

## UNLOCK THE SECRETS OF SLEEP

A Physician's Introduction to the Field of Sleep Medicine



THIS BOOK IS THE FIRST PART OF A FREE, INTRODUCTORY SERIES FOR PHYSICIANS FROM THE AMERICAN ACADEMY OF SLEEP MEDICINE.

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# SECRETS

#### Today we know more than ever before about the physiological processes that occur during sleep. However, sleep's essential purpose continues to elude us.

We understand that sleep impacts the function of all organ systems and is essential for cellular recovery, energy conservation and memory consolidation.<sup>1,2</sup> As one of the pillars of health, sleep is a necessity for individual well-being, population health and public safety.<sup>3</sup> Sleep inspires us to dream, and it fuels our waking ambitions.

But why? The answer to this fundamental question remains mysterious.<sup>4</sup>

#### In fact, you could argue that the question of why we sleep is medicine's greatest mystery.

Because of its secretive nature, sleep has always been a subject of fascination, preoccupying the minds of physicians, philosophers, painters and poets. It has infused the work of artists ranging from Shakespeare to Van Gogh, and from Dickens to Disney screenwriters.<sup>5-8</sup>

References to sleep disorders in medical writings go back centuries.<sup>9</sup> However, it wasn't until the mid-20th century that the multidisciplinary field of sleep medicine began to take root. The publication in 1953 of a study identifying rapid eye movement sleep, or REM sleep, sparked an age of discovery that continues today.<sup>10</sup>

Now sleep medicine is a recognized medical subspecialty with fellowship training programs that are approved by the Accreditation Council for Graduate Medical Education (ACGME) and a board certification examination that is administered by six member boards of the American Board of Medical Specialties (ABMS). More than 6,000 board-certified sleep medicine physicians are providing patient-centered care across the U.S., and more than 2,500 sleep disorders centers are accredited by the American Academy of Sleep Medicine (AASM). But this is only the beginning.

With millions of people throughout the U.S. suffering from a disorder of sleep and daytime alertness, many more sleep specialists are needed. Therefore, a new generation of sleep physicians is rising up.

Leading sleep teams of other health care providers – including nurses, physician assistants, psychologists and sleep technologists – and collaborating with primary care physicians and other specialists, they are striving to ensure that everyone has access to high quality sleep health care. These physicians are leveraging new technological advances to improve sleep diagnostics and therapies, and they are using telemedicine to open new avenues for sleep medicine expertise.

#### Can we solve the mystery of sleep? Perhaps you hold the key.

Continue reading to discover how you can unlock your medical career in the field of sleep medicine.

# SITUATION OF THE STATISTICS

## **70 MILLION**

Americans suffering from sleep problems. Nearly 60% of them have a chronic disorder.

(NCSDR)

## 30-35%

Global population affected by transient insomnia symptoms. The full clinical syndrome of chronic insomnia disorder occurs in about 10% of people.

(ICSD-3)

## \$63.2 BILLION

Estimated cost in lost work performance each year in the U.S. associated with insomnia.

(SLEEP)

## **69%**

U.S. high school students that fail to get the recommended 8 to 10 hours of sleep per night.

(CDC)

## 35%

U.S. adults that fail to get the recommended 7 or more hours of sleep per night.

(CDC)

## 6,400

Estimated total of fatal crashes caused by drowsy driving in the U.S. each year.

(AAA Foundation for Traffic Safety)

#### COUNTY-LEVEL SHORT SLEEP DURATION (<7 HOURS) AMONG ADULTS: US, 2014



#### **UNDIAGNOSED SLEEP APNEA: A HIDDEN HEALTH CRISIS**

In the U.S. the estimated economic cost of undiagnosed obstructive sleep apnea was nearly \$150 billion in 2015.



Source: American Academy of Sleep Medicine, 2016 | www.sleepeducation.org

## SLEEP DISORDERS

The International Classification of Sleep Disorders, published by the American Academy of Sleep Medicine, serves as a guide to clinicians in the identification of specific disorders of sleep and wakefulness. As is the case with many diagnostic systems, our current knowledge and understanding of the pathophysiology of many sleep disorders is inadequate. As a result, the ICSD employs a hybrid approach that utilizes pathophysiology, where known, but also relies heavily on phenomenology and organ system approaches.

#### THE ICSD CLASSIFIES SLEEP DISORDERS IN SIX MAJOR CLINICAL DIVISIONS:

#### 1. Insomnia

These sleep disorders are characterized by a persistent difficulty with sleep initiation, duration, consolidation, or quality that occurs despite adequate opportunity and circumstances for sleep, and results in some form of daytime impairment.

#### 2. Sleep Related Breathing Disorders

These disorders are characterized by abnormalities of respiration during sleep. They are grouped into obstructive sleep apnea (OSA) disorders, central sleep apnea disorders, sleep related hypoventilation disorders, and sleep related hypoxemia disorder.

#### 3. Central Disorders of Hypersomnolence

This is a group of disorders in which the primary complaint is daytime sleepiness not caused by disturbed nocturnal sleep or misaligned circadian rhythms. In this nosology, the term hypersomnolence is used to describe the symptom of excessive sleepiness, whereas hypersomnia refers to specific disorders, such as idiopathic hypersomnia.

#### 4. Circadian Rhythm Sleep-Wake Disorders

These disorders are caused by alterations of the circadian time-keeping system, its entrainment mechanisms, or a misalignment of the endogenous circadian rhythm and the external environment. Circadian rhythms are endogenous, near-24-hour biological rhythms that exist in all living organisms. The internal near-24-hour circadian clock is entrained or synchronized to the 24-hour light-dark cycle.

#### 5. Parasomnias

These disorders are undesirable physical events or experiences that occur during entry into sleep, within sleep, or during arousal from sleep. They encompass abnormal sleep related complex movements, behaviors, emotions, perceptions, dreams, and autonomic nervous system activity.

#### 6. Sleep Related Movement Disorders

These disorders are primarily characterized by relatively simple, usually stereotyped, movements that disturb sleep or its onset. Nocturnal sleep disturbance or complaints of daytime sleepiness or fatigue are a prerequisite for a diagnosis.

The following pages comprise brief summaries describing one sleep disorder from each of these six groups.



#### **Chronic Insomnia Disorder**

Insomnia Clinical Division

The essential feature of chronic insomnia disorder is a frequent and persistent difficulty initiating or maintaining sleep that results in general sleep dissatisfaction. The sleep complaint is accompanied by distress about poor sleep and/or impairment in family, social, vocational, academic, or other important areas of functioning. Furthermore, the sleep disturbance and associated waking symptoms occur despite having adequate time and circumstances each night to obtain necessary sleep. Chronic insomnia disorder can occur in isolation or comorbidly with a mental disorder, medical condition, or substance use.

Symptoms during wakefulness accompany the sleep difficulties and result in the impairment of normal functioning. Common waking symptoms include fatigue; reduced motivation; reduced concentration, attention, and memory functioning; and irritability or reduced mood. Complaints of subjective daytime sleepiness are also common, although, in contrast to patients with hypersomnolence conditions, many with this complaint are not able to nap in the daytime and few show unintentional sleep episodes. Reports of reduced performance at work or school or impaired social functioning also are common.

#### DEMOGRAPHICS

The full clinical syndrome of chronic insomnia disorder occurs in about 10% of the population, but the prevalence of transient insomnia symptoms is much higher (30% to 35% of the population). Chronic insomnia disorder is more common in women, those with medical/psychiatric/substance disorders, and in people in lower socioeconomic strata. It may occur at any age but is more commonly diagnosed in older adults.

#### PATHOLOGY & PATHOPHYSIOLOGY

Studies of the pathophysiology of chronic insomnia disorder have focused on one or more dimensions of physiological hyperarousal during sleep and wakefulness. Collectively these studies suggest increased physiological arousal among individuals with insomnia. These studies imply heightened activity of the sympathetic nervous system and hypothalamic-pituitary-adrenal axis across sleep and wakefulness that is thought to perpetuate sleep/wake dysfunction.

#### TREATMENT

Avoid use of hypnotics as primary therapy for chronic insomnia in adults; instead offer cognitive-behavioral therapy (CBT), and reserve medication for adjunctive treatment when necessary. CBT for chronic insomnia involves a combination of behavioral modification, such as stimulus control and sleep restriction, and cognitive strategies, such as replacement of unrealistic fears about sleep with more positive expectations.<sup>11</sup>

#### **UNRESOLVED ISSUES**

Much remains to be learned about the underlying pathophysiological pathways leading to insomnia and whether insomnia should be classified as a unitary disorder or subdivided into several subtypes.



#### **Obstructive Sleep Apnea (OSA)**

Sleep Related Breathing Disorder Clinical Division

OSA is characterized by repetitive episodes of complete (apnea) or partial (hypopnea) upper airway obstruction occurring during sleep. These events often result in reductions in blood oxygen saturation and are usually terminated by brief arousals from sleep. By definition, apneic and hypopneic events last a minimum of 10 seconds. Most events are 10 to 30 seconds in duration but occasionally persist for one minute or longer. Oxygen saturation usually returns to baseline values following resumption of normal breathing but may remain low if the apneic or hypopneic events are very frequent and prolonged, or if there is underlying pulmonary pathology.

Excessive sleepiness is a major presenting complaint in many but not all cases. The sleepiness is most evident during relaxing or inactive situations. In women, excessive sleepiness is a less prominent complaint.

#### DEMOGRAPHICS

OSA can occur in any age group. Estimates of prevalence are very dependent on how sleep related respiratory events are defined, as well as their frequency and other criteria used to define disease. Nevertheless, general populationbased studies from a number of countries indicate that OSA associated with daytime sleepiness occurs in 3% to 7% of adult men and 2% to 5% of adult women.

OSA should be approached as a chronic disease requiring long-term, multidisciplinary management.

#### PATHOLOGY & PATHOPHYSIOLOGY

Patients with OSA commonly have reduced crosssectional area of the upper airway lumen due to either excessive bulk of soft tissues or craniofacial anatomy, or both. During inspiration, negative pressure is generated in the lumen of the upper airway, promoting closure. However, pharyngeal dilating muscles act to maintain patency. The activity of these muscles decreases with sleep onset, but is normally adequate to maintain an open airway. In persons with OSA, the activity of the pharyngeal dilating muscles becomes insufficient to prevent narrowing and/or closure of the upper airway.

#### TREATMENT

OSA should be approached as a chronic disease requiring long-term, multidisciplinary management. Positive airway pressure (PAP) is the treatment of choice for mild, moderate, and severe OSA and should be offered as an option to all patients. Alternative therapies may be offered depending on the severity of the OSA and the patient's anatomy, risk factors, and preferences.<sup>12</sup>

#### **UNRESOLVED ISSUES & FURTHER DIRECTIONS**

Although substantial evidence implicates OSA as a risk factor for coronary artery disease and stroke, it has not been clearly demonstrated that treatment mitigates this risk. Although there is a clear association between OSA and type 2 diabetes mellitus, further studies are needed to determine whether OSA is an independent risk factor and what mechanisms are involved.



#### Narcolepsy Type 1

Central Disorders of Hypersomnolence Clinical Division

Narcolepsy type 1 is a disorder primarily characterized by excessive daytime sleepiness and signs of REM-sleep dissociation, the most specific of which is cataplexy. It has now been firmly established that narcolepsy type 1 is caused by a deficiency of hypothalamic hypocretin (orexin) signaling. Patients with low or undetectable concentrations of hypocretin-1 in the CSF compose a specific disease population with a single etiology and relatively homogenous clinical and polysomnographic features. Patients with sleepiness and low or absent CSF hypocretin-1 levels are classified as having narcolepsy type 1, even if they do not manifest cataplexy.

Patients with narcolepsy type 1 experience repeated daily episodes of an irrepressible need to sleep or lapses into sleep. Most patients awaken refreshed after a sleep episode but begin to feel sleepy again after variable times. Sleepiness generally has a serious impact on the ability of the patient to function in educational, social, and occupational situations.

#### **DEMOGRAPHICS**

Narcolepsy with cataplexy occurs in 0.02% to 0.18% of the United States and western European populations. A lower prevalence has been reported in Israel, whereas narcolepsy with cataplexy may be slightly more common in Japan (0.16% to 0.18%). Both sexes are affected, with a slight preponderance of males.

Narcolepsy type 1 is caused by deficiencies in hypocretin signaling.

#### **PATHOLOGY & PATHOPHYSIOLOGY**

It is now firmly established that narcolepsy type 1 is caused by deficiencies in hypocretin signaling, most likely due to a selective loss of hypothalamic hypocretin producing neurons. Several animal models lacking hypocretin neurotransmission demonstrate narcolepsy, indicating a causal relationship.

#### TREATMENT

A major objective of treatment of narcolepsy should be to alleviate daytime sleepiness and control nocturnal symptoms of disrupted sleep. Most of the agents used to treat excessive sleepiness have little effect on cataplexy or other REM sleep associated symptoms. Conversely, most antidepressants and anticataplectics have little effect on alertness. Compounds should be selected depending on the diagnosis and the targeted symptoms. Co-administration of two or more classes of compounds may be needed in some patients to adequately address their symptoms.<sup>13</sup>

#### **UNRESOLVED ISSUES & FURTHER DIRECTIONS**

Ten percent of patients with narcolepsy with cataplexy have normal hypocretin-1 levels in the CSF, which suggests that CSF levels either do not perfectly reflect brain hypocretin neurotransmission or that narcolepsy with cataplexy can be caused by factors other than hypocretin deficiency. The cause of the hypocretin cell destruction remains unknown, although an autoimmunemediated mechanism is suspected.



#### Non-24-Hour Sleep-Wake Rhythm Disorder (N24SWD)

Circadian Rhythm Sleep-Wake Disorders Clinical Division

N24SWD is characterized by symptoms of insomnia or excessive sleepiness that occur because the intrinsic circadian pacemaker is not entrained to a 24-hour light/ dark cycle. The non-24-hour period can be shorter or, more typically, longer than 24 hours.

Individuals typically present with episodes of difficulty falling asleep or staying asleep, excessive sleepiness or both, alternating with short asymptomatic periods. The severity of individual sleep-wake symptoms can be variable. Most individuals with nonentrained circadian rhythms are totally blind, and the failure to entrain circadian rhythms is related to the lack of photic input to the circadian pacemaker.

#### **DEMOGRAPHICS**

It is thought that over half of totally blind individuals have non-24-hour circadian rhythms; 50% to 80% of blind individuals complain of sleep disturbances.

#### **PATHOLOGY & PATHOPHYSIOLOGY**

The intrinsic period of the human circadian pacemaker is usually longer than 24 hours and requires daily input from the environment to maintain synchrony to the 24-hour day. The light-dark cycle is the most important environmental time cue (zeitgeber) in humans (as in other species), although nonphotic time cues also play a role in normal entrainment. A lack of photic input to the circadian pacemaker is clearly the cause of nonentrained rhythms in totally blind people. It has been suggested that, in sighted individuals, a systematic delay due to inadequate exposure to light may contribute to the development of N24SWD. In addition, the disorder may be caused by an extremely prolonged endogenous circadian period that is outside of the range for entrainment to the 24-hour cycle or by an alteration in the response of the circadian clock to the entraining effects of light.

#### TREATMENT

Research suggests that clinicians use strategically timed melatonin for the treatment of N24SWD in blind adults.<sup>14</sup>

#### **UNRESOLVED ISSUES & FURTHER DIRECTIONS**

There is only limited knowledge of the underlying pathophysiology of sighted persons with N24SWD. The primary risk factor in sighted persons appears to be a long circadian period that is beyond the range of entrainment to a 24-hour cycle or a progressive delay due to inappropriate exposure to light. This risk may explain the overlap between DSWPD and N24SWD.

Future studies are needed to understand the role of genetic predisposition, environmental or social cues and traumatic brain injury in the development of N24SWD, and to delineate other health consequences of the condition.

Most individuals with nonentrained circadian rhythms are totally blind.



#### **REM Sleep Behavior Disorder (RBD)**

Parasomnias Clinical Division

RBD is characterized by abnormal behaviors emerging during REM sleep that may cause injury or sleep disruption. RBD is also associated with electromyogram (EMG) abnormalities during REM sleep. The EMG demonstrates an excess of muscle tone during REM sleep, and/or an excess of phasic EMG twitch activity during REM sleep. Because RBD occurs during REM sleep, it usually appears at least 90 minutes after sleep onset.

A complaint of sleep related injury is common with RBD, which usually manifests as an attempted enactment of unpleasant, action-filled, and violent dreams in which the individual is being confronted, attacked, or chased by unfamiliar people or animals. Typically, at the end of an episode, the individual awakens quickly, becomes rapidly alert, and reports a dream with a coherent story. The dream action corresponds closely to the observed sleep behaviors.

#### **DEMOGRAPHICS**

The major predisposing factors are male sex, age 50 years or older, and an underlying neurological disorder, particularly Parkinson disease, multiple system atrophy, dementia with Lewy bodies, narcolepsy, or stroke.

RBD emerging in adults before age 50 years tends to have different demographics and associated features, including greater sex parity and increased rates of idiopathic RBD and parasomnia overlap disorder (POD), comorbid narcolepsy, antidepressant medication use, and possibly autoimmune diseases.

#### **PATHOLOGY & PATHOPHYSIOLOGY**

Current evidence suggests a selective association between RBD and neurodegenerative disorders. The synucleinopathies comprise a set of neurodegenerative disorders that share a common pathologic lesion composed of aggregates of insoluble a-synuclein protein in selectively vulnerable populations of neurons and glial cells. These pathologic aggregates appear to be closely linked to the onset and progression of clinical symptoms and the degeneration of affected brain regions in neurodegenerative disorders.

#### TREATMENT

Small case series and case reports describe efficacy of a wide range of medications, most prominently clonazepam. Melatonin also is suggested for the treatment of RBD with the advantage that there are few side effects. Maintaining a safe sleeping environment for both the patient and the bed partner, are paramount to injury prevention and should be enforced as an adjunct to therapeutic intervention.<sup>15</sup>

#### **UNRESOLVED ISSUES & FUTURE DIRECTIONS**

It is unclear why there is a male predominance of this disorder in middle-aged and older adults. It is unknown if RBD subgroups other than men age 50 years or older demonstrate increased risk for development of parkinsonism or dementia.

RBD usually appears at least 90 minutes after sleep onset.



#### **Restless Legs Syndrome (RLS)**

Sleep Related Movement Disorders

RLS is a sensorimotor disorder characterized by a complaint of a strong, nearly irresistible urge to move the limbs. This urge to move is often but not always accompanied by other uncomfortable sensations felt deep inside the limbs or by a feeling that is simply difficult or impossible to describe. Although the legs are most prominently affected, "restless legs" is a misnomer, in that 21% to 57% of individuals with RLS describe some arm sensations. The most common words adults use to describe RLS are restless, uncomfortable, twitchy, need to stretch, urge to move and legs want to move on their own. About half express their RLS sensations as painful. In clinical populations, disturbed sleep is reported in 60% to 90% of individuals with RLS, is typically the most troubling symptom, and is often the primary reason for seeking medical care.

Multiple clinic-based and population-based studies have shown an increased prevalence of mood and anxiety disorders in individuals with RLS. Similarly, increased rates of attention deficit hyperactivity disorder (ADHD) have been found in RLS, both in pediatric and adult studies.

#### DEMOGRAPHICS

The overall prevalence of RLS has been estimated at 5% to 10% in European and North American populationbased studies. However, in Asian countries, studies thus far indicate a lower prevalence. Prevalence is about twice as high in women than in men. In most studies, prevalence increases with age up to 60-70 years, except in Asian populations where an age related increase has not been found.

#### PATHOLOGY & PATHOPHYSIOLOGY

Brain iron deficiency, central nervous system dopamine regulation, and genetics appear to be primary factors in the pathophysiology of RLS. Iron is important in brain dopamine production and synaptic density, as well as in myelin synthesis and energy production. A connection between RLS and low brain iron is supported by autopsy data, MRI, brain sonography, and cerebrospinal fluid analysis.

#### TREATMENT

There are 2 types of therapies for RLS: pharmacotherapy and non-pharmacotherapy. The use of pharmacotherapy has been more widespread. Overall, dopaminergic agents are the most extensively investigated and used therapies for the treatment of RLS. Clinicians may use supplemental iron to treat RLS patients with low ferritin levels. There is insufficient evidence at present to evaluate the use of non-pharmacological therapy for RLS, including accommodative strategies, sleep hygiene, behavioral and stimulation therapies, compression devices, exercise, and nutritional considerations.<sup>16</sup>

#### **UNRESOLVED ISSUES & FURTHER DIRECTIONS**

The diagnosis of RLS relies on the subjective report of sensory symptoms that lie outside the range of common sensory experience. Many patients have difficulty describing the sensations. Further studies of the biological bases for RLS may lead to better classification of RLS and possibly to objective tests for diagnosis.

## SLEEP TECHNOLOGY

The clinical practice of sleep medicine involves the use of an intriguing blend of established and emerging diagnostic and therapeutic technologies.



#### POLYSOMNOGRAPHY

The use of PSG for evaluating sleep disorders requires recording the following physiologic signals: electroencephalogram (EEG), electrooculogram (EOG), chin electromyogram (EMG), airflow, oxygen saturation, respiratory effort, and electrocardiogram (ECG) or heart rate. Additional recommended parameters include body position and leg EMG derivations.



#### HOME SLEEP APNEA TESTING

HSAT records airflow, respiratory effort, and blood oxygenation to detect OSA. The biosensors used to monitor these parameters include an oronasal thermal sensor to detect apneas, a nasal pressure transducer to measure hypopneas, oximetry, and inductance plethysmography for respiratory effort. Peripheral arterial tone also can be used to identify respiratory events.

#### **THERAPIES**

Therapies commonly used to treat sleep disorders include positive airway pressure (PAP) therapy, cognitive behavioral therapy (CBT), bright light therapy, oral appliance therapy, surgery, and pharmacotherapy. Innovative treatments that have been introduced in recent years include vibro-tactile positional therapy, oral pressure therapy, and an implanted upper airway stimulation system.

#### **SLEEP TRACKERS**

Consumer sleep trackers – including mobile apps, wearable devices, and embedded devices – have become ubiquitous and are enabling individuals to gather unprecedented amounts of data about their sleep. While a lack of validation data limits current clinical uses, ongoing innovation is likely to give consumer sleep technology an increasingly prominent role in the clinical practice of sleep medicine.<sup>17</sup>



#### **TELEMEDICINE**

There is great potential for telemedicine to improve patient access to high quality sleep health care provided by board-certified sleep medicine physicians and the team of health care professionals at AASM-accredited sleep centers. Today sleep specialists are at the forefront of telemedicine implementation, removing the barriers between physicians and patients to allow direct delivery and seamless care.<sup>18</sup>

# SECIALISTS

Following medical school, the pathway to become a sleep specialist begins with the completion of a residency and board certification in one of these primary specialties:

- Internal Medicine
- Otolaryngology
- Neurology
- Pediatrics
- Psychiatry
- Anesthesiology
- Family Medicine



#### **Fellowship Training**

To become eligible for certification in the subspecialty of sleep medicine, you also must complete a one-year sleep medicine fellowship in a training program that is approved by the Accreditation Council for Graduate Medical Education (ACGME). During the program, fellows learn about the normal mechanisms of sleep physiology and the pathophysiology of sleep disorders. Fellows gain competency in the diagnosis and treatment of sleep disorders, developing skills for the interdisciplinary care of patients of all ages. Fellowships incorporate aspects of internal medicine, pediatrics, psychiatry, neurology, surgery, epidemiology and basic science.

A discounted AASM membership rate is available for medical students, residents and fellows.



#### **Board Certification**

Every two years, a board certification exam in sleep medicine is offered by six member boards of the American Board of Medical Specialties (ABMS). The exam lasts one day and is divided into several sessions. The primary medical content categories of the exam are: normal sleep and variants, sleep disorders, sleep in other disorders, and instrumentation and testing. Candidates who successfully pass the exam earn the distinction of being a board-certified sleep medicine physician. More than 6,000 physicians have earned board certification in sleep medicine since the first exam was offered by ABMS member boards in 2007.

American Academy of SLEEP MEDICINE<sup>™</sup>

#### **Professional Membership**

Physicians and other health care professionals who care for patients who have sleep disorders are encouraged to become a member of the American Academy of Sleep Medicine to stay at the forefront of the clinical practice of sleep medicine. Established in 1975, the AASM improves sleep health and promotes high quality, patient-centered care through advocacy, education, strategic research, and practice standards. A discounted membership rate is available for medical students, residents and fellows. Learn more at <u>aasm.org</u>.

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