

# SLEEP

## Normal sleep



### BACKGROUND

Sleep has been considered a reversible stage of unconsciousness but in reality, it's a time when the body is hard at work. The normal physiology of sleep is subject to sleep drivers and follows a specific architecture that includes both a non-rapid-eye-movement (NREM) component and a rapid-eye-movement (REM) component. Each plays a role in the work of sleep. Dental clinicians should understand the normal physiologic states during sleep so as to have a better comprehension of what sleep-disordered breathing does to the body and specifically what happens during apneic episodes.

### SLEEP DRIVERS

Humans are active during the day and physiologically programmed to sleep during the night. This diurnal cycle can be overridden, allowing the performance of work at normal sleep times and sleep during the daytime. Two drivers contribute collaboratively to maintain wakefulness or sleep (Figure 1). The homeostatic drive to sleep, termed Process S, is linearly related to the time since the previous sleep period. The longer it has been since you've slept, the stronger the desire is to sleep. The drive is related to a buildup of adenosine through the conversion of adenosine triphosphate to adenosine diphosphate and phosphate.

In Process C, which is the complementary drive, the circadian rhythm for sleep and wakefulness operates. During the day, as the homeostatic drive for sleep increases, Process C offsets this drive with a drive to maintain vigilance. Around 2 PM, many people experience a dip in the process and must fight off fatigue or take a nap. As the evening draws near, the circadian oscillations in hormone levels cause the activation of "sleep-on" cells in the basal forebrain and sleep takes over. Wakefulness increases the homeostatic drive to sleep, but sleep diminishes it. The longer the time spent in sleep, the lower the homeostatic drive becomes, then the circadian rhythm of Process C intervenes to consolidate the sleep time.

### SLEEP ARCHITECTURE

Sleep consists of a progressive rhythmic architecture of alternating periods of various types of sleep. It begins with NREM sleep, which passes from light, sleep-onset N1 sleep through 45 to 50 minutes of N2 sleep, and then to deep restorative sleep (N3), which should occupy 20% to 25% of the total time spent in sleep. N3 sleep is also termed deep sleep, slow wave sleep, and sleep stages 3 and 4. Good quality, uninterrupted N3 sleep is essential for the body. During this period there is profound cardiovascular stability, growth hormone secretion, a global drop in blood flow of up to 44%, diminished sleep cortical activity, and synchronized brain waves.

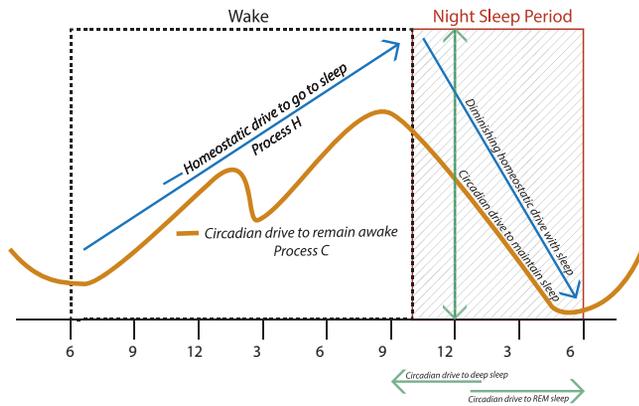
NREM sleep should occupy about 5% of the total sleep time and reappears within the 4 to 5 sleep cycles that occur during the night. The brain is essentially idling during this time, although the body can move.

After N3 sleep, the level of sleep progressively lightens through N2 and N1 up to REM sleep. Over the course of this process, brain wave frequency gradually increases until it resembles that of the waking state. During REM sleep, brain waves are desynchronized, blood flow to the brain and brain metabolism increase, and vivid dreaming may occur. Although the brain is highly active, the body's motor function is inhibited. Normal REM sleep should account for 20% to 25% of the total sleep time, with the first period occurring 70 to 90 minutes after sleep commences. Initially the duration of REM sleep is short, but with subsequent REM periods, the time lengthens.

The early sleep segment, from 9 PM to midnight, seems to favor deep restorative sleep, with the latter hours of the night devoted to REM sleep by the circadian drive. Thus, going to bed too late can decrease the opportunity to experience deep restorative sleep, and REM latency may be less than the usual 70 to 90 minutes. A short REM latency can be a sign of narcolepsy.

### REM SLEEP

Just as NREM sleep is the opportunity for the body to reset its mechanisms, REM sleep is the period for the brain to reset itself.



**Figure 1.** Physiologic drivers to sleep. Two complimentary physiologic processes interplay to maintain wakefulness during the day and consolidate sleep during the night. Process H is the physiologic drive to go to sleep. It increases linearly with sleep debt. Process C is the internal circadian rhythm that competes with Process H to maintain wakefulness during the day and conversely maintains sleep during the night as Process H diminishes as sleep is achieved. There is a circadian drive to be in deep sleep in the early part of the night and to be in REM sleep in the latter half. (Courtesy of Cole A-M: Understanding normal sleep, respiration and circulation. *CDA J* 46:513-517, 2018.)

REM sleep has a significant effect on emotional stability. Dysregulation may be a presaging and perpetuating factor in mood disorders, including depression. During REM sleep, a group of cells that produce the monoamine neurotransmitters cease their activity, which is substantial during wakefulness. They rest and recover during REM sleep so they can resume their activities during wakefulness. Disruption of the monoamine pathways can cause dysfunction in multiple systems. The effects can alter emotional stability, cognitive processing, hormonal stability, circadian rhythms, appetite control, and functional movement.

REM sleep occupies about 50% of a newborn's total sleep time but the percentage decreases with age. Premature infants without consolidated high REM sleep percentages are at increased risk for developmental delays compared to preterm infants who have high-quality REM profiles. Ontogenetic development can be altered by poor REM sleep.

## EFFECT ON BODY SYSTEMS

Several physiologic changes occur with sleep onset that involve the respiratory and cardiovascular systems. Oxygen saturation drops 2%, and carbon dioxide partial pressure increases 2 to 8 mm Hg related to the loss of the wakefulness drive to breathe, decreased basal metabolism, low muscle tone, reduced sensitivity of the chemoreceptors, and diminished lung capacity

related to the body's recumbent position. Survival becomes dependent solely on the autonomic systems. Blood pressure dips cyclically throughout the night. During this time the cardiovascular system can recuperate and reset for the next wakefulness period.

REM sleep increases parasympathetic tone but permits interspersed periods of very high sympathetic activity associated with the bursts of rapid eye movement. This results in a period of cardiovascular variability and potential instability. REM sleep can be a period of high risk for patients who have poor cardiovascular function.

The respiratory system is also altered during REM sleep. The homeostatic blood gas levels are increased for carbon dioxide, which is referred to as the apneic threshold. If the level drops below this threshold, a centrally mediated pause in breathing (central apnea) can occur, lasting until carbon dioxide retention increases again. A significant increase in carbon dioxide can cause increased breathing to get rid of the excess. Central apnea is not uncommon at the onset of sleep, but this is a physiologic, not a pathologic, mechanism, although it can signal unstable breathing control mechanisms. It's important to realize that the apneic threshold is not the same for everyone.

Respiration during N2 sleep is unstable, and susceptible patients can also experience apnea with irregular breathing. During steady N3 sleep, breathing is regular in rate and amplitude, making this the least risky period for airway obstruction.

### Clinical Significance

Understanding the natural fluctuations of sleep and the driving forces behind sleep's architecture and effects on body systems will help clinicians better understand how patients can experience sleep-disordered breathing. Being able to manage the care of these patients as part of a multidisciplinary team offers the opportunity to make life-altering changes in their quality of life and their overall health.

Cole A-M: Understanding normal sleep, respiration and circulation. *CDA J* 46:513-517, 2018

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