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# Sleep disorders in psychiatry

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#### **Abstract**

Sleep is an active state that is critical for our physical, mental, and emotional well-being. Sleep is also important for optimal cognitive functioning, and sleep disruption results in functional impairment. Insomnia is the most common sleep disorder in psychiatry. At any given time, 50% of adults are affected with 1 or more sleep problems such as difficulty in falling or staying asleep, in staying awake, or in adhering to a consistent sleep/wake schedule. Narcolepsy affects as many individuals as does multiple sclerosis or Parkinson disease. Sleep problems are especially prevalent in schizophrenia, depression, and other mental illnesses, and every year, sleep disorders, sleep deprivation, and sleepiness add billions to the national health care bill in industrialized countries. Although psychiatrists often treat patients with insomnia secondary to depression, most patients discuss their insomnia with general care physicians, making it important to provide this group with clear guidelines for the diagnosis and management of insomnia. Once the specific medical, behavioral, or psychiatric causes of the sleep problem have been identified, appropriate treatment can be undertaken. Chronic insomnia has multiple causes arising from medical disorders, psychiatric disorders, primary sleep disorders, circadian rhythm disorders, social or therapeutic use of drugs, or maladaptive behaviors. The emerging concepts of sleep neurophysiology are consistent with the cholinergic-aminergic imbalance hypothesis of mood disorders, which proposes that depression is associated with an increased ratio of central cholinergic to aminergic neurotransmission. The characteristic sleep abnormalities of depression may reflect a relative predominance of cholinergic activity. Antidepressant medications presumably reduce rapid eye movement (REM) sleep either by their anticholinergic properties or by enhancing aminergic neurotransmission. Intense and prolonged dreams often accompany abrupt withdrawal from antidepressant drugs, a reflection of an REM rebound after drug-induced REM deprivation. The postulated link between sleep and psychiatric disorders has been reinforced by the findings of modern neurobiology. © 2006 Elsevier Inc. All rights reserved.

## 1. Introduction

We spend one third of our life asleep. If we live to be 70 years old, approximately 27 years will have been spent in sleep, which is an active state critical for our physical, mental, and emotional well-being. In fact, sleep is a basic necessity of life, as fundamental for the maintenance of human health as air, food, and water.

Sleep is also important for optimal cognitive functioning. More than 90 sleep pathologies have been identified, and, when sleep is disrupted, individuals cease to function effectively. Insomnia is the most common sleep disorder in psychiatry [1].

Before the issues of sleep disorders and insomnia are discussed, it is important to consider normal sleep physiol-

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ogy. Sleep is a remarkable physiologic state that is essentially governed by 2 simple processes. The first is a homeostatic process that produces sleep based on time elapsed since the last sleep. The second process is our circadian rhythm which, in a way, allows us to be alert when homeostasis is at its maximum and allows us to be sleepy when (homeostatically) we are meant to be alert [2].

A variety of issues confound these 2 physiologic processes and cause an array of sleep difficulties. In earlier reports, it was found that behavior and psychologic problems are major disrupters of these primary biologic processes, and that age is also a big risk factor for sleep difficulties [3]. Indicators of homeostasis such as stages 3 to 4 of sleep are blunted in the elderly, and there are reliable data to suggest that, as we age, the amplitude of our circadian rhythm, as well as its timing, becomes abnormal. In a normal night's sleep it is recognized that, in terms of homeostatic processes, delta sleep (slowwave sleep)—an indicator of sleep homeostatic processes—occurs primarily in the first half of the night and then diminishes in the second half of the night. Homeostatic

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processes are the main triggers for the rapid eye movement (REM)-non-REM 90-minute cycle.

Equally important to homeostasis is our circadian rhythm [4]. It is becoming increasingly important to recognize the fact that the timing of sleep and wake is a critical variable. In fact, we now know that the locus of our circadian rhythm is the suprachiasmatic nucleus [5]. This nucleus is primarily governed by light and dark. The retinal hypothalamic track transmits light to the suprachiasmatic nucleus, allowing us to know—physiologically—when it is light and dark and, hence, when we should be asleep or awake.

### 2. Sleep and health

# 2.1. Prevalence and public health consequence of sleep disorders

At any one moment, 50% of adults are affected with 1 or more sleep problems [6]. These sleep problems include difficulties in falling or staying asleep, in staying awake, and in adhering to a consistent sleep/wake schedule. In addition, specific sleep disorders themselves occur with significant magnitude. Narcolepsy, for example, affects as many individuals as does multiple sclerosis or Parkinson disease.

Clearly, disrupted sleep has grave consequences for individuals and for society as a whole. There are economic consequences, for example, that result from the current sleep status of our population. The result is a tremendous loss for individuals and society. In addition, a 20% increase in physical problems occurs when there is a problem sleeping, and most adults have great difficulty making decisions when they are sleep deprived [7]. Sleep problems are especially prevalent in schizophrenia, depression, and other mental illnesses. Each year, sleep disorders, sleep deprivation, and sleepiness add billions to the national health care bill in industrialized countries [8].

There are also many transportation and safety consequences of disturbed sleep. Twenty percent of all drivers have fallen asleep behind the wheel, at least once. The most frequently cited probable cause of mass transportation accidents is fatigue, accounting for nearly one third of all fatal-to-the-driver trucking accidents. About 50% of all fatal crashes are caused by drowsy drivers. Annual sleep-related accidents in transportation alone claim thousands of lives, cause hundreds of thousands of injuries, and add significantly to health care costs, lost productivity, and damage to property [9]. A host of other consequences of disturbed sleep, ranging from shift work to jet lag, are at the core of health problems and economic loss.

Although insomnia has been treated in many ways over the centuries, it is still defined inconsistently among health professionals.

#### 2.2. Symptoms, durations, and causes of insomnia

Insomnia is defined as a symptom of difficulty in initiating sleep, maintaining sleep, or of having nonrestorative sleep. This symptom is typically associated with daytime consequences including interference and/or disruption in one's ability to function during the day. Typically, patients report fatigue, irritability, and/or impaired memory and performance [10].

Although insomnia is a symptom, it is also viewed by many to be a complaint, a disorder, a disease, or even the result of another disorder. It is obvious that there is a great need to define insomnia clearly and precisely, despite the major advances made in the identification and diagnosis of insomnia over the last 3 to 4 decades. These advances in our understanding of insomnia can be traced to the widespread use of polysomnography and the evolution of sleep medicine, as well as growth in the number of sleep specialists [11].

Unfortunately, the reality is that patients with insomnia complaints present their problems to a variety of different types of clinicians, most of whom do not specialize in sleep. It is not unusual for urologists, allergists, and even podiatrists to receive patients' complaints of insomnia. For example, psychiatrists are often exposed to patients with insomnia secondary to their depression, whereas patients with disturbed sleep due to sleep-related breathing disorders, such as sleep apnea, are usually treated by pulmonary physicians [12]. In fact, most patients discuss their insomnia with general care physicians, and it is this class of physicians for whom it is most important to provide clear guidelines for the diagnosis and management of insomnia.

Because insomnia is a symptom of heterogeneous origin, the first responsibility of the physician is to carefully evaluate and accurately diagnose the problem and determine those individuals who are affected with the disorder, or are most likely to have insomnia. When specific medical, behavioral, or psychiatric causes of the sleep problem are identified, appropriate treatment can be undertaken. However, it is important to recognize that multiple causes are the rule, not the exception. Thus, behavioral and/or symptomatic therapies must often be undertaken simultaneously. In addition, virtually every patient complaining of insomnia benefits from improved sleep hygiene and behavioral therapies, either by themselves or, more typically, as part of a broader treatment plan. It is also important to note that insomnia is not evenly distributed in the population. Some groups are at greater risk of developing insomnia than others. The groups at greater risk are women, the elderly, those with medical or psychiatric disease, and those who work at times other than the traditional period during the day.

Insomnia can be a single, transient event taking place from time to time or for one to a few nights. This is the least common type of insomnia. More typically, sleep problems tend to be recurring, lasting for 1 to 3 weeks. For many individuals, insomnia is a long-term problem that occurs most nights and continuing for more than 4 weeks. Studies show that 50% of the individuals who report having severe insomnia still have the problem 2 years later. Determining

the duration of insomnia is important because it contributes to the physician's understanding of its causes [13].

Transient, or short-term insomnia, which lasts for days, is typically associated with stress, changes in the timing of sleep, or the sleep environment. If the period of transient insomnia can be anticipated, sleep-promoting medications are appropriate and can be used prophylactically as the sole therapy [14]. Transient insomnia is usually accompanied by reports of daytime sleepiness and impaired performance the next day [15].

Usually transient insomnia has a single cause for each patient, such as changes in the timing of sleep, changes in the sleep environment, or acute illness. The most common causes of transient/short-term insomnia are external factors (such as change in sleep environment, timing of sleep, noise, conflicts and anxiety, as well as acute medical illness) [16]. On the other hand, chronic insomnia has many different—and usually multiple—causes. Basically, chronic insomnia can be considered to arise from medical disorders, psychiatric disorders, primary sleep disorders, circadian rhythm disorders, social or therapeutic drug use, or maladaptive behaviors. The most common causes of chronic insomnia include psychiatric disorders and primary sleep disorders as well as untreated transient insomnia that becomes chronic.

Unfortunately, most studies of insomnia, whether transient, recurring, or chronic, do not yield a precise conclusion as to causality—poor sleep and subsequent impairment of waking functions may both be results of a medical and/or psychiatric disease as well as behavioral dysfunction. Most medical conditions such as chronic renal failure, arthritic conditions, hyperthyroidism, heart failure, chronic lung disease, Parkinson disease, reflux esophagitis, and pain becoming worse during the night are likely to disrupt sleep. Therefore, it is very common for patients with medical disorders to have insomnia arising from disrupted sleep, that is, insomnia that occurs because sleep is fragmented.

#### 2.3. Psychiatric causes of insomnia

Most studies show that about half of patients with chronic insomnia have a sleep problem that arises because of a psychiatric disorder. The most prominent psychiatric causes of insomnia are psychoses, mood disorders, anxiety disorders, panic disorders, and dementia.

Psychiatrists have long had a strong interest in sleep and dreaming, especially whether sleep disturbances cause mental disorders or vice versa. In the last century, the famous English neurologist, Hughlings Jackson, said "Find out about dreams and you will find out about psychosis." Later, Freud made dream interpretation one of the key elements of psychoanalytic theory and practice [17]. After the discovery of REM sleep in the early 1950s, psychiatrists and sleep researchers studied the sleep patterns of schizophrenic and depressed patients, testing the hypothesis that hallucinations might represent waking dreams or some derangement of REM sleep, and determining whether or not

deprivation of REM sleep or sleep in general would be psychologically deleterious or beneficial [18].

The new neurobiology of sleep has important implications for depression, some types of alcoholism and schizophrenia, eating disorders, borderline personality disorder, and other clinical conditions associated with short REM latency, increased REM sleep, increased REM sleep density, and loss of stages 3 and 4 sleep [19].

The emerging concepts of sleep neurophysiology are consistent with the cholinergic-aminergic imbalance hypothesis of mood disorders, which proposes that depression is associated with an increased ratio of central cholinergic to aminergic neurotransmission. The characteristic sleep abnormalities of depression may reflect a relative predominance of cholinergic activity, originating within the lateral dorsal tegmental and pedunculopontine tegmental regions, in relation to noradrenergic and serotonergic activity originating within the locus coeruleus, and the dorsal raphe nucleus, respectively. Antidepressant medications presumably reduce REM sleep either by their anticholinergic properties or by enhancing aminergic neurotransmission. Intense and prolonged dreams often accompany abrupt withdrawal from antidepressant drugs, a reflection of a REM rebound after drug-induced REM deprivation [19].

Short REM latency has been found in some patients with schizophrenia, obsessive-compulsive disorder, alcoholism, eating disorders, narcolepsy, and borderline personality disorder. Both short REM latency and decreased delta sleep appear to be state and trait characteristics of depression [19]. It is of great interest that depression can be "turned off" quickly and predictably by "therapeutic" sleep deprivation. Further research designed to enhance our understanding of the mechanism of this striking effect is urgently needed.

In what other ways can depression be "turned" off and on as quickly and predictably as by "therapeutic" sleep deprivation and recovery sleep?

On a more practical clinical level, as understanding of mechanisms of arousal and sedation increases, the clinician will be able to deal more effectively with the symptoms of insomnia and hypersomnia associated with psychiatric illness, as well as the iatrogenic sleep problems often associated with medications. For example, many patients experience insomnia during treatment with selective serotonin reuptake inhibitors. These observations suggest that, under some circumstances, serotonin has an arousing effect—an effect that may be mediated by the serotonin 2 receptor. Medications that antagonize this receptor tend to increase sleep efficiency and sleep quality to a greater extent than selective serotonin reuptake inhibitors, which lack this property [20].

The clinical wisdom of the past, linking sleep to psychiatric disorders, has been reinforced by the discoveries of modern neurobiology. Sleep remains one of the "royal roads" to understanding psychiatric disorders, and to treating the patients who have them.

#### 3. Treatment

Insomnia, whatever its cause, has a negative impact on quality of life, exacerbating bodily pain, and adversely affecting general health, mental health, emotions, physiologic processes, social functioning, vitality, and physical functions. Thus, its treatment is of great importance. Of the various treatment options, hypnotic medications are the most universally effective [21]. Other treatment modalities such as behavioral therapy and hypnotherapy are also available.

Sleep disorders can be treated by sleep behavioral techniques [22]. It has been reported that sleep restriction therapy can improve certain types of insomnia. Other behavioral therapies such as sleep hygiene, relaxation techniques, and exercise cause an increase in core body temperature, which facilitates sleep, and this helps in the reestablishment of a regular circadian rhythm, which also facilitates sleep [23].

Additional behavioral techniques include avoidance of certain pharmacologic agents such as alcohol and caffeine. Moreover, behavioral techniques are widely available and may have beneficial effects on sleep difficulties.

Hypnotics are medications whose primary therapeutic effect is to improve sleep. As such, they can be the primary therapy used to treat transient insomnia as well chronic primary insomnia. In the current hypnotic treatment of insomnia, benzodiazepines have largely replaced barbiturates. In recent years, novel, nonbenzodiazepine hypnotic compounds have been introduced, which preserve the physiologic architecture of sleep and display a stabilizing effect on sleep patterns.

Pharmacologic management of insomnia has been around for several hundred years. Over the last 200 years, pharmacologic agents for the management of insomnia have improved in the sense that they have become safer and more effective. Probably the most important change in the pharmacologic management of insomnia occurred in the 1960s when the transition was made from general central nervous system suppressants to drugs that have more specific effects in the central nervous system. Specifically, discovery of the benzodiazepines revolutionized the pharmacologic treatment of insomnia by providing a class of medications that proved to be much safer and more specific than the earlier hypnotics.

In the 1980s and the 1990s, new nonbenzodiazepams, although they bind to the benzodiazepine site, have been found to exhibit enhanced specificity.

Certain guidelines should be followed to maximize the benefit of pharmacotherapy. These include prescribing the lowest effective dose and then not escalating it. The drug regimen should be tailored to each patient. Typically, long-term nightly use should be avoided. It is also important to combine pharmacotherapy with good sleep practices. Finally, if patients are being treated with long-term therapy, they should be followed regularly to make sure that the

hypnotic remains effective and that side effects do not develop.

#### 4. Conclusion

In conclusion, the following are messages that need emphasis. First, sleep is an active process; therefore, sleep disorders represent pathophysiology, which causes an overriding of these processes—processes that are probably homeostatic and circadian. Second, these processes are most profoundly affected by changes in age. And, finally, insomnia is a symptom that is prevalent and, most importantly, has serious consequences for health and well-being. Our task is to move forward in terms of education and to provide more knowledge to physicians and other health workers about the identification and treatment of these common debilitating disorders.

#### References

- Coleman RM, Roffwarg HP, Kemetry SJ, et al. Sleep-wake disorders based on a polysomnography diagnosis. A National Cooperative Study. JAMA 1982;247:997-1003.
- [2] Stewart C, Yotolsky SC, Hales RE. Neuropsychiatry and clinical neurosciences. Arlington, VA: American Psychiatric Publishing, Inc; 2002. p. 702-3.
- [3] Morin CM, Colecchi C, Stone J, Lood R, Brink D. Behavioral and pharmacological therapies per late-life insomnia: a randomized controlled trial. JAMA 1999;281:991.
- [4] Aschoff J. Circadian rhythms in man. Science 1965;148:1427-32.
- [5] Hales RE. Neuropsychiatry and clinical neurosciences. Arlington, VA: American Psychiatric Publishing, Inc; 2002. p. 703-4.
- [6] Worldwide project on sleep and health—WHO/MSA/MND/98.3. pg. 3.
- [7] Chokroverty S. Sleep disorders medicine: basic science technical considerations and clinical aspects. Boston: Butterwort - Honeymoon; 1994.
- [8] Sadock B, Sadock V. Comprehensive textbook of psychiatry 7th ed. vol. II. Lippincott, William & Wilkins; 2002. p. 1677.
- [9] Roth T. Insomnia, recognition diagnosis and public health influence. Sleep problems among psychological problems in general health care: form, frequency, comorbidity and related disability. Proceedings of the Sleep Symposium organized by the Japanese WHO Collaborating Center for Research and Training in Mental Health. Tokyo, Japan, April 23, 1996. Churchill Japan K.K., 1996. p. 19.
- [10] American Psychiatric Association. Diagnostic and statistical manual of mental disorder (DSM-IV). Arlington, VA: American Psychiatric Association; 2005.
- [11] American Sleep Disorder Association. The international classification of sleep disorders revised. Diagnostic and coding manual. Rochester, MN: American Sleep Disorder Association; 1997.
- [12] Sadock B, Sadock V. Comprehensive textbook of psychiatry 7th ed. vol. II. Rochester, MN: Lippincott, William & Wilkins; 2002. p. 1700.
- [13] The management of insomnia—guidelines for clinical practice—WPA Educational Program, 24-25, 1993.
- [14] The management of insomnia—guidelines for clinical practice—WPA Educational Program, 23-24, 1993.
- [15] The management of insomnia—guidelines for clinical practice—WPA Educational Program; 1993. p. 25.
- [16] The management of insomnia—guidelines for clinical practice—WPA Educational Program; 1993. p. 26.
- [17] Freud S. The interpretation of dreams. New York: Random House; 1950.
- [18] Jouvet M, Michel F, Courjon J. Sur un Stude d'activite eletrique cerebrale rappide au cours du sommeil physiologique. CR Seances Soc Biol Fil 1959;153:1028-9.

- [19] Sadock B, Sadock V. Comprehensive textbook of psychiatry 7th ed. vol. II. Lippincott, William & Wilkins; 2002. p. 1697.
- [20] Yotolsky SC, Hales RE. Neuropsychiatry and clinical neurosciences. Arlington, VA: American Psychiatric Publishing, Inc; 2002. p. 703.
- [21] Mellinger GD, Balter MB, Uhlenhuth EH. Insomnia and its treatment: prevalence and correlates. Arch Gen Psychiatry 1985;42:225-32.
- [22] Spielmon AJ, Caruso LS, Geovinsky PB. A behavioral perspective on insomnia treatment. Psychiatr Clinic North Am 1987;10:541-53.
- [23] Yotolsky SC, Robert RE. Neuropsychiatry and clinical neurosciences. American Psychiatric Publishing, Inc; 2002. p. 708.