Normal Sleep, Sleep Physiology, and Sleep Deprivation: General Principles

Last Updated: April 6, 2005

Synonyms and related keywords: non–rapid eye movement, NREM, rapid eye movement, REM, stage I sleep, light sleep, stage II sleep, stage III sleep, stage IV sleep, deep sleep, delta-wave sleep, polysomnography

AUTHOR INFORMATION

Author: **Michael B Russo, MD**, Director, Division of Aircrew Health and Performance, US Army Aeromedical Research Laboratory; Assistant Professor, Department of Neurology, Uniformed Services University

Michael B Russo, MD, is a member of the following medical societies: []
, American Academy of Neurology, American Academy of Sleep Medicine, and American Epilepsy Society

Editor(s): **Carmel Armon, MD, MSc, MHS**, Professor of Neurology, Tufts University School of Medicine; Chief, Division of Neurology, Baystate Medical Center; **Francisco Talavera, PharmD, PhD**, Senior Pharmacy Editor, eMedicine; **Jose E Cavazos, MD, PhD**, Assistant Professor, Departments of Medicine (Neurology) and Pharmacology, University of Texas Health Science Center at San Antonio; **Selim R Benbadis, MD**, Professor, Director of Comprehensive Epilepsy Program, Departments of Neurology and Neurosurgery, University of South Florida College of Medicine; and **Nicholas Lorenzo, MD**, Chief Editor, eMedicine Neurology; Consulting Staff, Neurology Specialists and Consultants

NORMAL SLEEP IN ADULTS, INFANTS, AND THE ELDERLY

Normal sleep is divided into non–rapid eye movement (NREM) and rapid eye movement (REM) sleep. The stages of sleep are stage I (light sleep), stage II, stages III and IV (deep or delta-wave sleep), and REM sleep; NREM sleep comprises stages I-IV. Sleep is an active process that cycles at an ultradian rhythm of about 90 minutes.

Waking usually transitions into NREM sleep. REM follows NREM sleep and occurs 4-5 times during a normal 8- to 9-hour sleep period. The first REM period of the night may be less than 10 minutes in duration, while the last may exceed 60 minutes.

For the purpose of analysis, overnight sleep has been divided into 3 equal time periods: sleep in the first third of the night, which comprises the highest percentage of NREM; sleep in the middle third of the night; and sleep in the last third of the night, the majority of which is REM. Awakening after a full night's sleep is usually from REM sleep.

Sleep in adults

In adults, sleep of 8-8.4 hours is considered fully restorative. In some cultures, total sleep often is divided into an overnight sleep period of 6-7 hours and a midafternoon nap of 1-2 hours.

Stage I is considered a transition between wake and sleep. It occurs upon falling asleep and during brief arousal periods within sleep and usually accounts for 5-10% of total sleep time. Stage II occurs throughout the sleep period and represents 40-50% of total sleep time. Stages III and IV delta sleep occur mostly in the first third of the night. They are distinguished from each other only by the percentage of delta activity and represent up to 20% of total sleep time. REM represents 20-25% of total sleep time.

Sleep in infants

Infants have an overall greater total sleep time than any other age group; their sleep time can be divided into multiple periods. In newborns, the total sleep duration in a day can be 14-16 hours.

Over the first several months of life, sleep time decreases; by age 5-6 months, sleep consolidates into an overnight period with at least 1 nap during the day. REM sleep in infants represents a larger percentage of the total sleep at the expense of stages III and IV. Until age 3-4 months, newborns transition from wake into REM sleep. Thereafter, wake begins to transition directly into NREM.

Overall, electrocortical recorded voltage remains high during sleep, as it does during periods of wakefulness. Sleep spindles begin appearing in the second month of life with a density greater than that seen in adults (see Sleep Physiology). After the first year, the spindles begin decreasing in density and progress toward adult patterns. K complexes begin by the sixth month of life.

Sleep in elderly persons

In elderly persons, the time spent in stages III and IV sleep decreases by 10–15% and the time in stage II increases by 5% compared to young adults, representing an overall decrease in total sleep duration. Latency to fall asleep and the number and duration of overnight arousal periods increase. Thus to have a fully restorative sleep, the total time in bed must increase. If the elderly person does not increase the total time in bed, complaints of insomnia and chronic sleepiness may occur. Sleep fragmentation results from the increase in overnight arousals and may be exacerbated by the increasing number of geriatric medical conditions, including sleep apnea, musculoskeletal disorders, and cardiopulmonary disease.

SLEEP PHYSIOLOGY

Sleep Disorders Learning Center

Sleep Disorders Learning Center

View all Sleep Disorders Articles

Sleep Disorders CME

Sleep Disorders Multimedia Library

Quick Find

Author Information
Normal Sleep In Adults, Infants, And The Elderly
Sleep Physiology
Circadian Rhythms That Influence Sleep
Effects Of Sleep Deprivation
Bibliography

Click for related images.

Continuing Education

CME available for this topic. Click here to take this CME.

Patient Education

Sleep Disorders Center

Sleep: Understanding the Basics Introduction

Sleep is a state of unconsciousness in which the brain is relatively more responsive to internal than to external stimuli. The predictable cycling of sleep and the reversal of relative external unresponsiveness are features that assist in distinguishing sleep from other states of unconsciousness. The brain gradually becomes less responsive to visual, auditory, and other environmental stimuli during the transition from wake to sleep, which is considered by some to be stage I of sleep.

Historically, sleep was thought to be a passive state that was initiated through withdrawal of sensory input. Currently, withdrawal of sensory awareness is believed to be a factor in sleep, but an active initiation mechanism that facilitates brain withdrawal also is recognized.

NREM sleep is controlled by complex initiating and maintenance mechanisms, the extent of which is not fully elaborated. Probably no single sleep-generating center exists. A more likely mechanism is sleep-generating circuits with inputs from brainstem and hypothalamic neuronal groups. Within these circuits, sleep initiation may begin with the emergence of inhibitory signals from the anterior hypothalamic preoptic nucleus directed caudally toward the brainstem reticular core and posterior hypothalamus. The preoptic nucleus inhibits the histaminergic posterior hypothalamic tuberoinfundibular region through GABA and probably acetylcholine.

The tuberoinfundibular region projects rostrally to the intralaminar nuclei of the thalamus and to the cerebral cortex. Inhibition of the tuberoinfundibular region is a critical step toward falling asleep because it results in functional disconnection between the brain stem and the more rostral thalamus and cortex. A decrease in ascending thalamic cholinergic transmissions occurs in association with decreasing cortical responsiveness. In addition to inhibiting higher cortical consciousness, the tuberoinfundibular tract projects caudally into the pontine reticular system and inhibits afferent transmissions from ascending cholinergic tracts.

NREM is an active state that is maintained partly through oscillations between the thalamus and the cortex. The 3 major oscillation systems are sleep spindles, delta oscillations, and slow cortical oscillations. Sleep spindles, a hallmark of stage II sleep, are generated by bursts of hyperpolarizing GABAnergic thalamic reticular neurons. These bursts inhibit thalamocortical projection neurons. As deafferentation spreads, corticothalamic projections back to the thalamus synchronize. As hyperpolarization of the thalamic reticular neurons progresses, delta waves are produced by interactions from both thalamic reticular and cortical pyramidal sources. Slow cortical oscillations are produced in neocortical networks by cyclic hyperpolarizations and depolarizations.

Although the functions of NREM sleep remain speculative, several theories have been put forth. One theory proposes that decreased metabolic demand facilitates replenishment of glycogen stores. Another theory, which utilizes neuronal plasticity, suggests that the oscillating depolarizations and hyperpolarizations consolidate memory and remove redundant or excess synapses.

REM sleep is generated by mesencephalic and pontine cholinergic neurons, hence these are referred to as REM-on neurons. As REM sleep initiates, monoadrenergic locus ceruleus and serotonergic raphe neurons become inactive and are called REM-off neurons.

REM is characterized by muscle atonia, cortical activation, low-voltage desynchronization of the EEG, and rapid eye movements. REM may be considered to have both tonic and phasic characteristics. Tonic muscle atonia is present throughout REM sleep. It results from inhibition of alpha motor neurons by clusters of peri–locus ceruleus neurons, which are referred to collectively as the dorsolateral small cell reticular group.

Projection of the presumed cholinergic dorsolateral small cell reticular group is through the medullary reticular formation, which projects through the ventrolateral reticulospinal tract to inhibitory spinal and bulbar interneurons. Glycinergic interneurons produce postsynaptic inhibition and hyperpolarization of the spinal alpha motor neurons. Tonic cortical activation with EEG desynchronization is promoted by projections from cholinergic lateral dorsal tegmental and pedunculopontine tegmental neurons to the thalamic nuclei. Other projections through brainstem reticular formation neurons are likely to be involved as well.

Phasic rapid eye movements are composed of lateral saccades generated in the paramedian pontine reticular formation and vertical saccades thought to be generated in the mesencephalic reticular formation. REM density is a term used to describe the frequency per minute of the eye movement bursts.

Phasic pontine-geniculate-occipital (PGO) spikes are another neurophysiological feature of REM sleep. These spikes appear to be generated by lateral dorsal tegmental and pedunculopontine tegmental neuronal bursts. They are projected to the lateral geniculate and other thalamic nuclei, and then to the occipital cortex. PGO bursts precede rapid eye movements by several seconds. Increases in PGO bursts are seen after REM sleep deprivation.

Other phasic features of REM sleep include periodic skeletal muscle twitches, increased heart rate variability, pupil dilation, and increased respiratory rate.

During NREM sleep, the metabolic demand of the brain decreases. This is supported by oxygen positron emission tomography (PET) studies, which show that, during NREM sleep, the blood flow throughout the entire brain progressively decreases. PET studies also show that, during REM sleep, blood flow increases in the thalamus and the primary visual, motor, and sensory cortices, while remaining comparatively decreased in the prefrontal and parietal associational regions. The increase in blood flow to the primary cortical regions may explain the vivid nature of REM dreaming, while the continued decrease in blood flow to the prefrontal cortex may explain the unquestioning acceptance of even the most bizarre dream content.

For excellent patient education resources, visit eMedicine's Sleep Disorders Center. Also, see eMedicine's patient education article Sleep: Understanding the Basics.

CIRCADIAN RHYTHMS THAT INFLUENCE SLEEP

Section 4 of 6 [Back Top Next]

Author Information Normal Sleep In Adults, Infants, And The Elderly Sleep Physiology Circadian Rhythms That Influence Sleep Effects Of Sleep Deprivation Bibliography

Circadian sleep rhythm is 1 of the several intrinsic body rhythms modulated by the hypothalamus. The suprachiasmatic nucleus sets the body clock to approximately 25 hours, with both light exposure and schedule clues entraining to the 24-hour cycle. The retinohypothalamic tract allows light cues to directly influence the suprachiasmatic nucleus. Light is called a "zeitgeber," a German word meaning "time-giver," because it sets the suprachiasmatic clock. A practical purpose has been proposed for the circadian rhythm, using the analogy of the brain being somewhat like a battery charging during sleep and discharging during the wake period.

The nadir of the rhythm is in the early morning. The downswing in circadian rhythm prior to the nadir is thought to assist the brain to remain asleep overnight for full restoration by preventing premature awakening. The morning upswing then facilitates awakening and through the day acts as a counterbalance to the progressive discharge of wake neuronal activity. After the circadian apex in the early evening, the downswing aids sleep initiation. This model explains the relatively steady cognitive function throughout wakefulness.

Body temperature cycles are also under hypothalamic control. An increase in body temperature is seen during the course of the day and a decrease is observed during the night. The temperature peaks and troughs are thought to mirror the sleep rhythm. People who are alert late in the evening (ie, evening types) have body temperature peaks late in the evening, while those who find themselves most alert early in the morning (ie, morning types) have body temperature peaks early in the evening.

Melatonin has been implicated as a modulator of light entrainment. It is secreted maximally during the night by the pineal gland. Prolactin, testosterone, and growth hormone also demonstrate circadian rhythms, with maximal secretion during the night.

EFFECTS OF SLEEP DEPRIVATION

Glucose-PET studies in individuals deprived of sleep have shown that after 24 hours of sustained wakefulness, the metabolic activity of the brain decreases significantly (up to 6% for the whole brain and up to 11% for specific cortical and basal ganglionic areas). In humans, sleep deprivation also results in a decrease in core body temperature, a decrease in immune system function as measured by white cell count and activity, and a decrease in the release of growth hormone. Sleep deprivation also has been implicated as a cause of increased heart rate variability.

As the function of sleep has not been fully determined, the absolute number of hours necessary to fulfill its function is still unknown. Some individuals claim full effectiveness with only 3-5 hours of sleep per night, while some admit needing at least 8 hours of sleep per night (or more) to perform effectively. Sleep deprivation is best defined at this point by group means and in terms of the tasks impaired.

With decreased sleep, higher-order cognitive tasks are affected early and disproportionately. Tests requiring both speed and accuracy demonstrate considerably slowed speed before accuracy begins to fail. Total sleep duration of 7 hours per night over 1 week has resulted in decreased speed in tasks of both simple reaction time and more demanding computer-generated mathematical problem solving. Total sleep duration of 5 hours per night over 1 week shows both decrease in speed and the beginning of accuracy failure.

Total sleep duration of 7 hours per night over 1 week leads to impairment of cognitive work requiring simultaneous focus on several tasks. In driving simulations, for example, accidents increase progressively as total sleep duration is decreased to 7, 5, and 3 hours per night over 1 week. In the same simulations, 3 hours total sleep duration was associated with loss of ability to simultaneously appreciate peripheral and centrally presented visual stimuli, which could be termed as a form of visual simultanagnosia and peripheral visual neglect.

In tasks requiring judgment, increasingly risky behaviors emerge as the total sleep duration is limited to 5 hours per night. The high cost of an action seemingly is ignored as the sleep-deprived individual focuses on limited benefit.

One explanation for decreasing performance in sleep deprivation is the occurrence of microsleep. Microsleep is defined as brief (several seconds) runs of theta or delta activities that break through the otherwise beta or alpha EEG of waking. It has been seen to increase with sleep deprivation. In studies in which polysomnography is recorded simultaneously, microsleep impairs continuity of cognitive function and occurs prior to performance failure. However, the occurrence of microsleep has not been shown in most instances of polysomnographic correlated performance failure. Other explanations for performance impairments include sensory perceptual impairments such as the development of visual neglect phenomena.

These experimental findings can be explained by glucose-PET studies, which show that individuals deprived of sleep for 24 hours have a decrease in metabolism in the prefrontal and parietal associational areas. The areas most important for judgment, impulse control, attention, and visual association are disproportionately hypometabolic compared to the primary sensory and motor areas necessary for receiving and acting upon the environmental inputs. This finding leads to the hypothesis that the areas of the brain most responsible for higher-order cognition are to some degree less functional during sleep-deprived waking activity.

Sleep deprivation is a relative concept. Small amounts of sleep loss (eg, 1 hour per night over many nights) have subtle cognitive costs, which appear to go unrecognized by the individual experiencing the sleep loss. More severe restriction of sleep for a week leads to profound cognitive deficits similar to those seen in some stroke patients, which also appear to go unrecognized by the individual. The lack of recognition of the effects of sleep deprivation appears to be a constant feature, one which, it is hoped, will be overcome by further research and education.

Acknowledgments: Thanks are extended to COL Gregory Belenky, MD, Director of the Division of Neurosciences; Thomas Balkin, PhD, Chief of Neurobiology and Behavior; and Maria Thomas, PhD, Chief of Biomedical Assessment at the Walter Reed Army Institute of Research for their comments and suggestions for this manuscript.

Disclaimer: The contents of this article reflect only the opinions of the author and do not constitute the official policy of the US Department of Defense.

BIBLIOGRAPHY

- Abrams JK, Johnson PL, Hollis JH, Lowry CA: Anatomic and functional topography of the dorsal raphe nucleus. Ann N Y Acad Sci 2004 Jun; 1018: 46-57[Medline].
- Anders TF: Maturation of sleep patterns in the newborn infant. In: Weitzman ED, ed. Advances in Sleep Research. New York: Spectrum; 1975:43.
- Benington JH, Heller HC: Restoration of brain energy metabolism as the function of sleep. Prog Neurobiol 1995 Mar; 45(4): 347-60[Medline].
- Bonnet MH, Arand DL: We are chronically sleep deprived. Sleep 1995 Dec; 18(10): 908-11[Medline].
- Borbely A: Secrets of sleep. New York: Basic Books; 1984.
- Chokroverty S: Sleep and breathing in neurological disorders. In: Edelman NH, Santiago TV, eds. Breathing Disorders of Sleep. New York: Churchill Livingstone; 1986:225.
- Chokroverty S: Physiologic changes in sleep. In: Sleep Disorders Medicine. Boston: Butterworth-Heinemann; 1999:95-126.

- Contreras D, Steriade M: Spindle oscillation in cats: the role of corticothalamic feedback in a thalamically generated rhythm. J Physiol 1996 Jan 1; 490 (Pt 1): 159-79[Medline].
- Contreras D, Destexhe A, Sejnowski TJ, Steriade M: Control of spatiotemporal coherence of a thalamic oscillation by corticothalamic feedback. Science 1996 Nov 1; 274(5288): 771-4[Medline].
- Contreras D, Destexhe A, Sejnowski TJ, Steriade M: Spatiotemporal patterns of spindle oscillations in cortex and thalamus. J Neurosci 1997 Feb 1; 17(3): 1179-96[Medline].
- Crick F, Mitchison G: The function of dream sleep. Nature 1983 Jul 14-20; 304(5922): 111-4[Medline].
- Dinges DF, Douglas SD, Hamarman S, et al: Sleep deprivation and human immune function. Adv Neuroimmunol 1995; 5(2): 97-110[Medline].
- Hairston IS, Knight RT: Neurobiology: sleep on it. Nature 2004 Jul 1; 430(6995): 27-8[Medline].
- Harrison Y, Horne JA: Should we be taking more sleep?. Sleep 1995 Dec; 18(10): 901-7[Medline].
- Horne J: Why We Sleep? . New York: Oxford University Press; 1988.
- Jennings JR, Monk TH, van der Molen MW: Sleep deprivation influences some but not all processes of supervisory attention. Psychol Sci 2003 Sep; 14(5): 473-9[Medline].
- Jones BE: Paradoxical sleep and its chemical/structural substrates in the brain. Neuroscience 1991; 40(3): 637-56[Medline].
- Kavanau JL: Memory, sleep and the evolution of mechanisms of synaptic efficacy maintenance. Neuroscience 1997 Jul; 79(1): 7-44[Medline].
- Kryger MH, Roth T, Dement WC, eds: Principals and Practice of Sleep Medicine. Philadelphia, Pa: Saunders; 1994.
- Kumar VM: Role of noradrenergic fibers of the preoptic area in regulating sleep. J Chem Neuroanat 2003 Oct; 26(2): 87-93[Medline].
- Mancia M, Marini G, eds: The Diencephalon and Sleep . New York: Raven; 1990.
- Metcalf DR: The effect of extrauterine experience on the ontogenesis of EEG sleep spindles. Psychosom Med 1969 Sep-Oct; 31(5): 393-9[Medline].
- Metcalf DR, Mondale J, Butler FK: Ontogenesis of spontaneous K-complexes. Psychophysiology 1971 May; 8(3): 340-7[Medline].
- Miller JD, Morin LP, Schwartz WJ, Moore RY: New insights into the mammalian circadian clock. Sleep 1996 Oct; 19(8): 641-67[Medline].
- Moldofsky H: Sleep and the immune system. Int J Immunopharmacol 1995 Aug; 17(8): 649-54[Medline].
- Porkka-Heiskanen T, Strecker RE, Thakkar M, et al: Adenosine: a mediator of the sleep-inducing effects of prolonged wakefulness. Science 1997 May 23; 276(5316): 1265-8[Medline].
- Russo M, Thorne D, Thomas M, et al: Sleep deprivation induced Balint's syndrome (peripheral visual field neglect): a hypothesis for explaining driving simulator accidents in awake but sleepy drivers. Sleep 1999; 22(Suppl 1): 327.
- Russo MB, Sing H, Santiago S, et al: Visual neglect: occurrence and patterns in pilots in a simulated overnight flight. Aviat Space Environ Med 2004 Apr; 75(4): 323-32[Medline].
- Russo MB, Kendall A, Johnson D: Visual perception, psychomotor performance, and complex motor performance during an overnight air refueling simulated flight. Aviat Space Environ Med 2005; 76: In press.
- Steriade M, McCarley RW: Brainstem Control of Wakefulness and Sleep. New York: Plenum; 1990.
- Steriade M, Biesold D, eds: Brain Cholinergic Systems. New York: Oxford University Press; 1990.
- Steriade M, Contreras D, Curro Dossi R, Nunez A: The slow (< 1 Hz) oscillation in reticular thalamic and thalamocortical neurons: scenario of sleep rhythm generation in interacting thalamic and neocortical networks. J Neurosci 1993 Aug; 13(8): 3284-99[Medline].
- Stern J: Eye activity measures of fatigue, and napping as a countermeasure. [USDOT Technical Report] FHWA-MC-99-028. January 1999. 26.
- Szymusiak R: Magnocellular nuclei of the basal forebrain: substrates of sleep and arousal regulation. Sleep 1995 Jul; 18(6): 478-500[Medline].
- Thomas M, Balkin T, Sing H, et al: PET imaging studies of sleep deprivation and sleep: implications for behavior and sleep function [abstract]. J Sleep Res 1998; 7(Suppl 2): 274.
- Thomas M, Thorne D, Sing H, et al: The relationship between driving accidents and microsleep during cumulative partial sleep deprivation. J Sleep Res 1998; 7 (Suppl 2): 275.
- Thomas M, Sing H, Belenky G, et al: Neural basis of alertness and cognitive performance impairments during sleepiness. I. Effects of 24 h of sleep deprivation on waking human regional brain activity. J Sleep Res 2000 Dec; 9(4): 335-52[Medline].
- Thomas M, Sing H, Belenky G: Neural basis of alertness and cognitive performance impairments during sleepiness. II. Effects of 48-72 hours of sleep deprivation on waking human regional brain activity. Thalamus Relat Syst 2003; 2: 199-229.
- Thorne D, Thomas M, Sing H, et al: Accident rate, attention and performance in a driving simulator during 64 hours of progressive sleep deprivation. Sleep Res 1997; 26(Suppl 1): 634.
- Thorne D, Thomas M, Sing H, et al: Driving-simulator accident rates before, during, and after one week of restricted night sleep. Sleep 1998; 21(Suppl 3): 235.
- Thorne D, Thomas M, Russo M, et al: Performance on a driving-simulator divided attention task during one week of restricted nightly sleep. Sleep 1999; 22(Suppl 1): 301.
- Welsh A, Thomas M, Thorne D, et al: Effect of 64 hours of sleep deprivation on accidents and sleep events during a driving simulator. Sleep 1998; 21(Suppl 3): 234.
- Wever RA: The Circadian System of Man: Results of Experiments Under Temporal Isolation. New York: Springer; 1979.
- White RM Jr: The Lengths of Sleep. Washington, DC: American Psychological Association; 1975.
- Williams RL, Karacan I, Hursch CJ: Electroencephalography (EEG) of Human Sleep: Clinical Applications. New York: Wiley; 1974.

Normal Sleep, Sleep Physiology, and Sleep Deprivation: General Principles excerpt

About Us | Privacy | Terms of Use | Contact Us | Advertise | Institutional Subscribers