ventilator modes in mechanically ventilated patients to be more consistent with patients' respiratory mechanics and improve sleep (Bosma et al., 2007; Fanfulla, Delmastro, Berardinelli, Lupo, & Nava, 2005; Parthasarathy & Tobin, 2002). With careful scrutiny, other patient care practices could be modified. Alternatively, behavioral strategies, such as massage or music, could be used in an anticipatory manner for patients requiring treatments that are likely to disturb sleep.

Medications should be reviewed for their potential impact on sleep disturbance and modified if possible (Bourne & Mills, 2004). Hypnotic agents may be beneficial in promoting sleep in some acutely ill patients (Young, Bourgeois, Hilty, & Hardin, 2009), but their use should be accompanied by or preceded by behavioral and environmental modifications, and they should be used carefully, especially in older adults, because of their potentially negative impact on daytime function and the high risk of delirium. Short-acting medications, such as zolpidem, are generally preferable to drugs with longer half-lives because of the decreased likelihood of falls and other daytime consequences.

Effective management of pain and anxiety—symptoms that are common in acutely ill patients may improve sleep. Although opioid medications

lead to decreases in REM and slow-wave sleep, the beneficial effects on pain may outweigh the effects on sleep architecture (Weinhouse, 2008). Case study 20.1 provides an example of a patient care situation related to the multiple influences on sleep and treatment strategies.

Preventing and Managing the Risks of OSA

Patients who have OSA and undergo anesthesia for surgery, endoscopy, or other procedures are at particular risk for cardiorespiratory complications and mortality. This risk occurs throughout the pre-anesthesia period into recovery, including transfer to the inpatient unit and transfer home for ambulatory patients (Gross et al., 2006). Opioid and sedative drugs also increase the risk across the hospital setting and beyond. Strategies for peri-anesthesia nursing care of patients with OSA are described in detail (Ead, 2009; Moos & Cuddeford, 2006) and are based on recommendations from the American Society of Anesthesiologists (Gross et al., 2006). Nurses who work in other parts of the acute care hospital (medical–surgical units, coronary care units, emergency departments, intensive care units) also interact with patients with sleep apnea and should include interventions to prevent complications.

Nursing care includes careful screening for factors associated with OSA (see Chapter 7, Sleep-Related Breathing Disorders and Table 20.1). Nursing care includes communicating information about OSA to other members of the health care team, especially the anesthesia provider; vigilance in providing support of the airway; supplemental oxygen as needed; judicious use of opioids and sedatives; promoting patient safety; patient and family education regarding avoidance of alcohol, smoking, and sedating medications; and initiating the use of continuous positive airway pressure (CPAP) (Ead, 2009) in patients who require it. The risk of airway collapse may continue for several days or a week after anesthesia because of the possibility of airway edema and occlusion. REM sleep rebound, in which high levels of REM occur after sleep deprivation, is associated with cardiovascular instability and REM-related sleep apnea. Patients with OSA may be at particular risk for cardiovascular complications during the postanesthesia period. Patients with sleep apnea are also at increased risk for atrial and ventricular electrocardiographic abnormalities, hypertension, and hyperglycemia. Case Study 20.2 provides an example of the issues associated with OSA in the acute care environment.

A peri-anesthesia care protocol for patients with sleep apnea was successful in improving compliance among nursing staff in strategies to reduce morbidity and mortality during the pre-anesthesia period and throughout postoperative recovery (Ead, 2009). The authors noted the potential financial implications of longer stay of patients with sleep apnea in the postanesthesia recovery area. However, the financial impact of reduced complications may offset these costs. Given the high societal prevalence of sleep apnea, especially in older adults and those with obesity, cardiovascular problems, and diabetes (who are a large proportion of adult hospital admissions), and the potential negative consequences of sleep apnea, it may be beneficial to implement similar protocols in other acute care patient areas (e.g., coronary care units, intensive care units, emergency departments, medical–surgical units). The clinical effectiveness, including reduction in complications, as well as costs should be evaluated.

CONCLUSIONS

Disordered sleep is common in patients hospitalized in demographical and clinically diverse acute and critical care settings. Careful assessment for factors that increase the risk for sleep disturbance and its consequences during hospitalization is needed. Although randomized clinical trials are sparse, the available evidence suggests the promise of multimodal interventions that reduce environmental stimuli or their impact. Given the high prevalence of OSA in the general population and its underdiagnosis, there is a compelling need for assessment and preventative interventions for this condition. Sleep-promoting strategies are most likely to be effective when institutional support and structured protocols are available. Research is needed on the short- and longer-term outcomes of sleep-promoting interventions on patients' function, quality of life, and morbidity.

CASE STUDY 20.1 Sleep in an Older Adult Admitted to a Medical Surgical Floor

Mr. C.J. is a 78-year-old male admitted to a telemetry floor for admission work up in anticipation of elective coronary artery bypass graft surgery. Mr. C.J. is a retired school administrator. He is 5 feet 9 inches, and weighs 170 lbs. He has a past history of smoking but stopped 30 years ago. He admits getting moderate exercise, mostly in the form of slow walking and gardening. His past medical history is significant for coronary artery disease and recent unstable angina pectoris. Mr. C.J. describes himself in general good health up until the time of the worsening angina. He takes the following medications: low-dose aspirin, nitroglycerin, and metoprolol.

Mr. C.J. lives with his wife; both are retired. They have two married daughters; one lives an hour away and the other lives out of state. Mrs. J. does not drive so Mr. C.J. drives them both to the grocery store, church, doctor appointments, and visits to their grandchildren. During the admission assessment, you note that Mrs. J. is responding to most of the questions concerning her husband's medical history. You administer the "Mini-Cog," a measure of mental status, and Mr. C.J. completes the clock drawing correctly but can only recall one of the three words in the word repetition. Your initial sleep assessment of Mr. C.J. reveals that he admits sleeping 5–6 hours/night. Mrs. J. adds that her husband takes one or two naps each day in front of the television to "catch up" on his sleep. He has no problem falling asleep at bedtime and often has 1–2 glasses of wine with dinner. Mr. C.J. does not routinely take sleeping pills but has recently been taking one Tylenol PM tablet since the cardiac symptoms started. You administer the Epworth Sleepiness Scale, and Mr. C.J. scores "8" with his highest points in the categories of "watching TV" and "sitting and reading."

1. Mr. C.J. is at risk for mild cognitive impairment or delirium following CABG surgery. What monitoring and interventions should the nurse include in Mr. C.J.'s plan of care?
2. Mr. C.J.'s sleep is likely to be disturbed during the hospitalization. What pharmacologic, environmental, and behavioral sleep-promoting strategies should the nurse incorporate during Mr. C.J.s recovery in Cardio-Thoracic ICU? On the telemetry unit upon transfer?
3. What education should the nurse provide to Mr. C.J. and his wife regarding sleep, physical function, and emotional state during his first 6 weeks of recovery from surgery?

CASE STUDY 20.2 Sleep Disturbances in the ICU

Mr. J. is a 63-year-old patient admitted to the ICU following an aorto-femoral bypass graft. He has a 10-year history of OSA and has been successfully managed with the use of nocturnal nasal CPAP since his diagnosis. He had an acute myocardial infarction (AMI) 18 years ago, with stents in the right coronary artery and left anterior descending artery. He has done reasonably well with occasional use of sublingual nitroglycerin for "occasional" chest pain. He stopped smoking at the time of his MI but had a 62-pack-year history at that time. His claudication has become increasingly worse with the inability to walk more than 60 yards without severe leg pain prior to surgery. His home medications include metoprolol, simvastatin, hydrochlorothiazide, aspirin, and clopidogrel. His ECG rhythm in the ICU 1 hour after discharge from the PACU is sinus tachycardia with frequent premature ventricular contractions and ST segment depression in Lead II. He is awake but intubated and on mechanical ventilation with pressure support. He appears to be comfortable and has a patient-controlled analgesic pump with morphine sulfate. His pain is rated as a 3/10. He has an arterial line and a pulmonary artery catheter in place. His pulmonary artery pressures are 40/22, wedge pressure of 15, and cardiac output and index at the lower range of normal. His vital signs are: 180/94-110-18, SpO_2 91%.

1. What special monitoring is needed for Mr. J.?
2. What perioperative management is required given Mr. J's history of OSA?
3. What special care should the nurse anticipate once he is extubated?
4. What sleep promoting strategies should the nurse implement during Mr. J's ICU stay?

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Sleep in Adult Long-Term Care

21

Rebecca A. Lorenz, Melodee Harris, and Kathy C. Richards

The phrase long-term care (LTC) describes a wide range of medical, social, personal, and supportive care needed by people who have chronic illnesses and disabilities that make it impossible to complete self-care activities without assistance (Special Committee on Aging, 2000). Care occurs in a variety of settings including home, daycare, assisted living, and skilled nursing facilities (Special Committee on Aging). For the purposes of this chapter, LTC includes only residential settings (assisted living facilities, continuing care retirement communities, and nursing homes). According to national surveys, there were over 3,000,000 people living in LTC facilities in the United States (American Association of Home and Services for the Aging, 2010; National Center for Health Statistics, 2004), and this number is predicted to rise dramatically along with aging of the population (Special Committee on Aging). People living in LTC settings usually have high levels of cognitive and physical disabilities, with 75% requiring assistance in three or more activities of daily living (ADLs) (Jones, 2002) and 50% having some form of dementia (Sahyoun, Pratt, Lentzner, Dey, & Robinson, 2001).

Older adults living in LTC settings suffer from extremely disturbed sleep associated with many negative consequences including agitation, excessive daytime sleepiness, and accelerated cognitive and functional decline (Martin & Ancoli-Israel, 2008). Numerous factors contribute to poor sleep in these settings. This chapter describes the nature of sleep disturbances in LTC, the scope of concerns relative to sleep disturbances, the consequences of sleep disturbances, sleep-promoting interventions, and implications for research and nursing practice.

SLEEP DISTURBANCE IN LONG-TERM CARE

Poor sleep is a predictor of LTC placement due to increased caregiver burden (Gaugler, Yu, Krichbaum, & Wyman, 2009) associated with nocturnal awakenings. After admission to LTC, about 70% of residents have sleep disturbances, a rate considerably higher than the rate in community-dwelling older adults (Fetveit & Bjorvatn, 2002). Nocturnal sleep disturbances among LTC residents include prolonged sleep onset latency, long wake after sleep onset, frequent awakenings, and low sleep efficiency as measured by actigraphy (Fetveit & Bjorvatn, 2002). As depicted in the conceptual framework, adapted from Sullivan and Richards (2004) (see Figure 21.1), a variety of individual characteristics and environmental factors contribute negatively to the circadian sleep/wake rhythm of LTC residents.

Age-Related Factors

Aging is accompanied by an increasing vulnerability to sleep disturbances for a variety of reasons. As people age, sleep architecture changes (increased light sleep and corresponding reduction in deep sleep) (Ohayon, Carskadon, Guilleminault, & Vitiello, 2004). Deterioration in the suprachiasmatic nucleus in the hypothalamus (Bliwise, 2000) leads to less circadian entrainment and blunts cues to wakefulness

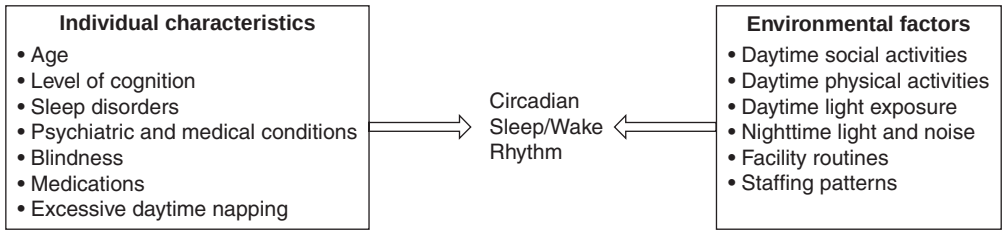

Figure 21.1 ■ *Factors that influence circadian sleep/wake rhythm in older adults. Source:* "Behavioral Symptoms of Dementia: Their Measurement and Intervention. Predictors of Circadian Sleep-Wake Rhythm Maintenance in Elders With Dementia," by S. C. Sullivan and K. C. Richards, 2004, *Aging and Mental Health, 8,* pp. 143–152.

Table 21.1 ■ *Factors that Contribute to Poor Sleep Among LTC Residents*

Age-related Factors

- Sleep architecture changes
- Weakening of the circadian entrainment
- Visual impairment or blindness

Environmental Factors

- High levels of nocturnal noise and light exposure
- Low levels of daytime light exposure
- Facility routines

Behavioral Factors

- Reduced daytime activity and excessive daytime napping
- Social disengagement

Medical and Psychiatric Factors

- Psychiatric/behavioral problems
- Gastrointestinal problems
- Infection
- Pain
- Alzheimer's disease and other forms of dementia
- Insomnia
- Medications
- Symptoms
- Sleep disorders

Sources: Clinical Practice Guidelines: Sleep Disorders in the Long-Term Care Setting (Rep. No. Product Code CPG21) by American Medical Directors Association, 2006, Columbia, MD: AMDA Foundation. "Evidence-Based Recommendations for the Assessment and Management of Sleep Disorders in Older Persons," by H. G. Bloom, I. Ahmed, C. A. Alessi, S. Ancoli-Israel, D. J. Buysse, M. H. Kryger, et al., 2009, *Journal of the American Geriatrics Society, 57,* pp. 761–789. "Sleep-Disordered Breathing and Agitation in Institutionalized Adults With Alzheimer Disease," by P. R. Gehrman, J. L. Martin, T. Shochat, S. Nolan, J. Corey-Bloom, and S. Ancoli-Israel, 2003, *American Journal of Geriatric Psychiatry, 11,* pp. 426–433. "Circadian Rhythms of Agitation in Institutionalized Patients With Alzheimer's Disease," by J. Martin, M. Marler, T. Shochat, and S. Ancoli-Israel, 2000, *Chronobiology International, 17,* pp. 405–418.

and sleeping (Dijk & von Schantz, 2005). Older adults may also have phase advances associated with the tendency to fall asleep earlier in the evening and wake up in the middle of the night or early morning (Loiselle, Means, & Edinger, 2005). Dementia, depression, body temperature, medical comorbidity, and medications can also interrupt the circadian rhythm (Sullivan & Richards, 2004). (See Chapter 2, Developmental Aspects of Sleep). Residents who have significant visual impairment may have free-running circadian rhythms (patterns that are not synchronized to environmental time cues and oscillate on a cycle slightly longer than 24 hours) that lead to recurrent insomnia and daytime sleepiness when the rhythms drift out of phase with the normal 24-hour cycle (Sack, Brandes, Kendall, & Lewy, 2000).

Environmental Factors

The LTC environment is not conducive to sleep due to shared rooms, numerous alarms, the sounds of other patients, low light levels, and hard-surfaced floors that transmit noise (Alessi & Schnelle, 2000). Reduced daytime exposure to light, frequent exposure to light at night, and nighttime staff care practices all contribute to reduced quality of sleep among LTC residents (Fiorentino & Ancoli-Israel, 2006).

Nocturnal Noise and Light Exposure

Nighttime noise and light interruptions occur several times per hour in the LTC setting (Schnelle, Ouslander, Simmons, Alessi, & Gravel, 1993), with 50% of resident awakenings

associated with interruptions due to incontinence care for the resident or a roommate (Cruise, Schnelle, Alessi, Simmons, & Ouslander, 1998; Schnelle et al., 1998). These findings suggest that interventions to reduce nighttime noise may improve nocturnal sleep among LTC residents.

Daytime Light Exposure

LTC residents often lack exposure to bright light during the daytime (Shochat, Martin, Marler, & Ancoli-Israel, 2000). Higher daytime light exposure is associated with reduced nighttime awakenings (Ancoli-Israel et al., 2003; Schnelle et al., 1993), and low daytime light and higher nocturnal light exposure is associated with higher levels of agitation throughout the day and night (Martin, Marler, Shochat, & Ancoli-Israel, 2000). These findings point to the importance of providing LTC residents with normal day/night light patterns.

Facility Routines

Medication administration and nursing tasks may be scheduled for the convenience of staff, as suggested by a study of LTC residents who were dissatisfied with facility routines imposing involuntary adaptation to early rise times and early bedtimes (Middelkoop, Kerkhof, Smildevan den Doel DA, Ligthart, & Kamphuisen, 1994). These care activities may be inconsistent with residents' habitual sleep patterns and interfere with sleep. Poor sleep hygiene can also account for sleep difficulties. Some sleep hygiene rules are listed in Table 21.2.

Table 21.2 ■ *Sleep Hygiene Rules*

Rule	Rationale
Avoid caffeine within 6 hours of bedtime	Promotes wakefulness
Avoid alcohol and nicotine	Promotes sleep fragmentation
Increase physical activity or exercise	Reduces daytime napping and depression
Increase bright light or sunlight exposure	Help maintain circadian rhythm
Avoid napping and excessive time in bed during the daytime	Weakens homeostatic drive to sleep
Maintain a comfortable sleep environment	Promotes sleep
Limit liquids in the evening	Reduces incidence of nocturia
Go to bed when drowsy	Promotes sleep onset
Maintain a regular wake time	Helps maintain circadian rhythm
Avoid heavy meals before bedtime	Reduces gastrointestinal reflux symptoms

Sources: "Diagnosis and Treatment of Sleep Disorders in Older Adults," by S. Ancoli-Israel and L. Ayalon, 2009, *Focus, 7*, pp. 98–105. "Sleep Disruption in Older adults. Harmful and by No Means Inevitable, It Should be Assessed for and Treated," by C. Cole and K. Richards, 2007, *American Journal of Nursing, 107*, pp. 40–49. "Sleep Promotion in Adults," by J. A. Floyd, 1999, *Annual Review of Nursing Research, 17*, pp. 27–56. *The Effects of Slow-Stroke Back Massage on the Sleep of Persons With Dementia in the Nursing Home: A Pilot Study* by M. Harris, 2009, Ph.D. University of Arkansas for Medical Sciences. "Effects of Caffeine on Human Behavior," by A. Smith, 2002, *Food and Chemical Toxicology, 40*, pp. 1243–1255.

Behavioral Factors

Reduced Daytime Activity and Excessive Daytime Napping

Researchers found that 69% of observed residents spent one-third of their day in their rooms, typically in bed, and a full 60% of these also had disturbed nighttime sleep (Martin et al., 2006). LTC residents frequently nap during meals, family visits, and recreational activities (Pat-Horenczyk, Klauber, Shochat, & Ancoli-Israel, 1998). These findings suggest the importance of engaging LTC residents in activities throughout the day.

Social Disengagement

Cognitive impairment, physical limitations, loss of spouse or close friends, and relocation to LTC facilities often lead to loss of social support (Bosse, Aldwin, Levenson, Spiro, & Mroczek, 1993). Residents spend 83% of their time alone and as little as 13% of their time in social interactions (VanHaitsma, Lawton, Kleban, Klapper, & Corn, 1997). Social disengagement contributes to daytime napping and poor nocturnal sleep.

Medical and Psychiatric Factors

LTC residents frequently have multiple chronic conditions that contribute to disruptions in sleep (see Table 21.3) (American Medical Directors Association, 2006; Gentili, Weiner, Kuchibhatil, & Edinger, 1997; Jennum, 2002; Martin, Shochat, & Ancoli-Israel, 2000; Voyer, Verreault, Mengue, & Morin, 2006). Depression may be a risk factor for Alzheimer's disease (Korczyn & Halperin, 2009), as well as sleep disturbance. Anxiety is also a likely contributor to poor sleep in LTC residents, as suggested by the finding that 44% of adults had comorbid anxiety and insomnia (Ohayon & Roth, 2001), but this relationship has not been examined in LTC residents.

Table 21.3 ■ *Chronic Conditions Associated With Poor Sleep in LTC Residents*

Condition	Effect
Alzheimer's disease and dementia	Sleep fragmentation and excessive daytime sleepiness
Delirium	Sleep-wake cycle fragmentation
Arthritis	Sleep onset difficulties and frequent awakenings
Restless legs syndrome	Sleep onset difficulties
Anxiety	Sleep onset difficulties
Depression	Sleep maintenance difficulty, daytime sleepiness
Obesity	Snoring, apnea
Infection	Sleep fragmentation
Pulmonary disorders (asthma and chronic obstructive pulmonary disease)	Awakenings due to coughing and shortness of breath
Gastroesophageal reflux disease	Awakenings due to reflux and heartburn
Congestive Heart Failure	Awakenings due to shortness of breath, nocturia
Nocturia	Awakenings
Diabetes Mellitus	Awakenings

Sources: Clinical Practice Guidelines: Sleep Disorders in the Long-Term Care Setting (Rep. No. Product Code CPG21) by American Medical Directors Association, 2006, Columbia, MD: AMDA Foundation. "Diagnosis and Treatment of Sleep Disorders in Older Adults," by S. Ancoli-Israel and L. Ayalon, 2009, *Focus, 7*, pp. 98–105. "Nocturia and Disturbed Sleep in the Elderly," by D. L. Bliwise, D. J. Foley, M. V. Vitiello, F. P. Ansari, S. Ancoli-Israel, and J. K. Walsh, 2009, *Sleep Medicine, 10*, pp. 540–548. "Evidence-Based Recommendations for the Assessment and Management of Sleep Disorders in Older Persons," by H. G. Bloom, I. Ahmed, C. A. Alessi, S. Ancoli-Israel, D. J. Buysse, M. H. Kryger, et al., *2009, Journal of the American Geriatrics Society, 57*, pp. 761–789. "Sleep-Disordered Breathing and Agitation in Institutionalized Adults With Alzheimer Disease," by P. R. Gehrman, J. L. Martin, T. Shochat, S. Nolan, J. Corey-Bloom, and S. Ancoli-Israel, 2003, *American Journal of Geriatric Psychiatry, 11*, pp. 426–433. *Primary Care Geriatrics: A Case-Based Approach* (4th ed., pp. 437–444) by J. L. Susman, 2001, St. Louis, MO: Mosby.

Dementia

More than 50% of LTC residents have some form of dementia or cognitive impairment (Sahyoun et al., 2001), and one-quarter to one half of these suffer from sleep disruptions (Gaugler et al., 2009). The severity of this problem was documented in a series of studies of demented LTC residents who did not have a single hour consisting of complete sleep or complete wake (Ancoli-Israel, Clopton, Klauber, Fell, & Mason, 1997; Jacobs, Ancoli-Israel, Parker, & Kripke, 1989). Both dementia and sleep disturbance may be due to an imbalance of neurotransmitters (acetylcholine, dopamine, noradrenaline, and serotonin) and cholinergic dysfunction. (See Chapter 1, Physiological & Behavioral Aspects of Sleep.)

Medications

Many prescribed and over-the-counter medications may alter sleep/wake patterns (see Table 21.4) (Neubauer, 2008). Aspirin and NSAIDs delay the onset of sleep, increase the number of awakenings, increase stage 2 sleep, and decrease slow wave sleep and sleep efficiency (Onen, Onen, Courpron, & Dubray, 2005). The effects of NSAIDs are due to the inhibition of prostaglandin synthesis, suppression of nighttime melatonin levels, and changes in body temperature. Medications should be reviewed at routine intervals and adjusted, if possible, to improve sleep.

Symptoms

Symptoms of chronic conditions, such as nocturia, pain, and dyspnea that result from chronic

Table 21.4 ■ *Medications Associated With Sleep Disorders*

Type (Example)	**Effects on sleep**
Diuretics (Furosemide)	Nocturia, especially when given at bedtime
Decongestants (Pseudoephedrine)	Sleep onset difficulties
Antihistamines (Diphenhydramine)	Daytime sleepiness (older varieties)
Antihypertensives (Beta-blockers, alpha blockers)	Insomnia, nightmares, vivid dreams, daytime fatigue
Corticosteroids (Prednisone)	Daytime fatigue, sleep onset difficulties, increase in nighttime awakenings
Bronchodilators (Theophylline, albuterol)	Sleep onset difficulties, increase in nighttime awakenings
Histamine Type 2 receptor antagonists (Cimetidine, rantidine)	Insomnia and excessive daytime sleepiness
Analgesics (nonsteroidal anti-inflammatory drugs, opoids)	Decreased sleep efficiency
Central nervous system stimulants (Modanfinil, caffeine)	Sleep onset difficulties
Stimulating antidepressants (Protriptyline, buproprion, selective serotonin reuptake inhibitors, monoamine oxidase inhibitors)	Reduced REM sleep, shorten total sleep time
Lithium	Daytime sleepiness
Antiparkinsonian drugs (Levodopa/carbidopa (high doses), dopamine agonists)	Insomnia, daytime sleepiness
Antipsychotic drugs (clozapine)	Sedation

Sources: "Sleep in the Elderly: Normal Variations and Common Sleep Disorders," by S. Ancoli-Israel, L. Ayalon, and C. Salzman, 2008, *Harvard Review of Psychiatry, 16,* pp. 279–286. "Medication Effects on Sleep". In *ACCP Sleep Medicine Board Review* (pp. 117–134) by D. N. Neubauer, 2008, Northbrook, IL: American College of Cardiologists. "Pharmacologic Treatment of Disturbed Sleep in the Elderly," by C. Salzman, 2008, *Harvard Review of Psychiatry, 16,* pp. 271–278.

conditions may contribute to sleep disturbances in LTC residents (Bloom et al., 2009; Neikrug & Ancoli-Israel, 2010; Voyer et al., 2006). Of these symptoms, nocturia and pain have been the most frequent topics of study among older adults.

Nocturia and Enuresis

Nocturia and enuresis contribute to interrupted sleep among older adults (Jennum, 2002; Ouslander, 1990), with incontinence affecting about 50% of LTC residents (Durrant & Snape, 2003; Ouslander, 1990). Nocturia, the need to wake during the night to urinate (van Kerrebroeck et al., 2002), leads to sleep fragmentation, daytime sleepiness, mood changes, and cognitive dysfunction (Jennum). Nocturia can be secondary to obstructive sleep apnea (OSA) (Umlauf et al., 2004), Parkinson's disease, and restless legs syndrome (Jennum), among other disorders. Enuresis, urination during sleep, results in sleep disruptions when staff awakens the individual to provide care. Careful assessment and treatment of these conditions may lead to improvements in sleep in LTC residents. Prompted voiding alone and combined with exercise provided every 2 hours improved daytime urinary continence (Fink, Taylor, Tacklind, Rutks, & Wilt, 2008). Unfortunately, additional staff would be necessary to translate this intervention into practice during the 24-hour day (Schnelle et al., 2002), and this would limit its feasibility due to increased costs. Medications, primarily antimuscarinic agents, are widely used by community-dwelling older

adults to improve urge incontinence. These medications have adverse effects including dry mouth, urinary retention, and cognitive impairment. Thus, their use in LTC residents cannot be recommended (Fink et al.). The use of incontinence pads/briefs or catheterization are alternative approaches to urine containment.

Pain

Almost 15% of U.S. LTC residents had persistent pain, and 41.2% of residents who were in pain at first assessment were in severe pain 60–180 days later (Teno, Weitzen, Wetle, & Mor, 2001). There were no differences in pain based on cognitive status, but cognitively impaired residents received less pain medication than those that were cognitively intact (Nygaard & Jarland, 2005; Reynolds, Hanson, DeVellis, Henderson, & Steinhauser, 2008; Wu, Miller, Lapane, Roy, & Mor, 2005). This may be due to the fact that geriatricians and nurses were more skilled at pain assessment in patients with no more than mild-moderate cognitive impairment (Cohen-Mansfield, 2005; Cohen-Mansfield & Lipson, 2002). The co-occurrence of pain, depression, and insomnia (Foley, Ancoli-Israel, Britz, & Walsh, 2004; Scherer et al., 2007) suggest that treatment of pain and depression may improve sleep. Improvements in evaluation and treatment of pain in patients who are cognitively impaired may also improve sleep. However, the effects of pain treatment on sleep remain understudied.

Sleep Disorders

Insomnia

Insomnia is prevalent among adults over 65 years in LTC care (Martin et al., 2006; NIH, 2005) and those with dementia (Bliwise, 2004) with rates varying according to the definition of insomnia (symptom versus syndrome) (see Chapter 6, Insomnia) and the characteristics of the population studied (Voyer et al., 2006). Voyer (2006) found that psychological distress and disruptive behaviors were associated with insomnia (Voyer et al., 2006) and insomnia is associated with excessive daytime sleepiness and nighttime falls. A secondary data analysis of Federal Minimum Data Set (MDS) found that untreated insomnia, but not hypnotic use, was associated with a greater risk of subsequent falls among LTC residents (Avidan et al., 2005). These findings illustrate the importance of identifying and treating insomnia in LTC residents. Given that poor daytime function is associated with use of benzodiazepines among older adults, the observations that nurses did not see any problems with these drugs and routinely administered them without evaluation of their effects (Anthierens, Grypdonck, De, & Christiaens, 2009) are troubling and suggest the need for education and policy changes that address this problem.

Sleep Apnea

Obstructive sleep apnea (OSA) is common among older adults, with rates as high as 70%–80% in residents of LTC who have dementia (Martin & Ancoli-Israel, 2008; Martin, Mory, & Alessi, 2005). (See Chapter 7, Sleep Related Breathing Disorders.) Although nocturnal hypoxemia is associated with cardiovascular disease, hypertension, and mortality in older adults (Hjalmarsen & Hykkerud, 2008), data on LTC residents are lacking. Yet, early studies suggest a strong relationship between dementia and sleep apnea (Ancoli-Israel, Klauber, Butters, Parker, & Kripke, 1991), and more recent work indicates that the apolipoprotein epsilon 4 (APOE epsilon 4) allele increases the risk for OSA (O'Hara, Luzon, Hubbard, & Zeitzer, 2009). Unfortunately, clinicians infrequently screen LTC residents who have dementia for sleep apnea (Resnick & Phillips, 2008). There is a pressing need for greater awareness of this problem and incorporation of screening and treatment. Treatment of sleep apnea with CPAP (see Chapter 7, Sleep Disordered Breathing) may improve the adverse consequences of sleep, such as daytime agitation (Gehrman et al., 2003), hypersomnolence, stroke, cognitive impairment (Kalra, Richards, & Kleban, 2009), and physical decline (Cole et al., 2009; Martin & Ancoli-Israel, 2008), among others, in older-adults. However, these effects are understudied in this population.

Periodic Limb Movement Disorder and Restless Legs Syndrome

Periodic limb movement disorder (PLMD), a condition associated with involuntary movements of the limbs during sleep, and restless legs syndrome (RLS), a condition associated with discomfort in the limbs and overwhelming desire to move while awake, are common among older adults (See Chapter 8, Movement Disorders.) These conditions seem to be especially prevalent among people who have dementia and may partially explain poor sleep (Richards et al., 2008; Richards, Shue, Beck, Lambert, & Bliwise, 2010; Rose et al., in press). Wandering and restlessness, particularly in the early evening, are associated with RLS, as well as dementia. Other factors that may contribute to RLS include selective serotonin reuptake inhibitors (SSRIs), selective norephinephine reuptake inhibitors (SNRIs), and chronic conditions such as arthritis (Bliwise, 2006). The observation that LTC residents who have dementia are unable to respond to the RLS diagnostic interview, the gold standard for diagnosis of RLS (Allen et al., 2003), suggests the importance of objective assessments, such as anterior tibialis EMG monitoring of leg movements (Richards et al., 2010).

Given evidence that sleep disorders are underdiagnosed and undertreated in LTC, it is apparent that there is a need for comprehensive assessment of risk factors for common sleep disorders. Interventions that focus on reducing risk factors as well as directly treating sleep disorders may improve outcomes for this vulnerable group of residents.

THE CONSEQUENCES OF SLEEP DISTURBANCES IN LTC

Poor sleep is believed to contribute to excessive daytime sleepiness, irritability, poor concentration and memory, slower reaction time, decreased cognitive and physical performance (Martin & Ancoli-Israel, 2008), and mortality (Dale, Burns, Panter, & Morris, 2001) among LTC residents, and these observations are supported by a large body of evidence obtained in the general population and older adults. (See Chapter 4, The Nature of Sleep Disorders and their Consequences.) Unfortunately, the consequences of sleep disturbances among LTC residents have not been well-studied (Cochen et al., 2009).

SCIENTIFIC EVIDENCE FOR SLEEP-PROMOTION INTERVENTIONS

Beginning with observational studies of sleep disruptions conducted in the 1980s and 1990s among LTC residents with cognitive impairment, researchers have continued to conduct studies, including some clinical trials of pharmacological and nonpharmacological interventions to promote sleep in LTC settings. Finding effective treatments that significantly improve sleep with the fewest consequences has proven to be challenging, and the evidence remains limited.

Environmental Interventions

Noise Reduction

A detailed discussion of the characteristics of noise that may have an impact on sleep among LTC residents is provided in Chapter 20, Sleep in Adult Acute Care Settings. Similar to findings in acute care settings, the use of noise abatement as a singular intervention in the LTC is ineffective (Schnelle, Alessi, Al-Samarrai, Fricker, & Ouslander, 1999).

Bright Light Exposure

Bright light exposure is one of the most frequently studied treatments for sleep disturbance (Ancoli-Israel, Martin, Kripke, Marler, & Klauber, 2002; Ancoli-Israel et al., 2003; Fetveit & Bjorvatn, 2004; Fetveit, Skjerve, & Bjorvatn, 2003). Unfortunately, results have been inconsistent, possibly due to the wide variability in light type and intensity used, and levels of dementia among participants (Koch, Haesler, Tiziani, & Wilson, 2006). Systematic reviews have concluded that there is insufficient evidence to recommend the use of light therapy to treat cognitive function, sleep, or behavior disturbances (Forbes et al., 2009; Koch et al., 2006; Skjerve, Bjorvatn, & Holsten, 2004).

Facility Routines

Modifying the scheduling for activities, mealtimes, and bedtimes to facilitate sleep and stimulate more robust circadian rhythmicity may be useful, but studies found that modifying these activities by providing increased resident control (Matthews, Farrell, & Blackmore, 1996) and use of a modified prompted voiding protocol (Ouslander, Ai-Samarrai, & Schnelle, 2001) were not efficacious.

Behavioral Interventions

Physical Activities

The positive effect of regular physical activity on sleep is a generally accepted truth. However, studies of the effects of a variety of low-intensity aerobic programs, like walking or wheelchair propulsion, among LTC residents with dementia have provided mixed results. For example, Alessi et al. (1995) found no differences in nighttime sleep using actigraphy. In contrast, Buettner and Fitzsimmons (2002) and Namazi, Zadorozny, and Gwinnup (2009) found significant improvements in sleep, reduction in restlessness, and higher levels of social engagement using staff ratings.

Studies of anaerobic exercise using progressive resistance training, such as bench press and leg press, found significantly improved sleep quality as measured by the Pittsburg Sleep Quality Index (PSQI); unfortunately, this improvement was not sustained 6 months after the program ended (Ferris, Williams, Shen, O'Keefe, & Hale, 2005). These findings indicate that exercise must be continued to maintain the positive effects on sleep. Additional research is warranted because the differences in the exercise protocols studied (aerobic or anaerobic, intensity, duration) and the lack of objective measures of sleep make it difficult to determine the most effective exercise program.

Social Activities

Individualized social activity led to decreases in daytime napping and significant improvements in nighttime sleep, reduced sleep latency, and less daytime sleepiness, as detected by actigraphy, in LTC residents who had dementia (Richards, Sullivan, Phillips, Beck, & Overton-McCoy, 2001; Richards, Beck, O'Sullivan, & Shue, 2005). These findings highlight the relevance of reductions in social isolation to sleep in LTC residents.

Combined Physical and Social Activities

Interventions that combine physical and social activity hold promise as sleep promotion strategies in LTC (Chen et al., 2007; Landi, Russo, & Bernabei, 2004). For example, Naylor et al. (2000) found increased slow-wave sleep and improvements in memory-orientated tasks without any change in body temperature, vigor, and mood after 2 weeks of daily low-intensity physical and social activities. More recently, Richards et al. (2011) reported significantly increased nocturnal sleep time, sleep efficiency, and non-REM sleep in a group that had combined high-intensity progressive strength training, walking, and social activity, compared with social activity alone, progressive strength training alone, or usual care.

Multicomponent Interventions

Several studies have examined multicomponent nonpharmacological interventions addressing both environmental and lifestyle factors known to affect sleep with mixed results (Alessi et al., 2005; Martin, Marler, Harker, Josephson, & Alessi, 2007; Ouslander et al., 2006). Although daytime sleep was reduced in all studies, no more than modest improvement in nocturnal sleep was achieved (Alessi et al., 2005; Ouslander et al., 2006). Two recent systematic reviews of studies conducted in LTC settings, however, found that multidisciplinary strategies such as combining a reduction in environmental noise, reduction of nighttime nursing care, and promotion of daytime activity to be the most effective intervention to promote sleep (Koch, Haesler, Tiziani, & Wilson, 2006; Haesler, 2004).

Complementary and Alternative Treatments

The benefits of complementary and alternative medicine (CAM) treatments including aromatherapy and herbal agents (valerian extract and

melatonin) are unproven (Moquin, Blackman, Mitty, & Flores, 2009). Melatonin was no more efficacious than placebo in improving sleep (Singer et al., 2003) and does not have established efficacy for treating insomnia (Gooneratne, 2008). (See Chapter 15, Complementary and Alternative Therapy for Sleep Disorders). At this time, it is unclear whether valerian and melatonin pose risks to LTC residents due to potential drug interactions (Shimazaki & Martin, 2007). Therefore, these substances cannot be recommended for use in this population at this time.

Massage and Acupressure

Research on alternative therapies, such as massage and acupressure, is sparse. Studies testing acupressure and acupuncture suggest a significant improvement in self-reported sleep (Matsumoto & Terasawa, 2001; Sun, Sung, Huang, Cheng, & Lin, 2010), possibly by reducing pain (Matsumoto & Terasawa). Harris (2010) found improvements in total nocturnal sleep time in a recent study of a 3-minute slow-stroke back massage in persons with dementia in LTC. Although the findings were not statistically significant and there were environmental factors that contributed to difficulty administering the intervention, these findings suggest that slow-stroke back massage may be efficacious. Further study is needed.

Sleep-Promoting Medications

Researchers have reported utilization of sleep-promoting medications, such as psychotropic drugs, including benzodiazepines, to be as high as 80% (Conn & Madan, 2006) among LTC residents. These medications, however, have not been found to improve nighttime sleep (Alessi, Schnelle, Traub, & Ouslander, 1995; Greco, Deaton, Kutner, Schnelle, & Ouslander, 2004; Simpson, Richards, Enderlin, O'Sullivan, & Koehn, 2006), and use of temazepam was associated with impaired neurological function and excessive daytime sleepiness (Glass, Sproule, Herrmann, & Busto, 2008). Therefore, the use of medications does not appear to confer benefits and may worsen function.

Treatment of Primary Sleep Disorders

Obstructive Sleep Apnea

The gold standard for treatment of obstructive sleep apnea is Continuous Positive Airway Pressure (CPAP). However, compliance in individuals with mild Alzheimer's disease living at home was poor (Ayalon et al., 2006). In one observational study of LTC residents who chose to continue to use CPAP after moving into a LTC residence, researchers concluded that CPAP should be the treatment of choice and residing in LTC should not preclude its use (Gehrman et al., 2003). Despite the high prevalence of obstructive sleep apnea in older people with dementia, CPAP use has never been tested in the LTC environment (Neikrug & Ancoli-Israel, 2010).

Movement Disorders

There are two FDA approved agents for the treatment of RLS: ropinerole and pramipexole. Neither of these medications have been studied in LTC residents (Martin & Ancoli-Israel, 2008).

IMPLICATIONS FOR FUTURE RESEARCH

Intervention studies provide evidence that assessment and management of sleep disturbances among LTC residents must be comprehensive and address the multiple factors that interfere with sleep. Unfortunately, there is little evidence on the safety and effectiveness of sleep-promoting medications and the specific management of sleep disorders in this setting (Alessi & Schnelle, 2000; Neikrug & Ancoli-Israel, 2010). Priorities for future research should include testing the efficacy and effectiveness of environmental changes, physical and social activities, and CAM methods, such as massage and acupuncture on sleep.

RECOMMENDATIONS FOR PRACTICE

Assessment of sleep is the essential first step to addressing sleep disturbances among LTC residents (see Table 21.5). Validated rating scales of sleep symptoms are available. For example, The Sleep Disorders Inventory was developed and validated for patients with dementia and

Table 21.5 ■ *Implications for Nursing Practice*

- Obtain a sleep history on all new LTC residents
- Carefully review the medical history, including medications for risk factors and indicators of sleep problems
- Practice patient-centered care
- Advocate for referral to a sleep disorders center when necessary
- Adequately treat pain and other chronic disease symptoms
- Routinely assess sleep
- Provide multi modal interventions, especially those that increase activity and social interaction: environmental changes alone are likely not sufficient

Table 21.6 ■ *Symptoms of Possible Sleep Disorders in LTC Residents*

Daytime

- Excessive daytime sleepiness and/or napping
- Agitation or irritability
- Reduced cognition: problems with concentration, memory, and attention
- Increased functional decline: falls, difficulty with activities of daily living

Nighttime

- Awakenings, possibly associated with wandering and falls
- Snoring, possibly associated with disordered breathing (characterized by apnea)
- Periodic movements of legs when sleeping or lying in bed awake
- Periodic apneic periods; could be associated with sleep position

Sources: Clinical Practice Guidelines: Sleep Disorders in the Long-Term Care Setting (Rep. No. Product Code CPG21) by American Medical Directors Association, 2006, Columbia, MD: AMDA Foundation. "Evidence-Based Recommendations for the Assessment and Management of Sleep Disorders in Older Persons," by H. G. Bloom, I. Ahmed, C. A. Alessi, S. Ancoli-Israel, D. J. Buysse, M. H. Kryger, et al., 2009, *Journal of the American Geriatrics Society*, *57*, pp. 761–789. "Sleep-Disordered Breathing and Agitation in Institutionalized Adults With Alzheimer Disease," by P. R. Gehrman, J. L. Martin, T. Shochat, S. Nolan, J. Corey-Bloom, and S. Ancoli-Israel, 2003, *American Journal of Geriatric Psychiatry*, *11*, pp. 426–433. "Circadian Rhythms of Agitation in Institutionalized Patients With Alzheimer's Disease," by J. Martin, M. Marler, T. Shochat, and S. Ancoli-Israel, 2000, *Chronobiology International*, *17*, pp. 405–418.

has been used in the LTC setting (Tractenberg, Singer, Cummings, & Thal, 2003). Residents with nighttime sleep disturbances should be assessed for symptoms of sleep disorders (see Table 21.6). Referral for polysomnography is indicated if a primary sleep disorder such as sleep apnea is suspected when etiology is unclear (Bloom et al., 2009).

Research suggests that interventions using singular approaches, such as reducing nighttime noise, have had little effect on sleep. Therefore, multidimensional interventions that include environmental and patient-related interventions are more likely to be useful. Disruptive nighttime activities, such as noise, should be avoided as they not only disrupt sleep but also creates a stress response so that residents become more alert and vigilant (Michaud et al., 2006). Although modifying light as a singular intervention was not effective, assuring adequate exposure to daylight during the day and dim lighting at night may contribute to sleep improvements. Although the effectiveness of sleep hygiene remains unproven (Stepanski & Wyatt, 2003), it has been recommended as usual care for insomnia in community-dwelling older adults (Kamel & Gammack, 2006); and may be useful as a first step in managing disturbances in sleep (see Table 21.2). Relaxation interventions, such as massage therapy, may be effective for improving many features of poor sleep, particularly sleep initiation. Promoting exercise and increased social interaction also hold promise as sleep-promoting interventions. Medication review should occur on a routine basis to identify those that may interfere with sleep and adjusted if possible. Residents should be referred for a sleep study if a sleep disorder is suspected. If sleep apnea is diagnosed, CPAP is the most efficacious treatment and should be used in the LTC setting.

Finally, nurses play a key role in developing nursing practice and policies to ensure that nursing students develop an awareness of the importance of sleep and become skilled at assessing and promoting sleep in the LTC care setting. More information on curriculum and learning experiences is provided in Chapter 24. Helpful Web sites are listed in Table 21.7. Nursing students need a theoretical basis for evidence-based practices to know how to manage

Table 21.7 ■ *Helpful Websites*

■ www.aasmnet.org/medsleep.aspx	Educational resources designed to help increase the sleep-related knowledge and skills of students, staff, and community health care providers
■ www.amda.com/tools/guidelines.cfm	American Medical Directors Association provides Clinical Practice Guidelines specifically for use in long-term care settings
■ www.commonwealthfund.org/Content/Publications/Other/2006/Apr/Transforming-Long-Term-Care--Giving-Residents-a-Place-to-Call--Home.aspx	Provides information including a video on the "resident-centered care" movement in nursing homes
■ http://content.healthaffairs.org/cgi/content/abstract/29/2/312	Koren, M.J. (2010). Person-Centered Care For Nursing Home Residents: The Culture-Change Movement. *Health Affairs 29(2). 312–317*
■ www.aacn.nche.edu/Education/gercomp.htm	Provides recommended Baccalaureate Competencies and Curricular Guidelines for Geriatric Nuusing Care
■ www.guideline.gov	A link to the National Guideline Clearinghouse, a resource for evidence-based clinical pratice guidelines
■ http://consultgerin.org	An evidence-based practice and continuing education resource; host of the "Try This" series
■ http://wwwgeriatricpain.org	A resource for teaching geriatric pain assessment and management in LTC
■ www.hbo.com/alzheimers/caregivers.html	A link to the HBO Series: The Alzheimer's Project

older adults in LTC. Nursing students also need clinical experience in an Accredited Sleep Disorders Center and LTC settings in order to learn how to take a history, make referrals, and provide pharmacological and nonpharmacological interventions that emphasize sleep among LTC residents (Lee et al., 2004).

SUMMARY

Sleep is critically important in LTC residents. Nurses play a key role in identifying and assessing for sleep disorders, devising interventions to improve sleep, and determining when referrals to sleep specialists are required. Because the nurse's personal beliefs about the importance of sleep influence practice (Spenceley, 1993), educational programs about the importance of sleep, the consequences of poor sleep, and evidence-based practice to promote sleep in the LTC environment are critical in supporting the implementation and continuance of practice patterns to address sleep problems and promote positive sleep outcomes.

CASE STUDY

MS is an 83-year-old Caucasian widow with advanced dementia admitted 12 days ago to a long-term care facility. She is aphasic, and the nursing staff anticipates most of her needs. In spite of repeated attempts by nursing staff to help her into bed, she may pace or wander the halls in the middle of the night, causing lower extremity edema. These nighttime behaviors result in tearfulness, agitation, generalized restlessness, and unsuccessful attempts by the nursing staff to redirect behaviors. MS frequently crosses her legs and rubs her lower extremities. The family is experiencing guilt over nursing home placement and requests a "sleeping pill" or "something to calm her down so we can take her home." During the night, lights are left on in the hallway as a "safety precaution" and she often drinks coffee with the nursing staff if she cannot sleep.

(*Continued*)

CASE STUDY *Continued*

Although MS is unable to verbalize lower extremity pain due to the severity of her dementia, her tearfulness, pacing, wandering, generalized restlessness, repeatedly crossing her legs, and rubbing her lower extremities are symptoms of RLS. MS should be screened for anemia and treated appropriately with ferrous sulfate and vitamin C, which may relieve painful symptoms. The nursing staff should inquire about her usual bedtime, individualize her nighttime routine, and monitor for adjustment to the facility. MS can engage in individualized social activities during the day that allow her to elevate her lower extremities. MS has a chart history of previous pramipexole and clonazepam use that was discontinued shortly after a hospital admission. Conservative pharmacological treatment may be considered if symptoms do not resolve.

Critical thinking questions – Discuss the assessment and nursing interventions for Mrs. MS.

1. What are the differential diagnoses for MS? Answer: Table 21.3
2. What is the first step to assess MS? Answer: Table 21.5
3. Why is it important to know that MS is constantly moving, rubbing her legs, and crossing her legs? Answer: Table 21.6
4. As a charge nurse, what factors can be identified for potential policy changes at the facility? Answer: Table 21.1 and Table 21.2

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Sleep Promotion in Occupational Health Settings

22

Jeanne Geiger-Brown and Kathleen M. McPhaul

Most adults spend a significant number of their waking hours at work. The work relatedness of employees' sleep is not always obvious to employers, as sleep is usually a private behavior. Yet there is much about how work is organized that influences the opportunity to sleep, the quality of sleep that is achieved, and the risk for sleep disorders. Circadian rhythm disruptions influence sleep when work schedules include very early start times, night shift work, or shift rotation. Reduced sleep opportunity from long working hours, shift overruns and overtime, long commutes, and being called in during time off may cause sleep deprivation. The physical surroundings of the job (light, noise) can increase or inhibit alertness, and over time can alter circadian rhythms. When work is physically or psychologically stressful, it can inhibit sleep by increasing sympathetic nervous system activity that is incompatible with restful sleep. Certain occupational groups (health care, transportation, public safety, food service, mining, construction, executive travel) are at particular risk for impaired sleep because of work stress and the scheduling of work hours. Because nurses care for workers throughout the life span in all health care settings, the nursing curriculum must teach the basics of sleep to entry-level nurses, nurse practitioners, and occupational health nurses (OHNs). (See Chapter 24, Future Directions in Sleep Promotion: Nursing Practice, Research, and Education.) This chapter discusses the work-related impediments to sleep and interventions to improve sleep, with implications for health promotion and occupational health programs in the workplace.

The consequences of acute and chronic sleep deprivation for workers are well documented. Workplace injuries and accidents are more frequent, causing pain and suffering, as well as lost productivity for the worker who is sleep deprived. Frequent or high cost claims can lead to higher costs to the employer for health benefits. Chronic sleep deprivation increases the risk for cardiovascular diseases such as hypertension, stroke, and heart disease, as well as metabolic disorders such as obesity and diabetes. These work-related health hazards can be addressed with active health promotion and occupational health programs and practices that minimize serious adverse outcomes.

Sleep promotion is ideally a shared responsibility of workers, their employers, and health care providers. Workers themselves must consider the priority they place on sleep when competing demands threaten to derail a healthy lifestyle and performance at work. They must also be aware when their sleep is abnormal, seek treatment, and adhere to treatment recommendations if a sleep disorder is detected. Employers who are trying to create a healthy work environment must have a systematic plan at all levels of the organization to recognize sleep-related aspects of the physical work environment, the intensity of workplace stressors, and how work is organized to advantage workers' sleep. They must provide health insurance coverage to ensure that workers receive specialty treatment for their sleep disorder-related symptoms and provide accommodations if chronic sleep disorders continue to impair functioning.

The employee health unit is the best place to coordinate the health promotion activities at work as well as screening, clinical care, referrals, and accommodation. The personnel in the employee health and/or safety departments should conduct exposure assessments of scheduling practices and monitor trends in injuries to inform healthy scheduling practices. When the exposure assessment identifies possible risk factors for sleep deprivation or sleep disorders, the occupational health nurse clinicians must incorporate thorough sleep and occupational exposure histories, provide health education regarding sleep and work, and tailor interventions to improve sleep quantity and quality. The health care providers in the employee health department can also recognize sentinel occupational health events, such as sleep complaints, drowsiness at work, and accidents and injuries which might indicate additional workers at risk for occupational sleep disorders (Figure 22.1). In the ideal situation, all are motivated to create a healthy workplace where workers can be safe and productive and then go home, sleep restfully and long enough, and enjoy a high quality of life. Combined, these concerns clearly have implications for workplace policy development to ensure worker safety and productivity.

WORK-RELATED IMPEDIMENTS TO SLEEP

Both work and nonwork factors influence sleep opportunity and the risk for sleep disorders (see Figure 22.2). The work-related impediments to achieving adequate sleep duration and good sleep quality are described below.

Early Day Shift Start Times

When work is scheduled to begin early in the morning (before 9 AM), workers achieve significantly less sleep, and have reduced alertness and increased sleepiness on the job when compared to those working more normal daytime hours (Ingre, Kecklund, Akerstedt, & Kecklund, 2004; Rosa, Harma, Pulli, Mulder, & Nasman, 1996; Tucker, Smith, Macdonald, & Folkard, 1998). These early shifts are common in transportation, mining, construction, and health care (U.S. Bureau of Labor Statistics, American Time Use Survey, 2010). Even with normal start times at work, commuters in some urban areas leave the house early to "beat the rush" (National Academies, 2006). When work must be scheduled in

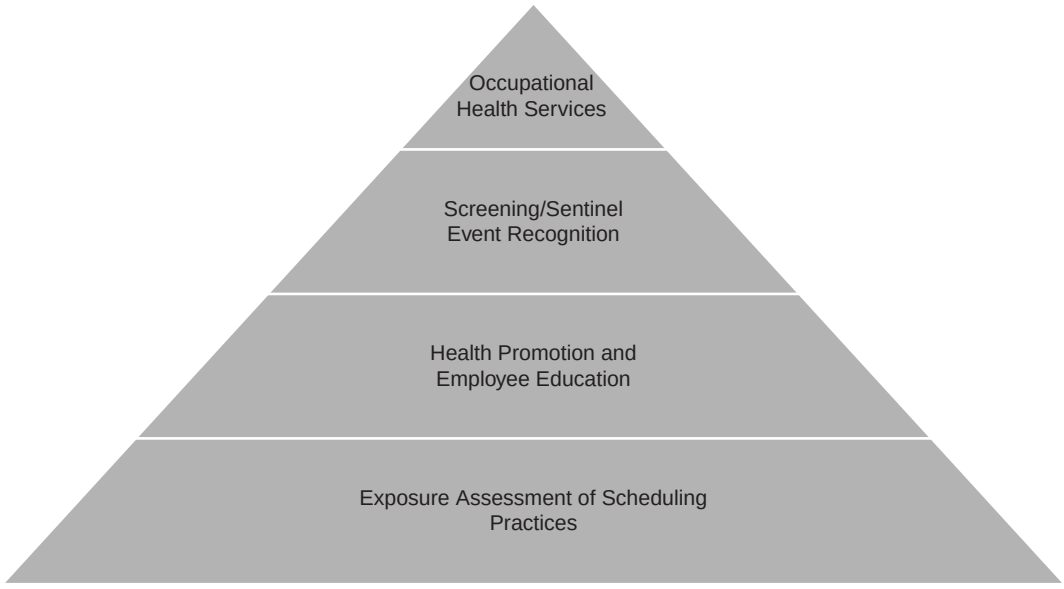

Figure 22.1 ■ Employee health services model to prevent work-related sleep disorders.

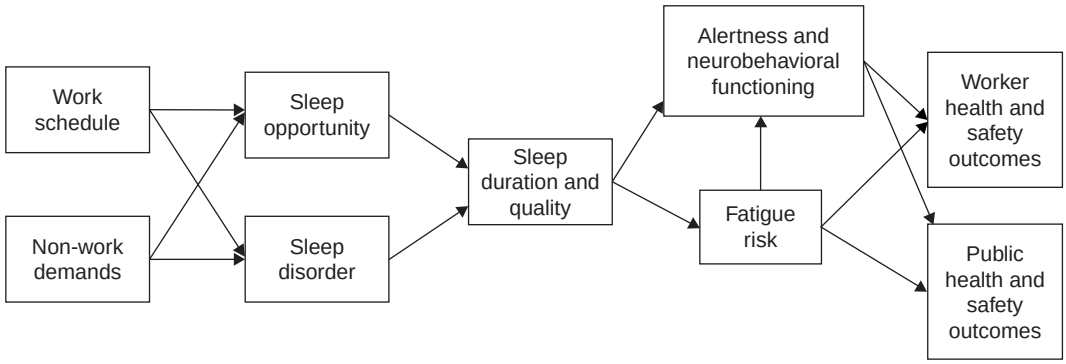

Figure 22.2 ■ Work and non-work factors related to sleep and outcomes.

the early morning, it is beneficial to offer early-shift workers some interspersed later start time shifts to allow them time for recovery sleep. Altering start times to improve sleep may be particularly important for some worker subgroups such as adolescents and young adults, where falling asleep before midnight and arising early in the morning is difficult (see Table 22.1) (Crowley, Acebo & Carskadon, 2007). In contrast, older workers whose circadian cycle has shifted to an early bedtime and wake time may more easily adapt to an early shift (see Table 22.1) (Dilk, Duffy, & Czeisler, 2000).

Shift Length

Extended work hours have become normal practice in some service occupations such as health care, where 12-hour shifts for nurses or 24 hour shifts for physicians are common. Other industries, such as utility companies or those responsible for responding to disasters intermittently have extended hours. Extended work hours reduce the opportunity for sleep, are related to short total sleep time, and result in cumulative sleep deprivation (Ferrie et al., 2007; Geiger-Brown, Rogers, Trinkoff, Bausell, & Scharf, 2010; Lockley et al., 2007).

Table 22.1 ■ *Common Work-Related Circadian Rhythm Sleep Disorders*

Delayed Sleep Phase Disorder
The worker cannot fall asleep at a desired sleep time, often falling asleep after midnight, and then has severe difficulty waking early in the morning to go to work. If sleep is undisturbed, the duration of sleep will be normal. The worker may describe himself as a "night owl." More common in adolescents and young adults, although may begin in childhood and persist into late life.
Advanced Sleep Phase Disorder
The worker gets sleepy several hours before conventional bedtimes of other adults, and wakes spontaneously very early in the morning. If sleep is undisturbed, the duration of sleep will be normal. The worker may describe himself as a "morning person." More common in older adults.
Shift Work Disorder
The worker is unable to sleep or sleeps poorly during the daytime when on the night shift. He is excessively sleepy during the work shift, and may have reduced or unsafe work performance. The duration of sleep will not be normal if sleep is undisturbed, but will be considerably shorter than normal. This occurs even if the worker is in an optimal sleep environment. The worker may describe himself as "unable to adjust to night shift, even though I tried."
Jet Lag Disorder
The worker who travels across time zones will have difficulty falling asleep at the appropriate clock time in the new environment, and may be sleepy during the daytime, with complaints of gastrointestinal upset and malaise. The severity and duration of symptoms depends on the number and direction of time zones crossed (eastward travel is more difficult), as well as individual tolerance to chronobiologic changes. The worker may describe himself as having "jet lag."

Night Shift Work

Shift work is an unavoidable consequence of around-the-clock operations in many industries, and 15%–20% of the working population work some night shift hours (Barger, Lockley, Rajaratnam, & Landrigan, 2009; Morganthaler et al., 2007). Night shift work may be mandated or voluntary, intermittent (as with rotating shifts) or permanent. However it occurs, studies demonstrate that night shift workers achieve about 10 hours less sleep per week than those working on the evening or day shift (Akerstedt, 2003). Being awake during daylight and sleeping in the dark of night is biologically hard-wired into humans, so adaptation to night shift is a continual and hard-fought battle (Akerstedt, 2007). The circadian rhythm regulating sleep becomes disrupted, and sometime desynchronized. Although most workers can adapt biologically to working at night, the circadian system is slow to resynchronize (Barnes, Deacon, Forbes, & Arendt, 1998), and when workers revert back to sleeping at night on days off, adaptation is reversed. (See Chapter 10, Circadian Rhythm Disorders.)

Domestic factors also influence ability to cope with shift work. If the sleep environment were perfect (noiseless, dark), night shift workers would still get less sleep because of the potent circadian influence, but ideal sleep environments are rare. It is common for night shift workers to be awakened by daytime environmental noise, for childcare needs and social obligations, and by daylight exposure encouraging wakefulness (see case example in Table 22.2).

Night shift is associated with a myriad of physical symptoms and disorders. Workers have gastrointestinal complaints ranging from dyspepsia to irritable bowel disorders; depressive symptoms, incipient mood disorders and exacerbation of existing mood disorders (see Chapter 13, Pediatric Sleep Disorders); higher risk for cardiovascular diseases such as hypertension, health disease and stroke and their metabolic precursors; and higher risk for some cancers, most notably breast cancer. Chapter 10, Circadian Rhythm Disorders, provides additional information about the circadian physiologic changes that are relevant to these disease processes.

Table 22.2 ■ *Case Example: Shift Worker*

A night shift worker arrives for her 12-hour shift at 7 PM. She has to fight sleep between 3 and 5 AM when the circadian nadir won't sustain alertness, but by the time she leaves work at 7:30 AM and arrives home at 8 AM, waking pressure from the circadian drive is already beginning to trigger wakefulness. Although she falls asleep easily upon arriving home (due to homeostatic pressure to sleep), by 2 PM there is so much circadian waking pressure that she finds she cannot remain asleep despite not having had enough sleep and desiring more. By 3 o'clock the children are home from school and clamoring for attention, and the worker must start dinner by 4:30 PM in order to feed the family so that the children's evening sports team practices can start on time. She returns to work the next night at 7 PM having had only 5 hours of sleep, and this cycle repeats between each night shift until the weekend when she sleeps 10 hours to "catch up" from chronic partial sleep deprivation. She is unaware that long sleep on the weekend is insufficient to counter the sleep debt that she has accumulated during the week, and does not realize that her early symptoms of hypertension and weight gain might be due, in part, to chronic sleep deprivation.
After a "near miss" safety incident at her job, the employee was referred to employee health per protocol for a urine drug screening. While her urine test was negative for substances, the occupational health nurse specialist conducted a thorough occupational history and sleep history learning that the employee was exposed to multiple 12 hour shifts, often 3 or 4 in a row without a day off. The accident occurred on the 4th straight 12-hour shift. The client's insufficient sleep together with the safety incident prompted a review of scheduling practices at the facility. The worker was referred to a sleep specialist who crafted specific scheduling requirements for the worker upon return to work. Coordination with the employee's primary care practitioner was also initiated. The OHN and nurse practitioner in the employee health unit communicated the work schedule requirements to the supervisor. At the next safety meeting the OHN raised the issue of unsafe scheduling practices. It was decided that night shift workers would be limited to two consecutive 12-hour shifts before a day off. A surveillance program for occupational injuries secondary to repeated 12-hour shifts or overtime was implemented by the occupational health nurse.

Shift Rotation

Rotating shifts (between day and night, or day, evening, and night) are difficult from a circadian perspective. Both the speed and direction of rotation affect sleep duration and quality, although few full time night shift workers (< 3%) ever really show circadian adjustment to their shift (Folkard, 2008). Based on these findings, it is unlikely that rotating shift workers can show circadian adaptation over relatively short periods. However, permanent night workers achieve slightly more sleep than workers who rotate to night shift (Pilcher, Lambert, & Huffcutt, 2000), perhaps because they make accommodations over time to achieve more sleep. Night shift workers often persist in choosing this schedule because of additional shift differential pay that significantly boosts their income (Camerino et al., 2008), the nature of the work itself (fewer "bosses"), or the need to accommodate caregiving responsibilities for a child or an aging parent. The most hazardous aspect of rotating shifts is the "quick return," a pattern where the worker has fewer than 10 hours off between shifts.

Work Stress, Spillover

Many workers spend 40–50 hours a week at their jobs, and some workers spend even more hours at their jobs. At any job there are sources of stress that can carry over into time away from the job as well. As such, there is mounting epidemiologic evidence that physical and mental work demands may be a contributing factor to difficulty falling asleep, awakening in the middle of sleep, and early morning waking, all of which can affect total sleep time (Akerstedt, 2006; Ferrie et al., 2007; Jansson & Linton, 2006). (See Chapter 6, Insomnia.) Physical demands over a long work day can increase adrenergic hormones that prevent sleep (Sonnentag & Zijlstra, 2006). High levels of mental stimulation in a stressful work environment can also impair sleep, especially if the worker anticipates problems during the next work shift (Akerstedt, 2006; Geiger-Brown, et al., 2004; Jansson & Linton, 2006; Sonnentag & Zijlstra, 2006).

In animal models, different forms of stress produced unique sleep architecture changes (Cui, Binglin, Suemaru, & Araki, 2008), with social conflict increasing the amount of NREM sleep, conditioned fear decreasing REM sleep, and novelty stress decreasing both NREM and REM sleep. In a nationally representative survey of more than 1,700 U.S. full-time workers, Knudsen, Ducharme, and Roman (2007) assessed the number of past-month days of difficulty falling asleep, maintaining sleep, and nonrestorative sleep. They found that work overload reduced the quality of sleep in all three aspects, where role conflict was associated with sleep initiation and restorative sleep. Repetitive tasks increased the number of days of difficulty falling asleep and maintaining sleep. Thus, occupational health nurses need to understand not only the type of work that the worker performs, aspects of the work schedule, but also the perceived stressors at work in order to fully appreciate the contribution of work to impaired sleep (Table 22.3).

WORK-RELATED SLEEP DISORDERS

Shift Work Sleep Disorder

Nearly all shift workers have some sleepiness while working the night shift, and most have difficulty achieving adequate sleep during the day although sleep education for shift workers can help to reduce these sleep problems

Table 22.3 ■ *Elements of the Occupational Health/Sleep History*

■ Occupation
■ Typical work schedule include shift, (day, evening, night) length of shift, and whether employee is required to rotate
■ Scheduling policies of employer; ability to request and work desired schedule
■ Total numbers of hours worked per shift, week and whether overtime hours are paid or unpaid
■ Total commute time, work stressors (pace, conflict, demands, flexibility, decision latitude, support), Family obligation schedule
■ Sleep disorder symptoms
■ Medical history and medication use

Table 22.4 ■ *Optimizing Sleep When Working the Night Shift*

While at work:

- Drink a caffeinated beverage early in your shift to help maintain alertness during the shift, but avoid caffeine after midnight to reduce fragmented sleep during the daytime after the shift is over.
- Do not succumb to the temptation to eat to remain awake, especially unhealthful snack foods. If you are hungry have a small protein-rich meal early in your shift.
- If you feel drowsy during your shift, go to a more brightly lit area, move around, have a conversation.
- Avoid tasks that require concentration between 3 and 5 AM or "buddy" with a coworker to ensure accuracy and safety.
- Take a nap break if possible; one 20-minute nap may refresh you and can help you to drive home safer. Your employer is obligated to provide and you are entitled to take breaks at work by Federal law. A break is a period of time when others are responsible for your job, not just a time when you get to sit down but are still "on duty." If you choose to nap during your break, make sure that you have a way to wake up at the desired time.
- If you feel too drowsy to drive home at the end of your shift, take a nap, or call a cab or a friend to drive you. Never drive drowsy. It could be fatal to you and to others on the road.
- Before you leave the building to go to your car, put on dark glasses or special blue-light blocking glasses and keep them on until you are ready to get into bed. This may help you to sleep a bit longer by fooling your body to think it is still night.
- Do not stay late after night shift. When your shift ends, leave quickly. Do not stay for meetings, or to work overtime.

When you get home from night shift:

- When you get home, go right to bed. There is a short window of sleep opportunity before your circadian clock will stimulate you to wake up, even if you have not had enough sleep, so do not waste it doing chores, shopping, or socializing.
- Make your bedroom very dark and quiet. This means investing in good quality blackout shades, disconnecting the phone, using a white noise machine or fan, (or earplugs and eye mask) and communicating to others your strong need to not be disturbed.
- If you did not get enough sleep when you first got home, try to nap before returning to work.
- Do not attempt to provide childcare or eldercare during your sleep period. You will need to find other alternatives (e.g., daycare) while on night shift in order to achieve sufficient sleep to be a safe driver, worker, and parent. Your child will not nap long enough for you to get enough sleep.
- If you are on permanent night shift, and if it is possible for you to keep the same wake/sleep schedule on the weekend and days off, that is ideal. If you cannot do this (most people cannot) at least keep an intermediate position by staying up until 3 AM and sleeping until noon on days off.
- Because night shift workers are chronically sleep deprived, try to do as little night shift as possible (days per week, weeks per year, years per career), and take opportunities to catch up on sleep whenever you can.
- If you have a chronic health condition such as diabetes, epilepsy, heart disease, cancer, or are pregnant, avoid the night shift as it may further impair your health.

(Table 22.4). Workers experiencing extreme shift work symptoms (sleepiness during the shift and daytime insomnia) may have a genetic inability to adjust to shift work, shift work sleep disorder (SWSD) (Table 22.1) (see Chapter 10, Circadian Rhythm Disorders) (Viola et al., 2007), although the boundaries of the disorder are not extremely clear. Few workers seek treatment for shift work complaints, as many think that excessive difficulty sleeping during daytime hours or remaining alert at night is a normal part of shift work and are not aware that there are treatments available for this physiologically based disorder. Sack et al., (2007) reviewed literature describing risk factors for SWSD and concluded that older workers may have more shift work intolerance; women may get less sleep than men due to social obligations and thus have greater sleepiness on the job; and workers who are exposed to bright light in the early morning can have maladaptive phase shifting that reduce their ability to sleep in the daytime. However, not all of these risk factors were supported by robust evidence. The prevalence of SWSD has only been estimated in one population based sample (Drake, Roehr, Richardson, Walsh, & Roth, 2004), where in a random telephone sample, 32% of night shift workers, and 26% of rotating shift workers had either insomnia or excessive sleepiness that was job related.

Table 22.5 ■ *Sleep Impairment and Disability*

The Americans with Disabilities Act (ADA), was designed to protect disabled workers from discrimination by ensuring that their disabilities were accommodated by the employer. Workers with sleep disorders have pursued legal remedies when their sleep disorder has not been accommodated, or when they were dismissed after disclosing a sleep disorder. Sleep is one of the "major life functions" that is part of the disability definition. However, courts have generally sided with employers, such that workers with significant sleep deprivation (5 hours per night) were found able, since "most people" could function adequately on little sleep.

Reasonable accommodations for shift work sleep disorder include the following.

- Reduce night shift participation.
- Allow planned naps during the night shift.
- Allow use of stimulant medication to preserve alertness.
- Increase ambient light in the work environment.

The ability of an employer to provide Americans with Disabilities Act (ADA) accommodation to a worker with SWSD depends on production schedules, available personnel, and budget. Occupational health departments should be instrumental in detecting workers with this disorder and working with the employer to tailor the job to improve the workers' health and safety as well as on-the-job performance (Table 22.5) (Moran, 2007; Morin, 2006).

Sleep Apnea

Sleep deprivation associated with shift work can lead to the development of sleep-disordered breathing through its direct effect on airway patency during sleep. (See Chapter 8, Sleep-Related Breathing Disorders.) Sleep deprived adults with mild or moderate sleep apnea have an increase in abnormal respiratory events and episodes of oxygen desaturation, but even adults with no sleep apnea have an increase in the percentage of time snoring, which could increase daytime sleepiness (Stoohs & Dement, 1993). This problem is compounded by weight gain as night shift workers snack during their shift to preserve alertness (Waterhouse, Buckley, Edwards, & Reilly, 2003), and over years of shift rotation, this can result in a higher body mass index (and increase the risk for obstructive sleep apnea) when compared to straight day shift workers (Parkes, 2002). Night shift workers with obstructive sleep apnea have higher apnea/hypopnea index scores when working the night shift, compared to when they work day shift (Laudencka, Klawe, Tafil-Klawe, & Zlomanczuk, 2007).

INTERVENTIONS TO REDUCE WORK-RELATED SLEEP PROBLEMS

Because work-related sleep problems are due to both individual and organizational factors, a robust worksite health promotion program designed around a thorough exposure assessment can influence employees' individual behavior toward a healthier lifestyle. Employers must integrate screening/surveillance, sentinel event recognition, and clinical interventions into a comprehensive approach to preventing work-related sleep disorders (Table 22.6). In this section, solutions at both levels of intervention that have been used in the past to promote sleep among workers are addressed. It is in the interest of the employer to maintain employee's health at a high level in order to ensure worker safety and productivity.

Individual Level Interventions

Even though much of sleep is affected by how work is organized, there are individual factors that strongly influence the worker's duration and quality of sleep. In order to improve sleep, there are lifestyle and drug therapies that can be used to induce and sustain sleep, or preserve alertness at work. Worksite health promotion programs educate employees to important lifestyle modifications included scheduling of bedtime, regulating and timing caffeine, obtaining appropriate child care when the worker needs to sleep without interruption, and improving the sleep environment. In this section, we describe these individual level interventions.

Sleep Timing

Because night shift workers usually revert to a day shift sleep pattern on their nights off,

Table 22.6 ■ *Elements of a Comprehensive Occupational Health Program to Prevent Sleep Disorders*

Employer	Worker/Staff	Supervisor/Nurse Manager	Occupational Health Nurse
Develop a Policy on Work Schedules	Participate in development of policies/know and understand policy	Participate in supervisor training/provide feedback on scheduling issues to management	Conduct an exposure assessment of all job titles for scheduling practices, overtime use, vacancy rates, participate in evaluation and feedback relating to schedule policy
Provide on-site or contractual, occupational health services	Report to occupational health services when feeling drowsy on the job, for insomnia, and after all injuries and accidents	Refer drowsy employees and those who sustain injuries or accidents to the employee health unit	Establish a protocol for sentinel occupational health events (SEIH) when employees with drowsiness, insomnia, and accidents/injuries come to the employee health unit. Utilize the SEOH to assess workers in similar job titles and work units for occupational sleep disorders.
Provide education and tools to implement policy to workers and supervisors	Participate in regular and periodic sleep hygiene education; communicate sleep hygiene practices and work schedule to primary care clinician	Utilize work scheduling tools such as software; participate in exposure assessment	Assist with development and teaching of sleep hygiene/health promotion classes to workers; monitoring participation rates of employees and units; provide feedback on participation to managers
Monitor and evaluate policy implementation/seek feedback from workers and supervisors	Provide feedback to supervisor on scheduling policies; go to employee health unit for health concerns related to sleep	Participate in evaluation of work schedule policies; provide feedback to managers; refer workers with sleep and safety issues to Employee Health unit	Take work and work schedule histories on admissions to employee health unit for occupational injuries and accidents; conduct surveillance on injuries and accidents related to sleep/fatigue/work schedule
Accommodate workers with occupational sleep disorders in jobs that will not exacerbate their symptoms	Follow recommendations for sleep hygiene and accommodations on the job	Evaluate job performance of employee	Monitor symptoms and health of workers on accommodations or light duty post diagnosis of work-related sleep disorder. Recommend follow-up and communication with primary care provider.

their circadian systems can become dysregulated, creating poor sleep throughout the work schedule cycle. Eastman's extensive laboratory research on timing sleep to produce alertness during the night demonstrates that it is possible to improve nighttime performance in carefully controlled conditions by phase-advancing the sleepiest portion of the night shift. Her most recent study recommends that night shift workers adopt a compromise sleep position by remaining awake into the early morning hours (go to bed at 3 AM) on nights off and then sleep during part of the day (arise at 12 noon) to partially phase shift their circadian system towards nightshift alertness (Smith, Fogg, & Eastman, 2009). Night shift workers should use blue-light blocking glasses in the morning to dampen the circadian upswing from bright morning light (Sasseville, Benhaberou-Brun, Fontaine, Charon, & Hebert, 2009) (Table 22.4). Employees with work-related travel that requires flights across time zones should have individualized sleep plans developed in conjunction with the employee health unit to ensure that they are alert and productive during their business, as well as protecting their health from adverse health consequences from circadian disruption.

Drug Therapy

There are several drugs that can be prescribed to promote either sleep or alertness for workers with SWSD (Schwartz & Roth, 2006). Hypnotics and melatonin can be tailored to induce sleep, sustain sleep, or assist patients to quickly return to sleep when sleep is interrupted based

on the pharmacokinetics of the specific drug (Aeschbach et al., 2009). Stimulants such as Modafinil can be used to sustain alertness during the work shift, as in the "go" pills used in military settings (Czeisler et al., 2005; Czeisler, Walsh, Wesnes, Arora, & Roth, 2009; Moran, 2007), although they should not be a replacement for spending adequate time in bed. (See Chapter 9, Narcolepsy.) Drugs are often prescribed as a first-line therapy by primary care providers without a full diagnostic assessment for sleep disorders. This can increase sleepiness at work if an older hypnotic is prescribed for a worker with an undiagnosed sleep disorder such as sleep apnea. Patients seen by sleep specialists are often those who were unresponsive to medication from primary care settings.

Organizational Level Interventions

Fitness-for-Duty Testing

The science of occupational fitness-for-duty testing to detect sleep deprivation in workers is in its infancy. There are instruments of high quality that are used in research settings to detect fatigue and microsleeps (unintended and uncontrollable episodes of sleep lasting up to 30 seconds) in ambulatory research conditions (e.g., Perclos to detect eye blink activity in truckers, psychomotor vigilance testing (PVT) to detect lapses in reaction to a visual stimulus), but these are not appropriate for "real time" testing of employees in actual work situations. Because detection is not feasible, a better approach is to reduce the work-related inhibitors of sleep by making sure that work schedules allow sufficient sleep opportunity. Software is commercially available and in common use in some settings to flag fatigue-inducing schedules in workers (Moore-Ede et al., 2004). In this section, we will review organizational measures to reduce the impact of work on sleep duration and quality.

Occupational Screening for Sleep Disorders

In safety-sensitive industries, fatigue and sleepiness at work greatly increase the risk for accidents, injuries, and errors. Occupational screening for sleep disorders has become a standard method for managing fatigue risk in the transportation industry (Hartenbaum et al., 2006), but this is rare in other industries (Linnan et al., 2008). In one study in which a large scale occupational sleep disorders screening program was introduced into industrial work settings (power plants, medical fabrication plants, heavy machinery repair), there was a 30% reduction of injury rates among excessively sleepy workers (Melamed & Oskenberg, 2002). A benefit of occupational screening is that patients without daytime symptoms often have occult sleep disorders, but generally do not seek medical attention. Lavie (2002) proposes that rather than waiting for symptomatic patients to be referred, screening programs should be used to identify individuals with sleep-disordered breathing at the youngest age possible (similar to hypertension and diabetes screening) to prevent cardiovascular morbidity and mortality. Also, some portion of the high fatigue seen in shift workers, often discounted as an unavoidable reaction to shift work, may actually be due to the presence of a treatable primary sleep disorder, such as sleep apnea or narcolepsy (Hossain, Reinish, Kayumov, Bhuiya, & Shapiro, 2003).

Screening for sleep disorders in occupational settings has several limitations. Most screening instruments are highly sensitive, but lack specificity (Hartenbaum et al., 2006). Thus employers will be faced with difficult decisions about when to follow up positive screening results with sleep specialist consultations and expensive polysomnograms. However, failing to heed the screening test result must be balanced with a risk of employing a person with a sleep disorder and then assigning him to a shift rotation schedule that will exacerbate his underlying condition and possibly cause an unsafe work environment for himself and others as well as reduced productivity and increasing the latent conditions for worker errors to occur.

Comprehensive Occupational Health Programs

Table 22.6 summarizes the elements of a comprehensive occupational health program to prevent sleep disorders. While these elements

may vary in order or intensity depending on the workplace, the basic outline begins with policies that cover scheduling practices. From there, occupational health services may or may not be provided on site, but linking safety systems, scheduling policies, and occupational health services is critical to preventing illness and injury secondary to scheduling practices. In many workplaces, a health and safety committee with multidisciplinary representation will coordinate evaluation of system level data with the employee health unit. Figure 22.1 displays a comprehensive model of occupational health services designed to prevent work-related sleep disorders and their health and work consequences.

The foundation (as depicted in Figure 22.1) is a thorough assessment of scheduling practices by department and job title typically conducted by the safety professional and the occupational health nurse. In addition to an assessment of scheduling practices, trends in occupational accidents and injuries can be examined simultaneously to benchmark their association with scheduling practices. Next, employee health promotion and education programs are vital to address employee lifestyle and sleep hygiene behaviors which impact work and health but largely occur on personal time. Occupational sleep disorders and their sequelae will go largely undetected without basic screening and surveillance during preplacement and routine encounters with workers in the employee health clinic. The practice of recognizing sentinel occupational events is related to screening and provides early clinical care to the affected worker. It also facilitates investigation of whether coworkers in similar jobs are experiencing risks for work-related sleep disorders. Sleep complaints, drowsiness on the job, and all injuries and accidents should be considered sentinel occupational events requiring further investigation at the population level. Finally, the clinical services for occupationally induced sleep disorder must include a thorough exposure and occupational health history, appropriate referrals and case management, and job accommodation upon return to work (Greenburg & Roger, 2006; McPhaul, 2002; Olson et al., 2005; Silverstein, 2005).

Sleep Hygiene, Shift Work, and Long Work Hours Education for Workers

Many workers have no orientation about steps that they can take to improve sleep duration and quality while working night shift, early morning start time, and long work hours. Sleep hygiene education is one of the most widely delivered basic treatments given to patients with sleep disorders in sleep medicine settings, and should be routinely incorporated into employee orientations (Harvey, 2000).

Napping at Work. Napping during the work shift is controversial in many industries, and for some workers it is grounds for dismissal. Yet, unplanned naps do occur when sleep pressure overwhelms the worker's drive for alertness. Certainly a planned nap under controlled circumstances is preferred to "falling asleep at the switch." Both laboratory and workplace studies have confirmed (by EEG) that a brief 15–20 minute nap during a work shift confers additional alertness, especially for workers with partial sleep deprivation or those working in monotonous tasks (Driskell & Mullen, 2005). A nap of 20 minutes reduces the risk for sleep inertia (grogginess upon waking), and the ideal time to take a nap is at the circadian nadirs (2–4 PM, 2–4 AM). Among 12-hour night shift workers, reaction times were quicker after a 20-minute nap was taken between 1 and 3 AM (Purnell, Feyer, & Herbison, 2002). However, only half of workers reported falling asleep during the scheduled nap period; the most common complaint was excessive noise, suggesting that creating appropriate conditions for napping may improve the outcome. Knowledge transfer of the benefits of napping have been slow, and this evidence-based information will only be applied when the specific constraints and incentives in individual worksites to overcome concerns from employers about "sleeping on the job" rather than "napping at the break" (Anthony & Anthony, 2005).

Ensuring Reasonable Work Schedules

Whether workers "self-schedule" or are assigned their work hours, having adequate sleep opportunity is critical, and work schedules are often

at odds with this. Fatigue risk software has been used since the mid-1990s to evaluate day-by-day fatigue and error risks related to job schedules in UK safety-sensitive industries such as rail, bus, chemical, nuclear and offshore operations (Folkard & Lombardi, 2006; Folkard, Robertson, & Spencer, 2007), and military, rail and airline applications in the United States (Hursh et al., 2004). Several software packages are available, and differ slightly in the prediction formulas and covariates entered into the models; however, there is general agreement that key elements must be present in order to predict fatigue risk. These are (1) a cumulative component where patterns of work on previous shifts influence the current shift, (2) timing of work including start time, shift length, and time of day throughout the shift, and (3) the nature of the work (job intensity) as well as patterns of breaks taken during the shift (Folkard & Lombardi, 2006). This approach was successful in a study where managers and dispatchers were trained scheduling methods to reduce driver fatigue using fatigue risk management software. This led to significant reduction in fatigue risk scores, fewer accidents, and lower insurance premiums 9 months after the intervention (Moore-Ede et al., 2004).

Most employers look at the issue of worker fatigue as inevitable and are unaware of the science that supports using fatigue risk tools to examine current scheduling practices to improve outcomes. Employers also believe that workers want to compress work schedules to "get it over with" so that they can have larger blocks of time off. This was recently contradicted in a study of police officers, where a healthier pattern of rotating shifts was tried. At the beginning of the study, officers were unhappy with having fewer days off per month with the healthy shift pattern, but at the end of the study they were more satisfied with their work schedules because they now had at least 16 hours off between shifts despite fewer days off per month (Kecklund, Ericksen, & Akerstedt, 2008). In general, evening shift workers have the longest and best quality sleep of 8-hour shift workers (Akerstedt, 2003).

Modifying Ambient Light on the Night Shift

Many work environments are dimly lit and without any circadian variation in light levels. Environmental modifications are an inexpensive way for employers to improve alertness in workers. Bright light has been shown to improve alertness in night shift workers, through both direct alerting effects (Campbell, 1995), and through suppression of melatonin production (Santhi, Aeschbach, Horowitz, & Czeisler, 2008). Exposure to blue-light phase shifts the circadian rhythm if the retina is exposed during specific sensitive periods in the circadian cycle (Cajochen et al., 2005). There have been studies using a variety of light "doses" and timing to improve alertness in night shift workers, and many have shown benefit (Boivin, Tremblay, & James, 2007; Shechter, James, & Boivin, 2008). Light has also been used to reduce decrements in alertness during the postprandial dip in dayshift workers (Hayashi, Masuda, & Hori, 2003). Amber glasses are also helpful to block blue-wavelength light from reaching the non-rod non-cone receptors in the retina, to prevent the circadian day from being triggered in night shift workers upon their departure from work.

GAPS IN SCIENCE AND IMPLICATION FOR FUTURE RESEARCH

Despite extensive research that documents the importance of sleep for workers, the consequences of their not receiving sufficient sleep, and remedies to reverse this trend, there are still gaps in the research literature. These gaps have prevented strong policy development leaving many safety sensitive industries without regulation and guidance. Areas of needed research are described:

- Most of the sleep deprivation laboratory studies that have shown performance decrements have used healthy young adults with no "work" during waking hours, and no social responsibilities during leisure hours. The effect of occupationally produced sleep deprivation needs to be studied in actual worker populations such as adolescent workers still in school, young parents with children, pregnant women, older workers with chronic

diseases and risk factors for chronic diseases, workers providing caregiving to elders, and aging workers. These special populations may need protections in the workplace to prevent illness and injury due to sleep deprivation.

- The science of screening for sleep disorders in occupational settings warrants additional research. Currently the questionnaires that can flag workers with a suspicion for sleep disorders lack specificity, and can be costly to companies that must follow through with confirmatory evaluations. Creating better user-friendly risk profiles can help companies to maintain productivity by locating and assisting workers with latent sleep disorders.
- There are no readily available "fitness for duty" tests to screen workers in real time for potential sleep deprivation-related safety hazards despite years of neurobehavioral research.
- Workplace health promotion efforts are effective in reducing back pain, diet, exercise and smoking cessation but have bypassed sleep as a key factor to promote worker health. There is adequate science to support a variety of workplace interventions, and these need to be combined into comprehensive programs that target the myriad of factors that reduce sleep in workers. Outcomes should include both proximal (sleepiness, fatigue), intermediate (injury, acute illness), and distal (chronic disease) events. This will require interdisciplinary collaboration and skill at translational science.
- The policy debate between external regulation and self-regulation for industries with sleep-destructive organization of work should be addressed. Lee (2006), writing about regulation of resident work hours, posits that only Federal legislation will be effective in curtailing dangerous hours of work. He carefully describes the failure of regulation efforts by States and by self-regulating accreditation bodies (e.g., ACGME), as well as the undesirability of allowing tort law to shape hours of work. Only the federal government has the strong national interest to promulgate regulation, and the financial and personnel resources to implement regulation across State boundaries.

Employers are required to provide a "safe and healthful workplace, free from recognized hazards." The association between work schedules, work stress, sleep disorders, and cardiovascular health outcomes creates an important hazard for consideration by the U.S Occupational Safety and Health Administration (OSHA). Dangerous work schedules should become an OSHA issue. The possibility of compensable claims via workers compensation should also be a focus of policy development. This will likely vary by State, but the stronger the evidence and more precise the screening and diagnostic tools, the higher the likelihood that occupationally induced sleep disorders will be compensable under most workers compensation schemes. Finally, in safety sensitive industries such as Health care, work schedules are coming under increasing scrutiny in terms of patient safety. Long hours for residents and nurses may be jeopardizing patient health and safety in acute care hospitals and possibly other Health care settings. It is clear, however, that these policy implications hinge on strong scientific evidence of the association between work schedules, job stress, and adverse health outcomes.

SUMMARY

The work relatedness of sleep is clear, and the responsibility to improve the duration and quality of sleep must be shared by the worker, the employer, and clinicians who are informed about sleep. Companies who require workers to be productive and safe need to consider all aspects of the employment situation to ensure that sleep deprivation is not compromising the health of their valuable workforce or their productivity. Similarly, workers themselves must make time for sleep, select a schedule that permits adequate sleep, seek treatment for sleep disorders, and adhere to treatment recommendations. Occupational health providers are in a prime position to improve the health of a large swath of the population by employing population-based principles to ensure that work-related sleep deprivation is reduced or eliminated, and that employees receive education and treatment to improve sleep quantity

and quality. For nurses as employees, this will protect their health, and possibly the safety of the patients that they serve. For occupational health nurses, this will add a new emphasis area to their practice, and allow them to use their current skills in an area that will reap great reward towards improving the health of their worker population.

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The Role of Advanced Practice Nurses (APRNs) in Specialized Sleep Practice

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Treatment of disorders of sleep and wakefulness has grown considerably since the first sleep disorders centers were established in 1970 (Tachibana, Ayas, & White, 2005). Nevertheless, 50–70 million people in the United States suffer from chronic sleep disorders (Institute of Medicine [IOM], 2006). Despite the presence of over 1,200 sleep centers in the United States (IOM, 2006), there is a shortage of health professionals who specialize in sleep disorders treatment and therefore, an insufficient number of providers to whom primary care practitioners can refer their patients. As a result, millions of individuals suffering from sleep disorders remain undiagnosed and untreated (Kapur et al., 2002; Young, Evans, Finn, & Palta, 1997). Sleep disorders treatments will likely become more commonplace as awareness of sleep disorders and their impact and support for the efficacy and effectiveness of treatment increase. The national shortage of sleep specialists and the broad-based skills of advanced practice nurses (APRNs) in assessment, diagnosis, health promotion, and management of episodic and chronic health conditions present an excellent opportunity for APRNs to develop practices in sleep. The purpose of this chapter is to discuss the roles, qualifications, and educational opportunities for APRNs in specialized sleep practice. Exemplars of practice models are provided to illustrate available opportunities.

ROLES OF ADVANCED PRACTICE NURSES IN SPECIALIZED SLEEP PRACTICE

Advanced practice nurses are members of a growing cadre of health care providers who provide health care for people with sleep disorders. This interdisciplinary field is comprised of over 13 medical and dental specialties (e.g., neurologists, pulmonologists, psychiatrists, pediatricians, otolaryngologists, oral and maxillofacial surgeons, dentists), psychologists, registered nurses, and APRNs, as well as sleep technicians and technologists. APRNs are ideally suited to provide expert clinical care, including cost-effective health promotion, disease prevention, and management of acute and chronic disorders. They are well prepared to provide complex behavioral interventions and act as skilled facilitators who ensure effective coordination and delivery of sleep services as independent practitioners or as members of collaborative practices.

The roles of APRNs in sleep disorders centers are quite varied and can evolve over time as the practitioner gains experience and skills and as the needs of the clinic change. In addition to management of a caseload of patients, there are often opportunities for advocacy, teaching, mentorship, and research. These opportunities often depend on the scope of the sleep disorders center and the interests and capabilities of the APRNs.

APRNs manage their own caseloads, conduct history and physical assessments, develop differential diagnoses, order and interpret tests, explain test results to patients, order treatments, including medications as needed, and follow-up with their patients to evaluate treatment efficacy. Depending on the scope of the sleep disorders center, the APRN might manage patients with obstructive sleep apnea (OSA), periodic limb movements, REM behavior disorder, insomnia, narcolepsy, idiopathic hypersomnia,

and circadian rhythm disorders, among other disorders. APRNs with appropriate training might also provide cognitive behavioral therapy for insomnia (CBT-I) for individuals and groups, while others may have more limited responsibilities, for example, managing follow-up visits for adherence to continuous positive airway pressure (CPAP) treatment. Depending on the focus of their APRN specialty (e.g., family, adult, psychiatric-mental health), they may work with patients across the lifespan or with patients who have medical or psychiatric conditions that are comorbid with their sleep disorders. Although the precise number of APRNs working in sleep specialty practice is not known at present, there seems to be growth in this subspecialty area. APRNs from a variety of specialties are currently working in specialty sleep disorders practices.

APRNs educate and support patients to adopt and adhere to CPAP and behavioral sleep treatments. They may also lead individual or group educational sessions for patients or patient support groups around specific sleep issues, such as narcolepsy, sleep apnea, or children's sleep needs. There are many opportunities to speak with community groups to raise awareness about sleep disorders and their treatment.

APRNs may also serve as advocates for sleep-related issues. These may include advocacy for "sleep-friendly" school start times for adolescents (see Chapter 2, Developmental Aspects of Sleep) or for prevention of sleep loss in shift-workers (e.g., see Chapter 22, Sleep in Occupational Health Settings). APRNs also work closely with patients and insurers to advocate for adequate reimbursement for sleep disorders treatments. The target groups for these activities also include the public and policy makers. Some APRNs have also provided interviews for the media to raise awareness of the importance of sleep and sleep disorders.

Given the pressing need for educating health professionals about sleep and sleep disorders, there are many opportunities for APRNs who specialize in sleep to present and publish on clinical management of sleep disorders. Audiences for this work include APRNs who are not sleep specialists, physicians, and other health care professionals who are interested in sleep, as well as the public, with relevant publications depending on the target audience. Two recent publications are examples of the work of APRN authorship or coauthorship (Blythe et al., 2009; Valerio, 2009).

Mentoring and precepting APRNs and other health professionals, including physicians and psychologists who are novices in the care of patients with sleep disorders, are also important roles for APRNs. These roles are critical, given the scarcity of such training opportunities. Increased awareness of the importance of sleep among educators of APRNs is likely to lead to increased needs for clinical placements of APRN students in sleep disorders centers.

Sleep disorders centers present many opportunities for APRNs to engage in research. Research conducted at sleep disorders centers is quite varied and ranges from descriptive studies that may be conducted with chart review data, for example, to Phase II–IV clinical trials of medications and other treatments. These trials may be initiated by the investigators at sleep disorders centers in academic medical centers or the centers may participate in multisite clinical trial in which the sleep center primarily recruits and studies patients. Studies may be funded by industry, foundations, or the government (e.g., NIH).

APRNs may serve as coinvestigators or as principal investigators, if they are doctorally prepared. For example, one author of this chapter (Teresa D. Valerio.) collaborates with a university-based nurse scientist. The other author of this chapter (Ann E. Rogers) served as a site primary investigator (PI) during the Phase III trial of modafinil, and recruited patients for several of her NIH-funded projects from her practice at the Michael S. Aldrich Sleep Disorders Center at the University of Michigan Medical Center. As site PI and the principal investigator of her own NIH-funded studies, Dr. Rogers was responsible for overseeing all aspects of subject recruitment, study methodology, data collection, quality assurance protocols, and data entry, analysis, and manuscript preparation for her own studies.

Some APRNs participate in research by recruiting, screening, and securing informed consent

from participants in clinical trials, and monitoring them for adverse events in follow-up visits. The APRN might be asked to assist in recruiting, screening, and consenting patients for participation in clinical trials, and monitoring them for adverse events during follow-up visits. If APRNs recruit patients from their own practices, they must carefully adhere to IRB requirements and avoid the perception of coercion. Patients may feel that they have the obligation to participate in the study and wish to please the health care provider. APRNs must make it clear to patients that they can refuse to participate without jeopardizing their care or their relationship with their health care providers.

EDUCATIONAL PREPARATION OF APRNs IN SLEEP AND SLEEP DISORDERS PRACTICE

Formal Educational Preparation in Sleep

Although APRN curricula provide an excellent foundation in health assessment, pharmacology, health promotion, and management of episodic, acute, and chronic conditions relevant to specific populations (e.g., family, adults, gerontology, pediatrics), there is a deficit of sleep content and clinical experiences with sleep disorders in nursing curricula, just as there is a deficit in medical school curricula (IOM, 2006; Lee et al., 2004). In fact, a recent survey of nurse practitioners (NPs) in Illinois found that about 28% of students reported no sleep content in their formal education, and when curricula included sleep, there was an average of only 2.1 hours devoted to this topic (Valerio, unpublished data). Similar results were obtained in a survey of 2,500 nurses that include a large percentage of APRNs who enrolled in an online educational program. More than 70% were exposed to 5 hours or less of sleep-related content across their entire professional careers (Phillips McEnany, 2010, personal communication), which spanned over 20 years for the majority of participants.

In the late 1990s and early 2000s, the National Institutes of Health awarded "Sleep Academic Awards" to medical schools throughout the United States, with the primary goal of increasing sleep content and clinical experiences in sleep in medical schools. Several nurses were involved in these projects in academic medical centers. Although an array of educational tools were developed and are now available online for incorporation into curricula (http://www.aasmnet.org/medsleep.aspx), uptake of these materials into nursing curricula has been sporadic. Although Lee and colleagues identified learning objectives and proposed content to be included in nursing curricula in 2004 (Lee et al., 2004), few nursing programs adopted this program or included elements of it in their undergraduate or graduate programs. There is a pressing need for systematic inclusion of content and clinical experiences with sleep, chronobiology, and sleep disorders in undergraduate and graduate-level nursing curricula. (See Chapter 24, Future Directions in Sleep Promotion: Nursing Practice, Research, and Education.)

Continuing Education and Informal Sleep Training in Sleep

Because most APRNs currently have little sleep content or clinical experiences in their formal undergraduate or graduate education, there is a pressing need for continuing education (CE) to address this gap and provide current information. Participants in the survey of Illinois nurses (above) (Valerio, unpublished data) reported completing an average of 9 hours of CE, with two-thirds reporting CE content devoted to sleep after completing their graduate nursing education. However, the amount and scope of the content of these courses was not elicited.

APRNs reported that they acquired their knowledge and skills in sleep disorders treatment informally through attendance at CE programs offered by the American Academy of Sleep Medicine (AASM), the annual meeting of the Associated Professional Sleep Societies (APSS), and mentoring from physicians and other sleep specialists (Valerio, unpublished data).

Several major initiatives have been completed or are underway to better prepare practicing APRNs with knowledge and skills necessary to practice in the specialty of sleep, with a particular

focus on nursing perspectives on sleep. With the partnership of the American Psychiatric Nurses Association and Sigma Theta Tau International, Geoffry Phillips McEnany PhD, APRN, BC professor at the University of Massachusetts Lowell developed an online CE program on sleep, consisting of 12 online CE modules on sleep and sleep disorders (http://continuinged.uml.edu/sleep/). Over 2,500 nurses representing a variety of clinical specialties participated in this accessible program.

Several nursing schools offer CE and/or postmaster's programs in sleep. The Nursing Department of the School of Health & Environment at the University of Massachusetts Lowell offers an online interdisciplinary (Nursing, Psychology, Social Work, Medicine and other health care disciplines) that provides a postmasters certificate in sleep (http://continuinged.uml.edu/online/Certificates/sleep.cfm). This program consists of four online courses that provide critical content in the fundamentals of sleep and chronobiology, diagnosis and intervention. The final course provides the participant with an opportunity to pursue a faculty-guided learning experience in a self-selected area of learning need. An advantage to this program is its interdisciplinary focus and accessible online format.

The University of Pennsylvania School of Nursing offers a postmaster's nursing certificate program in sleep (http://www.nursing.upenn.edu/academic_programs/grad/masters/program_detail.asp?prid=42). Inaugurated in 2009, it includes two online courses and a third, hybrid course that includes an on-campus residency and supervised clinical practicum.

In conjunction with the annual national Conference for Advanced Practice in Primary and Acute Care sponsored by the University of Washington (UW) Continuing Education, faculty in the UW Schools of Nursing and Medicine have presented all day workshops on the management of sleep disorders. These interdisciplinary workshops focused on the management of insomnia in primary care practice and on the diagnosis and treatment of sleep-disordered breathing disorders. The Web site for continuing nursing education is http://uwcne.net/ and for the specific workshop Web site is http://uwcne.net/secure/display3.asp?SKU=10131-C

APRNs can also complete interdisciplinary CE programs, such as those developed by the AASM for physicians and psychologists. Those who wish to learn CBT-I can take a course such as the one developed by Michael Perlis, PhD, at the University of Pennsylvania (mperlis@exchange.upenn.edu). APRNs who are prepared at the doctoral level are eligible to participate in the Mini-Fellowship Program for Behavioral Sleep Medicine sponsored by the American Academy of Sleep Medicine (http://www.aasmnet.org/BSMMiniFellow.aspx); or undergo longer behavioral sleep medicine training offered by one of nine different AASM BSM Accredited Training Programs (http://www.aasmnet.org/BSMPrograms.aspx). Clinical seminars and lectures are available at the annual meeting of the Associated Professional Sleep Societies. Large, level 3 sleep disorders centers, such as the Center for Sleep and Respiratory Neurobiology at the University of Pennsylvania, also offer weekly seminars on sleep-related topics that are accessible by video and the Web (http://www.med.upenn.edu/sleepctr/Conferences.shtml). In addition, some states have regional sleep societies that offer annual sleep symposia for clinicians and researchers; and CE courses are becoming more widely available at state and national nursing conferences, as APRNs gain interest and expertise in sleep disorders treatment.

Given the rapid growth in the field of sleep and the need for specialized clinical knowledge in this area, there is a growing interest in specialty certification for APRNs in this area to document clinical expertise. There is an ongoing exploration of the necessary credentialing, training, and identification of the appropriate certification body.

SPECIALIZED SLEEP PRACTICES AND SETTINGS

Sleep Laboratories and Sleep Disorders Centers

Sleep laboratories are primarily testing facilities and do not provide clinical evaluation or management of sleep disorders. Technicians perform tests and conduct initial scoring of

polysomnographic sleep studies, and physicians read and report the test results.

Sleep disorders centers provide laboratory services for diagnosis and specialized clinical services for evaluating and managing patients with sleep disorders. Some centers specialize in sleep-disordered breathing in pediatric, geriatric, and adult populations, whereas other centers provide more comprehensive services for patients with the full range of sleep disorders. Some centers are independent practices in one or more locations, whereas others are affiliated with academic medical centers. The latter often have health professional trainees and are more likely to have ongoing research protocols.

Over 2,000 sleep disorders centers in the United States have demonstrated proficiency in testing procedures, patient safety, and follow-up care, as well as physician and staff training and are certified by the American Academy of Sleep Medicine (AASM) (American Academy of Sleep Medicine, 2008a). However, not all sleep centers have AASM accreditation. Although sleep centers specializing only in sleep-related breathing disorders were once accredited by the AASM, this category of accreditation was phased out on January 1, 2011. All accredited centers are required to apply for reaccreditation as full service sleep disorders centers (American Academy of Sleep Medicine, 2008b).

APRN Practice in Sleep and Sleep Disorders

APRN practices in sleep are varied and occur in several different contexts. Given the evolving nature of the field of sleep and the growth in new opportunities, a variety of creative models may be employed. For example, some APRNs are the only nurse members of a team, members of a team that includes several APRNs in larger sleep disorders centers, and others are in solo practices or use consultant models of care.

Group Practice

The Illinois Neurological Institute Sleep Center (Peoria and Morton, Illinois) is a large practice that is owned by a health care system and provides comprehensive sleep disorders assessment and treatment to patients of all ages. Its staff includes four NPs, two physicians, registered nurses, medical office assistants, sleep technicians, and support staff. In 2009, there were 6,846 clinic visits and 3,052 sleep tests performed. The NPs provide the majority of the evaluation and management of sleep patients and in collaboration with a physician who is certified in sleep medicine through the American Board of Sleep Medicine. The two family NPs see children and adults. NPs diagnose and treat all types of sleep disorders and videoconferencing is available for off-site sleep physicians to talk with the patients.

The majority of patients are referred by their primary care or specialty provider to the Illinois Neurological Institute Sleep Center, although self-referrals are also accepted. Patient care begins with a complete sleep history and physical examination; clinical evaluation is done prior to testing or treatment, and follow-up care is available to monitor the patient and revise therapy if necessary. Referrals are available for otolaryngology, dentistry, psychiatry, psychology, general or specialty neurology, and home medical equipment.

Megin Meyers, MS, ANP represents the growing cadre of APRNs who are striving to increase their contribution to the field of sleep disorders care by seeking employment in well-staffed clinics that offer excellent teaching and training opportunities. The University of Pennsylvania Sleep Center hired her in 2006 and she is gradually expanding her scope of practice. She currently is responsible for follow-up of patients who are using CPAP and occasionally sees patients who have insomnia.

Susan McClusky, MSN, RN, an NP at the Philadelphia Veterans Administration (VA) Medical Center Sleep Disorders Center, manages her own caseload of patients 2 days a week. She evaluates patients on their initial visit, takes medical histories, performs physical exams, and does follow-up care. A board-certified sleep disorders specialist or sleep fellows read and interpret traditional polysomnographic recordings, PSGs. Ms. McClusky reads and scores all unattended sleep studies and auto-titration CPAP studies. She also answers outpatient crisis

phone calls and handles in-house referrals. In the past, she co-led a group CBT-I program with a psychologist. In 2009, in response to the long (6 months) waiting list at the VA Hospital sleep disorders center, Ms. McClusky began training another NP at a community-based VA clinic in New Jersey to see patients with suspected sleep disorders to increase the access of the VA patients to sleep specialty care. She is now developing a program in conjunction with a gastrointestinal clinic at the VA Hospital to ensure that patients suspected of having OSAs are screened before receiving anesthesia (see Chapter 7, Sleep Disordered Breathing and Chapter 20, Sleep in the Acute & Critical Care Setting) and receive appropriate ventilator support during the procedure. She also initiated research into the prevalence of sleep apnea symptoms among veterans attending general (i.e., not sleep disorders) outpatient clinics at the Philadelphia VA Hospital.

Independent Practice

Frank Breznyak, FNP, practices in a pulmonary medicine clinic at the Penobscot Respiratory Clinic in Bangor ME (population 35,495). He is part of a team that includes several pulmonologists, three of whom are board-certified in sleep disorders and one in critical care medicine. Mr. Breznyak is the only NP at the Penobscot Respiratory Clinic and the only APRN in the area specializing in sleep disorders medicine. He worked for 24 months under physician supervision before establishing an independent practice, as allowed by Maine law and subsequently completed the postmaster's certification course in sleep at the University of Pennsylvania. Currently, Mr. Breznyak consults with a collaborating physician, as required by the clinic's liability insurance, whenever he has questions. (The collaborating physician also reads and interprets the results of the sleep studies obtained on his patients). Two days each month he staffs a satellite clinic in Pittsfield, ME (population 4,500), where he sees patients of all ages, most of whom are adults and have a wide variety of sleep disorders. Like other APRNs in Maine, he is eligible for reimbursement through Mainecare and other third-party insurers.

Consultative Model

Dana Epstein PhD, RN, the associate chief of the nursing service for research at the Phoenix VA Health Care System in Arizona is employed by the nursing service and specializes in insomnia. She does not work out of the sleep disorders centers but clinicians from a variety of clinics refer patients, including those with HIV/AIDS, depression, post-traumatic stress disorder, and traumatic brain injuries who have insomnia. She also holds educational sessions on insomnia and sleep for patients at other clinics. If participants in these groups need more intensive treatment, Dr. Epstein holds small group sessions for 5–6 weeks focusing on the management of insomnia. The increase in combat veterans who are diagnosed with PTSD or survive traumatic brain injuries is associated with an increased demand for her services.

Dr. Epstein also mentors other nurses who are implementing evidenced-based practice protocols or developing research proposals, and conducts her own research on cognitive behavioral treatment for insomnia.

Private Solo Practice

Psychiatric APRNs are in an interesting position regarding sleep-related dimensions of practice. Sleep dysregulation is ubiquitous across the psychiatric disorders and nurses deal with these issues routinely in practice. (See Chapter 19, Sleep in Psychiatric Mental Health Settings). Psychotherapeutic modalities, including individual, group, and family therapy are standard components of Psychiatric APRN training, and cognitive behavioral therapy is one of the most frequently utilized modalities due to the strong evidence for its efficacy. Therefore, they are well-prepared to provide CBT-I because its underlying principles are essentially no different from other forms of CBT.

Geoffry Phillips McEnany, PhD, APRN, BC is a professor at the University of Massachusetts Lowell and maintains a private practice in Boston, focused on the treatment adults with psychiatric illnesses that are often comorbid with sleep disorders. Treatment of these patients is

multifaceted and usually includes pharmacotherapy and psychotherapy. Cognitive behavioral therapy is a mainstay of treatment for many of the patients, given the perceptual distortions experienced in the course of an illness and the impact of these symptoms on clinical outcomes. He uses CBT in the treatment of insomnia and other cognitive dimensions of psychiatric conditions. His practice richly informs the teaching of graduate and doctoral students and has served as the foundation for innovative initiatives aimed at educating health care professionals across the disciplines, some of which are discussed in this chapter. He believes that psychiatric NPs and clinical nurse specialists are well-positioned to provide CBT-I, given their solid training in CBT. However, there is a need for additional formal education of these nurses in the application of CBT expertise to the specifics of sleep dysregulation.

Telehealth Sleep Consulting Services for APRNs Practicing Outside Sleep Clinics

A significant number of patients with sleep disorders, especially insomnia are treated in primary care or other settings, but do not see sleep specialists. For example, a survey of NPs found that only 9 of 410 respondents caring for patients with insomnia practiced in sleep disorders centers (Goolsby, 2006). APRNs who provide care for patients with a wide variation of conditions, such as autism, cardiovascular disease, and dementia outside sleep clinics also frequently encounter sleep disorders among these populations, and both patients and APRNs would benefit from the availability of consultations with sleep experts. In the future, these APRNs may be able to consult with APRN sleep specialists through telehealth sleep services now being developed by some sleep centers.

SUMMARY

APRNs are well-positioned to develop specialized sleep practices, based on their strong preparation in health assessment, health promotion, and management of episodic and chronic health care conditions in a wide variety of populations and the pressing societal need for an increase in sleep specialists. Their expertise in promoting lifestyle change is a particular strength. Possible roles for APRNS are multidimensional and encompass clinical practice, education, advocacy, and research. Creative models of practice are needed and continue to emerge. While there is a growing cadre of APRNs with specialized sleep practices in a variety of settings, there is a need for more systematic opportunities for education in sleep, chronobiology, and sleep disorders in basic nursing education, graduate school, and CE.

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Future Directions in Sleep Promotion: Nursing Research, Practice, and Education

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Geoffry Phillips McEnany and Nancy S. Redeker

There is abundant evidence of the importance of sleep and sleep disorders in nursing practice. The earlier chapters in this book provide extensive state-of-the-science information intended to guide nursing practice and education regarding assessment and treatment of sleep and sleep disorders from a nursing perspective. The purpose of this chapter is to provide a perspective on future directions in nursing research, practice, and education relative to sleep promotion and prevention and treatment of sleep disorders. The content here provides an opportunity to examine some of the exciting possibilities and challenges for advancing sleep science and the implementation of this evidence in the discipline of nursing. We provide an overview of opportunities and possible directions for nursing scholarship related to sleep, provide an overview of current trends that intersect with the need for evidence-based practice in sleep promotion, and suggest implications for nursing curricula.

NURSING SCHOLARSHIP RELATED TO SLEEP AND SLEEP DISORDERS

Given the focus of the discipline of nursing on health and health promotion, as well as the intersections between biology and behavior, it should be no surprise that nurses and nurse scientists have enthusiastically embraced research on sleep. Nurses are engaged in conducting cutting-edge research on sleep in individuals and families across the life span and states of health. Studies include important qualitative and quantitative work describing characteristics of sleep and sleep disorders, explanatory studies, and randomized clinical trials. Collectively, this work employs a full range of sleep measurement methods and occurs in the context of basic science, the sleep laboratory, and clinical and community-based settings.

In many ways, sleep-related nursing science reflects the rich diversity of the larger domain of the discipline of nursing. Most nurse somnologists have their roots in traditional nursing specialty areas of practice and scholarship and use their interests in sleep to enrich these specialties. They have also contributed significant nursing perspectives (e.g., focus on the lived experience of human health, symptoms, adherence, self- and family management, developmental approaches) to the interdisciplinary field of somnology and broader areas of public health and medical and psychiatric practice. The inter-, multi- and transdisciplinary nature of our collaborations with other nurses from a variety of specialties and scientists and clinicians from many other health-related disciplines continue to enrich the field of sleep. This is evident in the wide range of venues in which nurses publish and present their research.

Evolution of Nursing Knowledge about Sleep

Florence Nightingale emphasized the importance of sleep as early as 1860 in *Notes on Nursing: What it is and What it is Not*. Other leading

nurse scholars, including Virginia Henderson, included sleep-rest as a human need in the early part of the 20th century. With Bertha Harmer, Ms. Henderson provided detailed explanation about nursing interventions related to sleep (Harmer & Henderson, 1939; Henderson, 1955). Although this predated modern sleep research, much of the advice provided presaged current findings of sleep research. Although full descriptions are beyond the space available in this book, sleep and activity-rest are important components of other nursing frameworks that evolved in the latter half of the 20th century and are in use today.

Dr. Elizabeth "Betty" Giblin established the first sleep laboratory in a school of nursing at the University of Washington (UW) in the late 1970s. Her early research addressed sleep apnea and sleep patterns in people with Alzheimer's disease and chronic obstructive lung disease—areas of scholarship that continue to be important today. (http://www.son.washington.edu/about/pr_2-25-05.asp). The UW sleep laboratory was inaugurated within 10 years of the development of the criteria for polysomnographic sleep scoring by Rechtschaffen and Kales in 1968 and was contemporary with identification of obstructive sleep apnea as an important condition. The UW School of Nursing continues to operate a research-focused sleep laboratory and established the UW Center for Research on Management of Sleep Disorders, an interdisciplinary center funded by the National Institute of Nursing Research and focused on developing interventions to improve sleep quality across the life span. The University of Illinois at Chicago School (UIC) of Nursing jointly operates the Center for Narcolepsy, Sleep and Health Research with the UIC Medical School. Several other schools of nursing have sleep laboratories or share facilities with other interdisciplinary departments through collaborations in clinical research centers.

Technological developments have contributed in many ways to the advancement of sleep research overall and nursing scholarship in particular. For example, the development of portable electronic devices enabled sleep recordings in home and institutional environments that facilitated the ecological validity of sleep measurement and overcame the limitations of sleep measurement in the laboratory setting. For example, wrist actigraphy facilitates sleep measurement over weeks and months in a non-intrusive manner. (See Chapter 1, Physiological and Behavioral Aspects of Sleep). Nurses and our colleagues are now conducting cutting-edge genetic and genomic analyses to enhance understanding of the causes and consequences of sleep disorders. We expect that continued technological developments will go hand-in-hand with emerging science.

Exemplars of current nursing scholarship related to sleep are presented in Table 24.1. This list includes data-based publications and reviews that nurses have published as primary (first or last) authors from 2005 to 2010 and that are available on the Internet through PubMed. The list is not intended to be comprehensive, and we apologize if significant contributions have been overlooked. This list demonstrates the depth and breadth of nursing inquiry in sleep consistent with many of the most pressing current public health concerns, as well as important concerns of the discipline of nursing. The growth in the cadre of nurse somnologists is also impressive and includes a second and third generation of scholars who have emerged as scientists since Dr. Giblin's early work.

Opportunities for Training in Nursing Research on Sleep

Opportunities for nurses to obtain training in sleep and sleep disorders at the doctoral, postdoctoral, junior faculty, and mid-career levels are abundant. Potential mentors and faculty include the growing cadre of established nurse somnologists and interdisciplinary colleagues in medicine and the basic and social sciences. Research training in sleep can be funded through faculty research grants, institutional training grants (e.g., NIH T32 programs), and individual NIH doctoral and postdoctoral training grants. Some of the training opportunities are explicitly in the field of sleep, but opportunities are also available to study sleep as it relates to the foci of other areas of interest in U.S. nursing schools

Table 24.1 ■ *Examples of Data-Based, Peer-Reviewed Journal Articles and Reviews With Nurses as Primary Authors (2005–2010)*

Sleep Measurement

Berger, A. M., Wielgus, K. K., Young-McCaughan, S., Fischer, P., Farr, L., & Lee, K. A. (2008). Methodological challenges when using actigraphy in research. *Journal of Pain and Symptom Management, 36*(2), 191–199.

Chasens, E. R., Ratcliffe, S. J., & Weaver, T. E. (2009). Development of the FOSQ-10: A short version of the Functional Outcomes of Sleep Questionnaire, *Sleep, 32*(7), 915–919.

Redeker, N. S. (2008). Challenges and opportunities associated with studying sleep in critically ill adults. *AACN Advances in Critical Care, 19*(2), 178–185.

Developmental Aspects of Sleep

Archbold, K. H., Johnson, N. L., Goodwin, J. L., Rosen, C. L., & Quan, S. F. (2010). Normative heart rate parameters during sleep for children aged 6 to 11 years.*Journal of Clinical Sleep Medicine, 6*(1), 47–50.

Floyd, J. A., Janisse, J. J., Jenuwine, E. S., & Ager, J. W. (2007). Changes in REM-sleep percentage over the adult lifespan. *Sleep, 30*(7), 829–836.

Sleep and Gender/Women's Health

Baldwin, C. M., Grant, M., Wendel, C., Hornbrook, M. C., Herrinton, L. J., McMullen, C., et al. (2009). Gender differences in sleep disruption and fatigue on quality of life among persons with ostomies. *Journal of Clinical Sleep Medicine, 5*(4), 335–343.

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Woods, N. F., & Mitchell, E. S. (2010). Sleep symptoms during the menopausal transition and early postmenopause: Observations from the Seattle Midlife Women's Health Study. *Sleep, 33*(4), 539–549.

Ye, L., Pien, G. W., Ratcliffe, S. J., & Weaver, T. E. (2009). Gender differences in obstructive sleep apnea and treatment response to continuous positive airway pressure. *Journal of Clinical Sleep Medicine, 5*(6), 512–518.

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(e.g., self and family management, symptoms, biobehavioral nursing, genetics, health disparities) and in laboratories and sleep programs situated within basic science departments, sleep laboratories, and other settings.

Funding is available for research training for junior and mid-career faculty who wish to focus or refocus their research programs on sleep-related topics through the NIH K-series awards (research career awards). Sleep research training may also be fundable through specialty organizations (e.g., Oncology Nursing Society, American Heart Association) and foundations.

Funding of Sleep Research

The National Institutes of Health established the National Center for Sleep Disorders Research (NCSDR) as a component of the National Heart, Lung and Blood Institute, and this center plays a leadership role in identifying needs for sleep research. However, many other NIH institutes and centers, including the National Institute of Nursing Research, fund research on sleep and sleep disorders as they are related to their primary missions (e.g., symptoms, self-management, aging, diabetes, cancer, cardiopulmonary disorders, and mental health). The NIH also periodically issues requests for applications (RFAs) and program announcements to inform the research community of opportunities to submit applications on sleep-related basic, clinical, and translational science.

Future Directions for Nursing Scholarship on Sleep

There are numerous opportunities for nurses to engage in research on sleep and sleep disorders. The NCSDR published a strategic plan for research in 2003 (http://www.nhlbi.nih.gov/health/prof/sleep/res_plan/index.html) and solicited input into the development of a new one to be released as this book goes to press. This plan will provide an agenda and focus for sleep research funded by the NIH in the near future. Although there has been a dramatic increase in scientific knowledge about sleep and sleep disorders, there are many unanswered questions. Some are discussed throughout this book, and a few are briefly summarized in Table 24.2.

FUTURE NURSING PRACTICE RELATED TO SLEEP AND SLEEP DISORDERS

One of the resounding and pointed questions among nurses in practice is *how do we adapt information from sleep science into practice?* Although somnology is a young branch of science and the body of sleep research conducted by nurses and others is growing significantly, the need for translation of sleep science into clinical practice remains mainly unmet within the discipline of nursing. The opportunity for the advancement of evidence-based approaches to sleep in nursing practice is tremendous.

Table 24.2 ■ *Suggested Directions for Future Research in Sleep and Sleep Disorders*

- Cost-effectiveness and comparative effectiveness of sleep treatments (e.g., behavioral treatment for insomnia, CPAP for obstructive sleep apnea)
- Strategies for more effective integration and delivery of sleep disorders assessment and treatment in primary care settings focused on patients across the lifespan and state of health
- Effectiveness of algorithms to address sleep in various clinical settings
- Efficacy & effectiveness of interventions to improve sleep in patients hospitalized in acute and long-term care settings
- Health disparities in sleep disorders & sleep disorders treatment
- Effects of sleep loss & circadian rhythm disorders and their treatment on
  - daytime performance
  - injuries & errors (including medical errors)
  - academic performance
  - psychiatric disorders
  - medical conditions (e.g., diabetes and heart disease)
  - self-care & self-management
- Biobehavioral mechanisms that explain the associations between sleep disorders, stress, and psychiatric & mental health
- Genetic underpinnings of sleep disorders and response to treatment
- Effects of complementary & alternative therapies on sleep
- Associations between sleep disorders/sleep loss and obesity & impact of weight loss/gain on sleep
- Biobehavioral mechanisms for the effects of behavioral treatments on sleep
- Interventions to improve adherence to sleep disorders treatments
- Effectiveness of public health interventions and messages to promote healthy sleep
- Family-focused strategies to promote healthy sleep
- Interactions between sleep and other symptoms in people with chronic conditions (e.g., heart disease, cancer)
- Sensitive & specific measures & biomarkers of sleepiness
- Cultural perspectives on sleep and sleep disorders

However, there is a need for a broad-based plan that integrates this evidence with important trends in nursing and society.

The dynamism of nursing practice impressively reflects across the history of our discipline. Over the past 150 years, the work of nurses evolved in form and function, shaped by powerful forces both within and outside of the discipline. The discipline of nursing grows by evolution and convolution, and these patterns add texture to the fabric of nursing practice today. In examining some of the future directions for practice as they related to the developing field of somnology, select trends are discussed here. Although the list is not exhaustive, the topics point to areas of current concern in nursing practice related to sleep, while highlighting opportunities for future growth. Given the current climate in health care in the United States, clearly this is no time for nursing to slumber. The opportunities for growth in clinical applications of sleep-related science to practice, research, and education are impressive and have great potential to improve health across the spectrum of health-illness and the human life span.

Public Health and Health Economics

Trends in public health and the treatment of medical and psychiatric illnesses related to sleep assessment and treatment are addressed throughout this book. There are many examples of the inextricable links between important public health problems and sleep. The obesity epidemic is a good example. As reported by the NIH, 68% of adults are either overweight or obese. Rates of obesity in children between 2 and 17 years of age range from 12.4% to 17.6% respectively. Obesity carries significant risks for mortality with more than 112,000 excess deaths due to cardiovascular disease, over

15,000 excess deaths due to cancer, and over 35,000 excess deaths due to noncancer, noncardiovascular disease per year in the U.S. population (Source: http://www.win.niddk.nih.gov/statistics/index.htm#what). Absent from these data is an appreciation of the role of dysregulated sleep relative to morbidity, mortality, and quality of life. It has been well-documented that obstructive sleep apnea syndrome (OSAS) (See Chapter 7, Sleep-Related Breathing Disorders), short sleep duration, and insomnia are highly associated with obesity, hypertension, cardiovascular diseases and morbidity and mortality. But how often do nurses in primary care or other specialty practices think about obesity or hypertension from the perspective of sleep? Commonly, medications are prescribed for hypertension and weight loss and exercise is prescribed for obesity, often in the absence of a comprehensive assessment of sleep. Similarly, nurses routinely deal with common clinical complaints of sleepiness, frequently in the presence of symptoms of sleep-related breathing disorders (See Chapter 5, Conducting a Sleep Assessment). But do nurses recognize that sleepiness is a dimension of a formal sleep disorder? How clearly do nurses understand the public health implications for safety related to sleepiness (See Chapter 22, Sleep Promotion in Occupational Health Settings)? How often do nurses mistake formal sleep disorders for other conditions such as depression (See Chapter 4, The Nature of Sleep Disorders and Their Impact and Chapter 12, Sleep and Psychiatric Disorders) or other medical conditions? There is clearly a need to retool clinical practice and align it with evidence-based approaches to sleep disorders.

Due to limited understanding about sleep disorders, nurses (and other health care professionals) may attribute symptoms of sleep disorders to etiologies unrelated to sleep. This leads to failure to treat sleep disorders. In the case of the person with OSAS and associated hypertension, the hypertension may be treated, whereas its cause is ignored. The clinical outcome is likely to be poor because the sleep disorder persists and contributes to ongoing sleepiness, poor cognitive performance, loss of quality of life, lost productivity, uncontrolled hypertension (and possible stroke), and a recidivistic pattern of treatment-seeking due to poor symptom control (Stores, 2007). The implications are overwhelming from the perspectives of clinical outcomes, public health, and cost containment.

Although studies have repeatedly documented that primary care physicians often fail to diagnose and treat OSAS (Institute of Medicine [IOM], 2006; Doghramji, 2008) and other sleep disorders, there are few empirical data related to nurses' knowledge or practice related to sleep. Nevertheless, we suspect that there is a need for greater awareness about this important issue and the need for practical strategies to incorporate sleep into nursing education and practice.

Development of approaches to the inclusion of sleep disorders assessment and treatment in practice settings is complex because it involves change across all levels of the health care system. At the level of the individual clinician, there is clearly a need for knowledge and understanding of the specific focus and meaning of sleep assessment and treatment. These clinical issues are described in depth in the earlier chapters of the book. However, these strategies must be supported within the practice setting by algorithms, guidelines, and procedures that facilitate sleep promotion. Some of these are well developed and can be directly implemented. Others remain to be developed. For example, algorithms and guidelines are available for screening and treatment of sleep disordered breathing (such as those of the American Academy of Sleep Medicine), but there are few evidence-based guidelines for sleep promotion in acute care settings, partly due to limitations in available evidence. We hope that the ideas presented in Unit III of this book contribute to the dialogue about developing meaningful algorithms. To be effective, algorithms not only need to take into account the science of sleep but also need to address the realities of clinical practice settings and variations in preparation of the health care providers who use them (e.g., staff nurse vs. advanced practice nurse; clinical specialty).

There is also a need for structured approaches to facilitate the incorporation of relevant strategies into clinical practice (e.g., computerized prompts to ask about sleep, evaluation of nurses on other providers about whether they ask about sleep). We expect that some of the ideas presented earlier in this book will guide the development of effective practical strategies to address this problem.

At the level of both public and private payors, evidence-based information needs to be incorporated into policy. There is a need for education of insurers and leaders in the industry about sleep, consultation from experts in sleep, and strategic planning for changes in policy to assure payment of services for the evaluation and treatment of sleep disorders.

Cost-benefit analyses of sleep service utilization needs to be conducted within health care organizations, but the results of the analysis may be complicated because the data for these analyses are based on current patterns of clinical utilization of sleep-related services, (e.g., sleep laboratory costs), rather than actual needed services. For example, costs may be skewed by patterns of practice that include the inappropriate and costly use of services such as polysomnography, which may not always be needed. If clinicians are not precisely identifying and treating sleep disorders, cost analyses for the utilization of these services is likely to include a wide margin of error. A good example is assessment for insomnia. Patients with insomnia may be referred for polysomnography, even though it is not needed for this condition. The consequences of these practice patterns are wasteful expenditures. Strategies to correct these problems need to include broad-based efforts to educate clinicians and to utilize evidence-based approaches to both assessment and intervention. As cost-effective and cost-conscious health care providers, nurses can provide important leadership in this arena.

Changing Demographics of the People Nurses Serve

The ethnic and racial characteristics of the United States are changing at a rapid pace. Immigration, race/ethnicity, and multiracial identification are morphing into what Lee and Bean (2004) refer to as "America's changing color lines." The notion of a predominantly biracial society is outdated because the ethnic and racial composition of United States is now heavily influenced by the influx of people from Latin, Hispanic, and Asian countries. In 2008, there were 16,028,758 foreign born naturalized citizens in the United States with an additional 21,650,834 who were foreign born but not U.S. citizens (Source: http://factfinder.census.gov/servlet/STTable). These shifts in population demand skillful approaches to cultural competency in the clinical practice of sleep.

Meleis' (2008) comment that nurses must be culturally competent to embrace the array of ethnic and cultural differences they face in practice has direct implications for nursing practice in sleep. The experience of sleep is steeped in a cultural milieu, and the nursing approach to sleep assessment and intervention must incorporate cultural perspectives. Chapter 15 addresses a number of health disparities and their implications for sleep health across diverse groups. However, there is surprisingly little research available that describes cultural perspectives on sleep. Rather, the primary focus has been on disparities in rates of sleep disorders.

A recent research experience with Brazilian immigrants highlights the need for a culturally contoured approach to the way in which questions related to sleep are asked. In this example, the use of the term bed partner was not appropriate to describe a co-sleeper. Bed partner carried negative connotations and implied sexual liaison that was not appropriate for the discussion (Phillips McEnany, Personal communication, 2010). The example was powerful, especially in light of the common use of the term bed partner in the sleep community to refer to an individual who shares the bed. In this context, bed partner does not have a sexual connotation. This lesson clearly demonstrates that semantics shape the quality of clinical exchange and ultimately, clinical outcome. But this example speaks only to an issue with two spoken words. Linguistic concerns clearly are just one slice of

cultural competence. Traditional sleep assessment often includes a variety of questions that may be perceived as deeply personal and at times, considered private information. Nurses are required to ask sensitive questions across many dimensions of health and illness. How, when, where, and who asks the questions may have powerful impact on the quality of the responses. Data provided by patients drives nursing care planning. Accurate information is essential to build effective plans of care. The broader concern relates to the impact of culturally competent care on reduction of health disparities (Harris, 2010). If patients are comfortable with the skill with which clinicians sensitively navigate clinical encounters, they are more likely to engage the services offered.

Diversity initiatives are essential for the success of health care delivery to a population of people from wide-ranging ethnic, racial and cultural backgrounds. The Institute of Medicine's 2010 *Future Directions for the National Healthcare Quality and Disparities Report* (Source: http://www.iom.edu/~/media/Files/Report) prioritizes patient and family-centered clinical approaches as an essential component of quality care. Nurses working in centers where sleep-related services are offered need to be well versed in culturally-sensitive approaches to care. This can be facilitated by supporting policy development, implementation of nationally accepted standards and diversity education provided at the level of the health care organization. The success of clinical service delivery to the culturally diverse individual who seeks assessment and treatment of sleep-related conditions is dependent on sensitive strategies to address these substantial challenges.

Evidence-Based Nursing Care Competing With Demands for High Productivity

In an era of fiscal constraint, the demand for clinical productivity often presents a challenge to high quality care. Many clinicians feel great pressure to provide more services that are usually mandated by health care administrators whose goal is the fiscal success of the organization. Models of enhanced productivity connect productivity with the number of clinical encounters. Thus, 10 minute clinical appointments are common in many practice settings today and leave little time for important communication with patients about topics such as sleep. Nurses have been concerned for many years about the implementation of policies that demand greater productivity (Abel, 1994) because of the potential negative impact on clinical care delivered.

Nurses value the quality of the clinical relationship because it is the foundation from which they facilitate healing. While *presence* may be a quality of the nurse-patient relationship, many nurses find it difficult to actualize a healing presence in a practice with compressed demands for increased service delivery. Presence is critical to nurses' work with patients who have sleep disturbances. Sleep disordered patients are often anxious about being unable to sleep well and believe that the sleep disturbance is out of their control. Control of anxiety affects the clinical outcome (Marcks, Weisberg, Edelen, & Keller, 2010). When the nurse cannot be present to adequately deal with a patient's anxiety over sleep concerns, the clinical outcome is likely to be diminished.

Time demands are likely to have a negative impact on the quality of the interpersonal exchange in the nurse-patient relationship. This phenomenon has not been studied in relation to nurses' services focused on sleep, but it is surely a factor in the exchange between patients and nurses. Often, patients complain that time with providers is too short and perceive that the available time is not possible in a system that has high demand for productivity. Does this then become a perpetuating factor in the sleep disturbance? Implementation of sleep-related practice guidelines and standards of practice can only be effective tools in a system that accommodates the time required for their implementation. Conversely, there is a need to develop sleep promotion strategies that are practical to implement in time-pressured clinical environments. Research is needed to

determine the optimal balance between time spent with patients, costs, and the need to be present with the patient.

FUTURE NURSING EDUCATION RELATED TO SLEEP AND SLEEP DISORDERS

One of the keys to addressing the important gap between growth in evidence about sleep and sleep disorders and implementation in practice is knowledge. The NIH State of the Science statement on manifestations and management of chronic insomnia (2005) and many others (IOM, 2006) identified sleep education of health professionals as a significant and pressing priority. However, efforts in nursing education lag behind the state of the science. One reason may be failure to include existing evidence-based practice guidelines for sleep disorders in curricula, but another reason may be that these guidelines do not always account for the broad-based context of nursing (vs. medicine) and have often not included nursing perspectives or considered the nature of nursing practice and the contexts of care. There is a need for further evaluation of these factors. Although data are limited to our own observations, systematic inclusion about sleep may be isolated to university settings in which there is a cadre of nurse scientists or academicians who specialize in sleep. At present, there have been few efforts to understand learning needs regarding sleep from the perspective of the discipline of nursing. Such data will be invaluable in guiding national efforts to devise a plan to meet these needs.

Huijer (2010) points out that innovations and opportunities are often born out of crisis, and nurse educators have numerous opportunities to meet nurses' learning needs in the area of sleep. The successful development and execution of such a plan will be enhanced with transformational nursing partnerships between academia and practice (Huijer, 2010). These relationships reflect principles based on recommendations from the American Association of Colleges of Nursing and include the importance of mutual goal setting, maximization of shared expertise/resources, and the enhanced benefit of the bidirectional knowledge exchange between educators and nurses in practice. Other potential benefits include opportunities for the generation of research questions and providing state of the science knowledge to support excellence in nursing practice. Academic/practice partnerships hold great promise for addressing the learning needs of both groups. For example, there is a need for practitioners and leaders in the practice setting to influence curricular needs regarding sleep and the performance of nurse graduates, but there is also a need for academic researchers to further develop and test the efficacy and effectiveness of sleep promoting interventions.

Curriculum

A major challenge to inclusion of the sleep content and learning experiences in nursing curricula is the breadth of the needs across the discipline related to sleep. Recent data reveals that over 70% of practicing nurses who were employed in clinical practice or education, prepared at the baccalaureate or masters levels and enrolled in an online sleep course ($n = 539$), had received less than 5 hours of sleep education in their entire program of nursing education (Phillips McEnany, Black, Zhang and colleagues, 2010). If, as we expect, these findings hold true for the larger U.S. nursing community, there is a pressing need for further nursing education in sleep.

Potential Approaches to Meeting Learning Needs About Sleep

In several of the chapters of this book, authors discussed a paper published by Lee and colleagues (2004), *Sleep and chronobiology: Recommendations for nursing education.* This seminal work, authored by many of the contributors to this book, includes strategies to guide the education of nurses across the undergraduate and graduate levels, recommendations for content, and learning objectives related to sleep. While there are no objective benchmarks to measure the application of the recommendations, the question arises about why the content

is not more robustly represented in nursing curricula across the United States after almost half a decade. The present is an ideal time for educators to apply the recommendations in nursing education. From a broader perspective, the core competencies suggested in the paper may be encompassed by the core competencies recommended by influential organizations such as the National League for Nursing and the American Association of Colleges of Nursing.

Table 24.3 offers suggestions for curriculum development that are critical to both the undergraduate and graduate didactic and clinical learning experiences. We recognize that space and time are limited within the curriculum, but these suggestions can be integrated within

Table 24.3 ■ *Recommendations for Nursing Curriculum Content in Sleep*

Undergraduate Classroom Content

1. Normal and developmental changes in sleep across the lifespan
2. Basic neuroanatomy related to sleep and wakefulness
3. Gender differences in sleep across the lifespan
4. Fundamental content related to sleep across medical and psychiatric comorbidity
5. Primer of the major formal sleep disorders
6. Sleep deprivation and its health-related liabilities
7. Pharmacology of sleep and wakefulness
8. Basic sleep history and assessment
9. Principles of sleep hygiene
10. Risk reduction and safety concerns related to sleep

Undergraduate Clinical Content

1. Clinical experience in an accredited sleep lab or clinic
2. Observational experience with sleep/wakefulness-related testing
3. Identification of patients at risk for sleep difficulties
4. Development of teaching plans for sleep promotion
5. Development of nursing care plans for patients with diagnosed sleep disorders
6. Utilize sleep/wake logs to calculate sleep efficiency and to address problems identified

Graduate Classroom Content

1. Advanced sleep assessment
2. Use of psychometric measures in sleep such as the Epworth Sleepiness Scale, the Insomnia Severity Index, or the Pittsburgh Sleep Quality Index
3. Advanced pharmacology of sleep and wakefulness
4. Patient referral to an accredited sleep center for further work up
5. Comparative exploration of the use of actigraphy and polysomnography in the evaluation of sleep disorders
6. Identification of risk factors associated with treatment, e.g., adherence to prescribed treatments
7. Assessment of medical and psychiatric comorbidities associated with sleep disorders

Graduate Clinical Content

1. Interpretation of a report of polysomnography
2. Development of teaching materials for patients with sleep disorders
3. Observational experience with CBT-I
4. Engage in interventions related to treatment adherence, e.g., CPAP
5. Participate in pharmacology consultation with the patient diagnosed with a sleep disorder
6. Family teaching related to sleep promotion

Source: "Sleep and Chronobiology Recommendations for Nursing Education," by K. A. Lee, C. Landis, E. R. Chasens, S. Merritt, K. P. Parker, N. Redeker, et al., 2004, *Nursing Outlook*, *52*(3), pp. 126–133. Adapted with permission.

existing curricula and courses. Case studies and critical thinking questions provided in this book are adaptable for use in various levels of curriculum. Content and learning experiences can also be included in specialty courses (e.g., psychiatric mental health, medical-surgical nursing) around sleep issues specific to these settings. Much of this information is detailed in Unit III of this book.

We recognize that it is necessary to identify the appropriate level of content and experiences consistent with the level of the curriculum (prelicensure, graduate). However, until basic information is universally available at the undergraduate level, it will be necessary to include basic information even in graduate programs. Continuing education on sleep is also needed to provide ongoing learning about emerging evidence.

Evidence-based guidelines (EBP), such as those developed by the American Academy of Sleep Medicine (AASM) (http://www.aasmnet.org) and others (some of which are yet to be developed) may be useful in guiding student learning experiences about sleep. The use of EBP strategies with nursing students has benefits, including greater confidence in clinical decision making and clinical preparedness for the work of a professional nurse (Brown, Kim, Stichler, Fields, 2010). Students exposed to EBP in nursing school are more likely to rely on the use of evidence based approaches after graduation. In the future, nursing practice will be powerfully driven by evidence-based information, and sleep-related evidence will provide foundations for the nurse educator to begin the process of including sleep content at all levels of nursing education. We hope that nurses prepared with doctorates in nursing practice might collaborate with nurse scientist to develop and test practice guidelines for use in nurse-intensive settings such as acute/critical care or long-term care.

Meeting the learning needs of the large numbers of nurses who require education to retool their perspectives on sleep, health, and illness comes in an age when technologies allow for real time global exchange. Family, professional, social, and economic demands are increasing for many Americans today, including nurses, and many educators recognize that these changes are critical indicators of the need to create user-friendly learning at all levels of nursing education. Weekend courses, blended online and in-class strategies, full online learning and use of lecture-capturing technologies all allow student access to education through updated teaching/learning strategies. All of these strategies might be useful in providing education about sleep.

Consistent with global trends in education (Bhutta et al., 2010), each of the chapters in this book provides links to sites located on the world wide web, providing resources to anyone in any part of the globe who has access to a computer terminal or a hand held device capable of accessing the web. Newer technologies available to educators allow for classrooms that include students from across the globe, adding to the diversity of the classroom and certainly bringing the discussions in the class to a truly global level. An example is an online international educational initiative developed by one of the authors of this chapter (GPM) and a group of colleagues at the University of Massachusetts Lowell in partnership with the American Psychiatric Nurses Association and The International Honor Society for Nursing, Sigma Theta Tau. They provided a 36-hour introductory course on sleep and chronobiology. Twenty five hundred spots were available. Students from 8 countries and 48 states earned continuing education credits. The high demand for this course underscores the pressing needs for accessible sleep education and the appeal of the international classroom. There is a great need to think outside of the box when conceptualizing new approaches to nursing education, including distance education. This is no less true of sleep education.

CONCLUSION

The future of sleep-related science holds great relevance and provides exciting directions for the discipline of nursing and nursing practice and education. While the contributions of

nurses to sleep science are growing, the application of the science to practice and pedagogy lags behind scientific progress. There is a need for a structured and multi-pronged approach that addresses these gaps. This includes structured learning activities and content within all levels of nursing curricula, continuing education of practicing nurses who did not have content on sleep in their formal education, the use of innovative teaching/learning technology, and formal practice guidelines for clinical settings, with systems in place to assure their utilization, including reimbursement. The effort will require creativity, dedication, strategic planning and successful interdisciplinary collaboration, as well as collaborations within nursing.

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Critical Thinking Questions

CHAPTER 1
PHYSIOLOGICAL AND BEHAVIORAL ASPECTS OF SLEEP

1. As a nurse, you work with people who routinely deal with sleep disturbances, and teaching is a critical dimension of the care. For the patient who naps routinely throughout the day, how would you translate concepts from the Two Process Model in helping her/him to understand the impact of napping on homeostatic drive for sleep?
2. You are teaching a group of undergraduates about the neurochemistry involved in sleep and wakefulness. How would you respond to a student's question about how clonazepam (a benzodiazepine) influences the sleep/wake on-off switch?
3. You have just offered a lecture on sleep at a community health center for a group of patients from general medical practices. In the talk, you discussed measures of sleep including polysomnography (PSG) and actigraphy. A woman raises her hand to say that she is scheduled for PSG evaluation but "I know that I can't sleep in a strange bed in a clinic" and wants to know why she can't wear an actigraph for a few days. Outline your response and rationale.
4. A colleague is forced to work the night shift for 14 days every 6 months. During those 2 weeks her sleep is very poor and she is quick to note that "I am not a night person at all." She has diabetes and while she is careful about her diet and adherence with medication during the night shift cycles, she wonders why her blood sugar levels are less stable during these times. How might this be associated with sleep physiology?

CHAPTER 2
DEVELOPMENTAL ASPECTS OF SLEEP

1. As a school nurse, the father of a 13-year-old student contacts you seeking consultation about his son's sleep. He had received a report from one of the student's teachers stating that the student was dozing off in class. The father states that he and his son argue routinely "because he won't go to bed by 10 p.m. and then I can't get him up in the morning…now he's falling asleep in classes!" How would you help the father to understand the driving forces behind the sleep difficulties and what would you recommend?
2. You are functioning as a nurse practitioner in a primary care office. A 37-year-old woman comes in for a routine physical examination and she complains of extreme sleepiness. She is a single mother of three small children and is working a full-time job and two part-time jobs to make ends meet. Her mother helps care for the children. She states that she can't stop working this much but wants your advice on how to help her feel more refreshed.
3. An elderly man approaches you because he trusts your advice as a nurse. He reports that he wishes that he could sleep "like when I was 40" and worries about not getting adequate sleep. As a solution to his self-perceived poor sleep, he takes two tablets of acetaminophen with diphenhydramine nightly. His anxiety about sleep is high. What is your plan of care?

4. In examining sleep across developmental stages, what factors do you consider reliable indicators of poor sleep outside of self-reports?

CHAPTER 3
GENDER AND SLEEP

1. The sleep architecture of women varies across the follicular and luteal phases of the menstrual cycle. How would you translate these sleep-related changes into an explanation of the lived experience of "feeling really tired" in the late luteal phase? Specifically, what happens to sleep during this time?
2. A 46-year-old man has been placed on an antiestrogen drug, tamoxifen, which is usually used to treat breast cancer. His clinician has prescribed it as an off-label treatment for a specified condition. He is now complaining of persistent insomnia. What could the underlying mechanism be with the tamoxifen and insomnia?
3. Sleep changes for women in the perimenopausal transition. Given this reality, what are the dimensions of a thorough assessment for women in this period, given the concurrent health-related risks?
4. There is an established link between sleep and testosterone. What are the potential impacts on sleep related to overuse of androgen hormone replacement?

CHAPTER 4
THE NATURE OF SLEEP DISORDERS: DIAGNOSTIC AND EPIDEMIOLOGICAL CONSIDERATIONS

1. How would you differentiate sleep symptoms associated with mental disorders from primary sleep disorders?
2. You are planning a community health program to raise awareness about the importance of sleep loss. Which specific groups would be your highest priority?
3. Which groups are at the highest risk for injury related to sleep loss?
4. You are a faculty member teaching an undergraduate class on sleep. In the class, you want to help students think about assessment in creative ways. Discuss your teaching plan related to clinical assessment of and interventions with excessive daytime sleepiness.

CHAPTER 5
CONDUCTING A SLEEP ASSESSMENT

1. Recognizing that there is a bidirectional relationship between sleep and medical/psychiatric disorders, how does the nurse determine the cause of the sleep disturbance and select which condition should be addressed first?
2. What are three major risk indicators in children that would indicate the presence of a formal sleep disorder?
3. How would you approach a sleep assessment of an individual who is from a culture that is unfamiliar to you? What are some of the issues that face you as a clinician in the provision of an accurate and thorough assessment?
4. Discuss clinical circumstances that would indicate the need for referral to a sleep specialist.

CHAPTER 6
INSOMNIA

1. As a nurse, you are assessing patients who have insomnia. How would you distinguish primary insomnia from a secondary insomnia related to subclinical depression?
2. A patient arrives in your practice or clinic and states "I really need your help. I have *literally* not slept in the last six days, not for one minute." How would you respond? What would you do?
3. Hypertension and diabetes are relatively common diseases in the United States. If you were a nurse in a cardiology or endocrine practice, how would you consider the nature and course of these diseases from a perspective of sleep?
4. A patient tells you that they are "incredibly sleepy" due to poor sleep. What obligations do you have to the care of this person related to safety?

CHAPTER 7
SLEEP-RELATED BREATHING DISORDERS

1. Obstructive sleep apnea does not occur exclusively in people who are obese. In the

absence of obesity, discuss presenting symptoms that would be suspicious of apnea in a thin person.
2. In the absence of examining symptoms through a lens that includes recognition of sleep-related breathing disorders, many people receive hypnotic medications because their presenting symptoms may be nonrestorative sleep or poor sleep. Discuss when the use of hypnotics would be appropriate in someone with sleep-related breathing disorder.
3. One of the major challenges to the treatment of sleep apnea is adherence to the prescribed regimen of CPAP. What measures would you take to facilitate adherence? How would you know if your strategy was effective?
4. What clinical measures can you utilize to accurately assess cognitive functions, especially memory and executive functions?

CHAPTER 8
MOVEMENT DISORDERS

1. RLS, PLMS, and PLMD share some similarities in symptom presentation. Symptoms may be related to disease or iatrogenic. Discuss three conditions (e.g., medications) under which abnormal movements may occur and identify groups of patients who may be vulnerable to abnormal movements related to these circumstances.
2. Dopamine agonists are commonly used in the treatment of RLS. Discuss a common side effect of this class, discuss its implications for quality of life, and develop a nursing approach to dealing with the medication-related difficulty.
3. Self-report and the report of bed partners are important in the assessment of movement disorders. Consider the case of a person with mild to moderate dementia who sleeps alone. How would you approach assessment of this individual?
4. A mother reports to you that her child has night terror. Discuss the main points of your discussion with her and outline your recommendations for dealing with her child's symptoms.

CHAPTER 9
NARCOLEPSY

1. Hypnagogic hallucinations and sleep paralysis are often very frightening experiences. Role play a discussion that you would have with a person who reports these symptoms but has not yet been diagnosed with narcolepsy.
2. Mr. K, a 22-year-old man, has just been diagnosed with narcolepsy. Develop your teaching plan for him. Address ways in which you would include evidence-based information in your discussion with him.
3. As part of your teaching plan, you want to help Mr. K understand the pathophysiology of narcolepsy and how stimulants work to relieve symptoms. Apply information from Chapter 1 (Physiological and Behavioral Aspects of Sleep) to your teaching points.
4. Modafinil and methylphenidate SR are commonly used medications in the treatment of narcolepsy. Discuss the differences in mechanism of action and how these mechanisms equally address the presenting symptoms of narcolepsy.

CHAPTER 10
CIRCADIAN RHYTHM DISORDERS

1. You are working with an elder who describes a phase advanced sleep/wake pattern. She complains of the pattern because it interferes with the time she spends with her family when everyone is home in the evening. Develop a nursing care plan to adjust the pattern to align her wakefulness with her desired schedule.
2. Mr. S is a nurse who routinely works the 3 p.m.-11 p.m. shift on a transplant unit. Three nurses recently resigned their positions to return to school and two of these nurses worked the 11 p.m.–7 a.m. shift. Mr. S has been working overtime, often doing a double shift from 3 p.m.–7 a.m. He complains that his sleep is "a mess," with difficulty sleeping during the day and sleepiness during the time that he is awake. The schedule is erratic. How would you advise him to improve his sleep?

3. What circadian-based conditions are appropriately assessed with polysomnography?
4. Light therapy is often used in the treatment of select circadian rhythm disorders. Develop a teaching plan that outlines the physiological mechanism by which this intervention works.

CHAPTER 11
SLEEP AND CHRONIC MEDICAL CONDITIONS

1. Develop a plan of nursing care focused on sleep for the person with chronic but stable heart failure.
2. A patient was recently diagnosed with obstructive sleep apnea syndrome and diabetes simultaneously. In his bewilderment, he asks you about which condition came first. Discuss your response, addressing the physiological interrelationship between the two conditions.
3. Ms. Z is a 44-year-old woman who has been diagnosed with breast cancer. She was treated with a mastectomy and reconstruction and chemotherapy (recently concluded) but is now undergoing a course of radiation therapy. She is taking tamoxifen. She complains of difficulty falling asleep and staying asleep. Her energy is low during the day. Outline the potential factors related to her sleep disturbance and develop a nursing approach to the same.
4. Apply the concepts discussed in this chapter related to the assessment of sleep for a practicing nurse in a hospital palliative care unit.

CHAPTER 12
SLEEP AND PSYCHIATRIC DISORDERS

1. Mr. L has a diagnosis of dysthymia and generalized anxiety disorder. He is terrified of not getting sleep at night because it affects his work as an executive. Outline a plan of nursing care to address his presenting symptoms.
2. Role play a discussion that responds to a patient's question "Someone told me that sleep disturbances in bipolar illness can be compared to chest pain in cardiac illness. Is this true and if so, how?"
3. You are working with a patient who has panic disorder with nocturnal panic attacks. She or he has been treated with fluoxetine (20 mg a day), which resolved the panic but has caused iatrogenic sleep disturbance with fragmented, nonrestorative sleep. What is your plan to address these issues?
4. Ms. O has bipolar I disorder that is well controlled on lithium carbonate. She has difficulty falling asleep during the late luteal phase of her menstrual cycle and heard that trazodone would be a good medication for sleep because she is "afraid of getting addicted to clonazepam-like drugs." How would you advise her?

CHAPTER 13
CHILDHOOD SLEEP DISORDERS

1. Charlie is a 2-year-old who has limit-setting insomnia. His parents have been instructed to use extinction as the best technique to address the problem. His father cannot tolerate hearing his son screaming during the extinction intervention. How would you advise him?
2. Eduardo is an 18-year-old freshman in college, living in the dorms. His sleep is characterized by a phase delay. He is failing in the 8 a.m. calculus class because he is routinely oversleeping. Develop a plan to address his sleep problem.
3. You are offering community education classes and have been asked to prepare a class for grammar school teachers on sleep. What are your key points and how would you address them in the class?
4. One of the teachers from the class approaches you and asks you how she can engage the children in learning about sleep. She is particularly used to online learning tools. How would you advise her?

CHAPTER 14
COMPLEMENTARY/ALTERNATIVE THERAPY AND SLEEP DISORDERS

1. Mr. V is a 31-year-old man who is in recovery treatment for alcoholism. He wants to avoid medications for his fragmented sleep and daytime sleepiness. His provider recommended

acupuncture. He spoke with you and said "Are you sure that this acupuncture isn't just some form of placebo?" Formulate your response as to its effectiveness.
2. A patient is interested in using herbal remedies for insomnia but wants to know about potential drug interactions between the herbals and prescribed medications. Formulate a specific plan to address the patient's question.
3. A patient approaches you to say that he has visited a traditional Chinese herbalist who prepared a mix of herbs and instructed him to make a tea with the herbs and to drink one cup of the tea each night for insomnia. He wants to know if he should take it. What's your advice?
4. You are working with someone who has chronic pain and secondary insomnia. She very much wants to try Hatha yoga but is concerned about its safety for her, given her pain. How would you assess the appropriateness of this modality for her?

CHAPTER 15
HEALTH DISPARITIES IN SLEEP DISORDERS

1. You are a nurse working in an urban primary care clinic. A Laotian man comes in for evaluation of "a sleep problem." He recently broke his arm during sleep and fears that it is the result of a spiritual problem. You suspect REM sleep behavior disorder. He is Hmong and wants a Shaman involved in his care. You've never heard of the Hmong people and have had no contact with Shamans. How would you proceed?
2. There is a large community of Brazilians who are immigrants in your community. Many of them work three jobs to support their families here and in Brazil. Sleep patterns in these people are severely fragmented and their daytime function reflects this fact. Discuss your plan of care.
3. Workups for obstructive sleep apnea syndrome (OSAS) are being performed on a number of the Brazilians noted in the previous question. They commonly use *maracuja* (passiflora, common to the Amazon region) for sleep. How would you determine its safety in those with suspected OSAS?
4. What are the lived experience implications of the documented reductions in stage 3 non-REM sleep in African Americans?

CHAPTER 16
SLEEP AND THE CHILDBEARING FAMILY

1. A nursing mother has been diagnosed with postpartum depression. She has been given a prescription for citalopram (a selective serotonin reuptake inhibitor) and wants to know if the drug enters her milk and if so, is this safe for her baby. What's your advice?
2. Ms. Valdez and her husband recently had a healthy baby boy. They are at their first checkup for the baby and both parents complain of "terrible sleep" as they both take turns with feeding the baby during the night. How would you advise them on making a plan to enhance sleep for both of them during the baby's first months at home?
3. A new mother with a past history of major depression relapsed with depression 2 weeks after the baby was born. The mother has been successfully treated with fluoxetine in the past and it has been recommended that she resume the medication now. She is breastfeeding and is reluctant to take medication because she wants to continue breastfeeding. She asks for your advice on what to do. How would you counsel her on this issue?
4. How would you advise parents when they seek your advice about having the baby co-sleep with them? What is your clinical reasoning? How would you ensure cultural competence in your response?
5. Ms. West is in her 29th week of pregnancy with twins. Although everything is going well, she complains of sleep problems related to the fact that she cannot get comfortable in bed, given her size at this point in the pregnancy. What suggestions can you make?

CHAPTER 17
SLEEP IN CHILDREN'S HEALTH CARE SETTINGS

1. Yogendra is a 9-year-old girl who is being treated for cystic fibrosis at a leading medical center where she is receiving an experimental

treatment for her condition. She and her father have come from her home in Budapest for her to receive these specialized treatments. She has difficulty falling asleep and staying asleep. When she awakens during the night, she often has periods of crying. She has not been eating. Her father reports that these behaviors are very unusual for her. You suspect depression. How would you assess if your suspicion about depression is correct?

2. Jack is a 12-year-old boy who is at a summer camp for children who have recently been diagnosed with type 1 diabetes. The counselor assigned to his cabin notes that he stays up past the scheduled bedtime, and then sleeps during the night but he is excessively sleepy during the day. How would you assess the nature of the sleepiness and what is your plan of action?
3. You are a graduate student in a pediatric outpatient clinical practicum and you decide to develop materials for the waiting room on sleep. Identify three topics that are important to sleep in children and outline the content for these materials.

CHAPTER 18
SLEEP AND PRIMARY CARE OF ADULTS AND OLDER

1. You administer the Epworth Sleepiness Scale to an older adult who complains of disturbed nighttime sleep. The client scores 14 and indicates that he has occasionally fallen asleep when driving. When you ask how he got to today's appointment, he says he drove in by himself. What is your first action?
2. A 44-year-old man has recently moved from another state and comes for his first visit with his new primary care provider. The patient is being treated for depression and has been given bupropion SR 150 mg twice a day and quetiapine 50 mg at bedtime due to the activating effects of the bupropion SR. Since he started this regimen 11 months ago, he has gained 32 pounds. Outline your comprehensive assessment and intervention plan for this patient.
3. In your practice, you frequently use the Insomnia Severity Index (ISI) and the Epworth Sleepiness Scale (ESS). Ms. Haddad comes in for evaluation but only speaks Arabic. Through a family member, she reports middle of the night awakening and significant sleepiness during the day. You would like to complete the ISI and ESS because it would enhance your assessment. How could this be done while ensuring that the psychometric properties of the instruments are maintained?
4. A patient comes in for a routine checkup and asks you "What sleeping medicines are best?" She notes that a friend of hers received a prescription for gabapentin for sleep from her primary care provider, but when she checked the drug out on Google, she found that it is a medicine used to treat seizures. How would you respond to her question? What are the circumstances in which an off-label use of a medication could be a preferred substitute for a benzodiazepine receptor agonist?

CHAPTER 19
SLEEP IN PSYCHIATRIC-MENTAL HEALTH SETTINGS

1. Hypersomnolence is not an uncommon feature of some types of bipolar illness. In a person with bipolar illness, how would you determine if the hypersomnolence were from the mood disorder or a formal sleep disorder?
2. Sleep and psychiatric illnesses share a very strong bidirectional relationship. Given that many of the antidepressants are not "sleep-friendly," what measures could be taken to reduce the potential morbidity associated with the use of sleep-disturbing drug interventions to treat the illness?
3. Many of the psychotropic drugs have sleep-related movement disorders associated with their use. Prepare a presentation to be given to staff nurses on an inpatient psychiatric unit to alert them to this issue. Develop assessment measures that the nurses can use in their practices.

4. Insomnia is often treated with medications but not with adequate behavioral measures. This pattern leads to the perpetuation of prescription drug use, which has a cost burden associated with it. How would you address this issue with a group of hospital administrators who are interested in cost-saving pharmacoeconomic measures?

CHAPTER 20
SLEEP IN ADULT ACUTE & CRITICAL CARE SETTINGS

1. What is the impact of alcohol on sleep architecture? How would you incorporate this information into a teaching plan? How would you use this information to help the patient understand the lived experience of nonrestorative sleep associated with alcohol use?
2. A 24-year-old woman is admitted for management of a severe flare of lupus. She is receiving high doses of steroids to reduce the symptoms and consequently she is sleeping about 2 hours a night. What is your intervention plan? If medication is indicated, discuss the mechanism of action of the drug. Does this drug affect the sleep/wake switch?
3. A 56-year-old man has been admitted for neurosurgery to repair nerve impingement from a compressed nerve at the level of L3. He has significant neuropathic pain and was initially prescribed amitriptyline for pain management. He stopped this medication because of severe daytime sleepiness. His physician then prescribed gabapentin. The patient had great pain relief but was again excessively sleepy during the day. What are your teaching and intervention plans for this man?

CHAPTER 21
SLEEP IN ADULT LONG TERM CARE

1. Mr. Balakas is a 72-year-old man who has dementia of the Alzheimer's type and he resides in a long-term care facility. He has significant sleep apnea and is treated with CPAP. The nurses are concerned because he is unable to tolerate the CPAP and removes it within an hour after he goes to bed. His apnea/hypopnea index is 30. What is your plan to address this problem?
2. Mrs. Milton is a resident in a long-term care facility and has severe chronic obstructive pulmonary disease and congestive heart failure. She describes her sleep as "abysmal" and sleeps in short periods of time. In her sleep-deprived state, her irritability escalates and she often yells at other residents in the facility in addition to staff. What can be done to enhance the quality of this woman's sleep? What evidence base is available to support your plan?
3. Increased risk for falls is associated with untreated insomnia. Yet despite this evidence-based fact, family members of a resident in a long-term care facility demand that their family member *not* receive hypnotics. The resident had received a high-dose hypnotic on one occasion and became confused and combative, and the family fears that this will occur again. You are meeting with the family to discuss the issue. Outline your discussion points for the family meeting.

CHAPTER 22
SLEEP IN OCCUPATIONAL HEALTH SETTINGS

1. In an industry that requires 24-hour service, what is an optimal schedule of operations that achieves the highest duration and quality of sleep for the most workers?
2. In a safety-sensitive industry that has not adapted regulation to promote alertness in workers, what should administrators do to ensure fitness for duty with respect to having had adequate sleep before the work shift?
3. Should napping be allowed for night shift workers? If so, how could it be implemented and still maintain production of work with an adequate margin of safety?
4. If a worker in a safety-sensitive industry is found to have obstructive sleep apnea and is prescribed CPAP, what are the employer's and employee's responsibilities regarding adherence to the treatment?

5. A radio newscaster was fired and denied long-term disability payments when he claimed shift-work sleep disorder after having to work overnight shifts and do frequent shift rotation when the station began to broadcast 24 hours a day. Should he be granted accommodations under the Americans with Disabilities Act, and what should they be?

CHAPTER 23
NURSES' ROLES IN SPECIALIZED SLEEP CENTERS

1. An APRN is working in a sleep center and decides to start a CBT-I group. What selection or exclusion criteria should be used in determining the group membership? Who is a good candidate for group CBT-I?
2. You are a nurse in a sleep center and you have been invited to give a presentation to a group of elders living in an elder community. What are the main points of your presentation to this group and why?
3. As a nurse specializing in sleep, a local university has asked you to join their faculty advisory board to consult with them about curriculum development at the master's/DNP level. How would you approach the consultation? What resources would guide your work with the faculty members?
4. You are an APRN working in primary care and would like to work with your employer to develop a plan to implement an interdisciplinary training program on the fundamentals of sleep. What levels of the organization would you engage in your plan and what strategy might you use to advance the initiative?

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