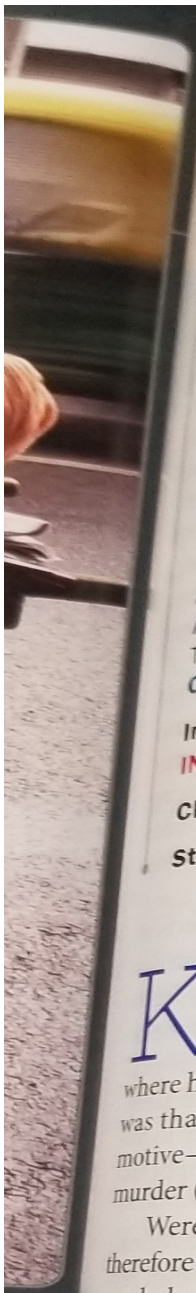


Sleep and Consciousness

Kenneth Parks got up from the couch where he had been sleeping and drove 14 miles to his in-laws' home. There he struggled with his father-in-law before stabbing his mother-in-law repeatedly, killing her. He then drove to the police station, where he told the police that he thought he had "killed some people." In court, his defense was that he was sleepwalking. Based on the testimony of sleep experts and the lack of motive—Ken had an affectionate relationship with his in-laws—the jury acquitted him of murder (Broughton et al., 1994).

Were Ken's actions possible for someone who was sleepwalking? Was he really asleep and therefore not responsible? This case raises the question of what we mean by *consciousness*. Many psychologists, and especially neuroscientists, avoid the topic because they think that consciousness is inaccessible to research. This has not always been so; consciousness was a major concern of the fledgling discipline of psychology near the end of the 19th century. But the researchers' technique of *introspection* was subjective: The observations were open only to the individuals doing the introspecting, who often disagreed with each other. This failing encouraged the development of behaviorism, which was based on the principle that psychology should study only the relationships between external stimuli and observable responses. Behaviorism was a necessary means of cleansing psychology of its subjective methods, but its purge discarded the subject matter along with the methodology. The interests of psychologists would not shift back to include internal experience until the emergence of the field of *cognitive psychology* in the 1950s and 1960s.

Many cognitive psychologists were finding it difficult to understand psychological functions such as learning and perception without taking account of various aspects of consciousness. Still, few of them tackled the subject of consciousness itself. The problem seemed too big, there was no clear definition of consciousness, and the bias that consciousness was a problem for philosophers still lingered. Gradually, some of them began to ally themselves with philosophers, biologists, and computer experts to develop new research strategies for exploring this

last frontier of psychology. The greatest inroads have been made in the study of sleep, largely because sleep is readily observable. Also, because sleep is open to study by objective techniques, it has not had the stigma among researchers that characterizes other aspects of consciousness. We will begin this last leg of our journey with the topic of sleep and dreaming.

Sleep and Dreaming

Each night, we slip into a mysterious state that is neither entirely conscious nor unconscious. Sleep has intrigued humans throughout history: Metaphysically, dreaming suggested to our forebears that the soul took leave of the body at night to wander the world; practically, sleep is a period of enforced nonproductivity and vulnerability to predators and enemies.

In spite of thousands of research studies, we are still unclear on the most basic question—What is the function of sleep? The most obvious explanation is that sleep is *restorative*. Support for this idea comes from the observation that species with higher metabolic rates typically spend more time in sleep (Zepelin & Rechtschaffen, 1974). A less obvious explanation is the *adaptive* hypothesis; according to this view, the amount of sleep an animal engages in depends on the availability of food and on safety considerations (Webb, 1974). Elephants, for instance, which must graze for many hours to meet their food needs, sleep briefly. Animals with low vulnerability to predators, such as the lion, sleep much of the time, as do animals that find safety by hiding, like bats and burrowing animals. Vulnerable animals that are too large to burrow or hide—for example, horses and cattle—sleep very little (Figure 15.1). In a study of 39 species, the combined factors of body size and danger accounted for 80% of the variability in sleep time (Allison & Cicchetti, 1976).

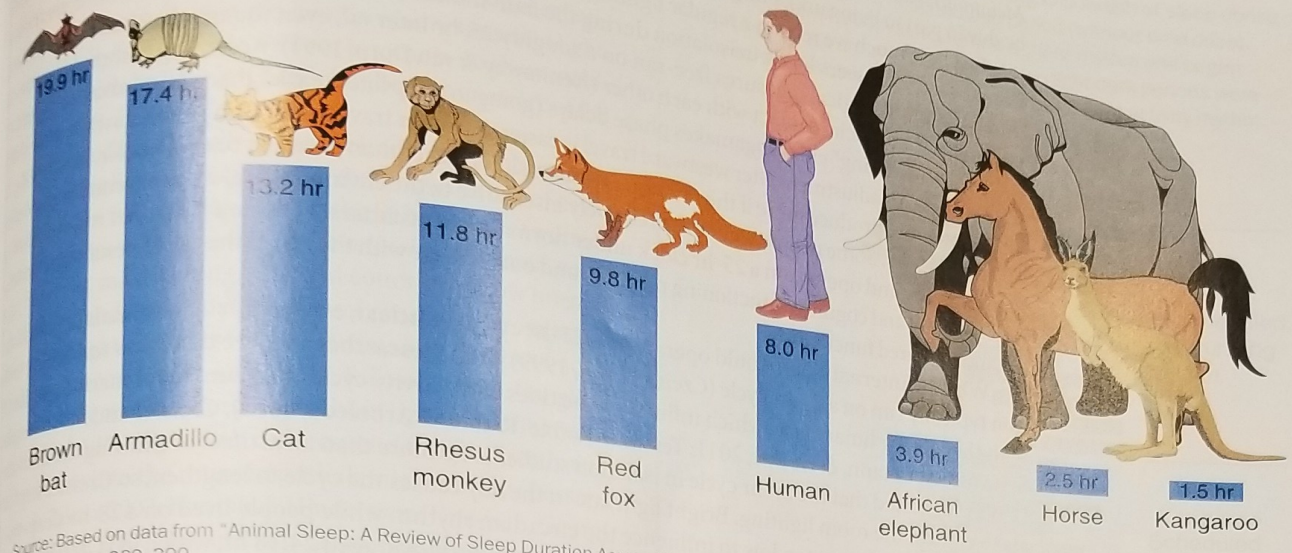
An interesting new idea is that the brain cleanses itself of toxins during sleep. Researchers at the University of Rochester in New York recently discovered a network of channels formed by glia, which transport cerebrospinal fluid (CSF) through the brain. By injecting a colored dye in the brains of mice as they slept and a dye of another color when they woke up, the researchers determined that large amounts of CSF flowed through the brain during sleep but not during the awake state. The researchers then injected β-amyloid proteins into the brains of the mice and found that the CSF cleared the proteins out of cells twice as fast during sleep (Xie et al., 2013). (Students note: This is one more argument for getting enough sleep!)

Whatever the function of sleep may be, its importance becomes apparent when we look at the effects of sleep deprivation. These effects are nowhere more evident than in shift work. Shift workers sleep less than day workers, and their work performance suffers as a result (Tepas & Carvalhais, 1990). Also, they typically fail to adjust their sleep-wake cycles adequately, because their sleep is disturbed during the day and they conform to the rest of the world's schedule on weekends. With their work and sleep schedules at odds with their biological rhythms, shift workers find that sleep intrudes into their work and daytime arousal interferes with their sleep. Another study with mice may give us a clue why this happens. After just a few days of sleeping three to five hours a day, the mice had lost 25% of the neurons in the locus coeruleus, a part of the brain that is important for alertness (J. Zhang et al., 2014).

In long-term sleep deprivation studies, impairment follows a rhythmic cycle—performance declines during the night and then shows some recovery during the daytime (Horne, 1988). The persistence of this rhythm represents a safety hazard of gigantic proportions when people try to function at night. The largest number of single-vehicle traffic accidents attributed to "falling asleep at the wheel" occur around 2 a.m. (Mitler et al., 1988), and the number of work errors peaks at the same time (Broughton, 1975). In addition, the Three Mile Island nuclear plant accident took place at 4 a.m.; the Chernobyl nuclear plant meltdown began at 1:23 a.m.; the Bhopal, India, chemical plant leakage, which poisoned more than 2,000 people, began shortly after midnight; and the *Exxon Valdez* ran aground at 12:04 a.m., spilling 11 million gallons of oil into fragile Alaskan waters (Alaska Oil Spill Commission, 1990; Mapes, 1990; Mitler et al., 1988).

Travel across time zones also disrupts sleep and impairs performance, particularly when you travel eastward. It is difficult to quantify the effects of *jet lag*, but three researchers have attempted to do so in a novel way by comparing the performance of baseball teams. When East Coast and West Coast teams played at home, their percentage of wins was nearly identical—50% and 49%, respectively. When they traveled across the continent but had time to adjust to the new time zone, they showed a typical visitor's disadvantage, winning 45.9% of their games. Teams traveling west without time to adjust won about the same, 43.8% of their games, whereas teams traveling east won only 37.1% (Recht, Lew, & Schwartz, 1995). Sleep quality is better when you extend the day's length by traveling west, rather than shorten it as you do when you travel east. One way of looking at this effect is that it is easier to stay awake past your bedtime than it is to go to sleep when you are not sleepy. We will examine a more specific explanation in the next section, when we consider circadian rhythms.

FIGURE 15.1 Time Spent in Daily Sleep for Different Animals.
Observations support the hypothesis that sleep is an adaptive response to feeding and safety needs.



Source: Based on data from "Animal Sleep: A Review of Sleep Duration Across Phylogeny," by S. S. Campbell and I. Tobler, 1984, *Neuroscience and Biobehavioral Reviews, 8*, pp. 269–300.

Circadian Rhythms

We saw in Chapter 14 that a *circadian rhythm* is a rhythm that is about a day in length; the term *circadian* comes from the Latin *circa,* meaning "approximately," and *dia,* meaning "day." We operate on a 24-hour (hr) cycle, in synchrony with the solar day. We sleep once every 24 hr, and body temperature, alertness, urine production, steroid secretion, and a variety of other activities decrease during our normal sleep period and increase during our normal waking period, even when we reverse our sleep-wake schedule temporarily.

? Why are circadian rhythms important?

The main biological clock that controls these rhythms in mammals is the *suprachiasmatic nucleus (SCN)* of the hypothalamus. Lesioning the SCN in rats abolishes the normal 24-hr rhythms of sleep, activity, body temperature, drinking, and steroid secretion (Abe, Kroning, Greer, & Critchlow, 1979; Stephan & Nunez, 1977). The SCN is what is known as a *pacemaker,* because it keeps time and regulates the activity of other cells. We know that the rhythm arises in the SCN, because rhythmic activity continues in isolated SCN cells (Earnest, Liang, Ratcliff, & Cassone, 1999; Inouye & Kawamura, 1979). Lesioned animals do not stop sleeping, but instead of following the usual day-night cycle, they sleep in naps scattered throughout the 24-hr period. So the SCN controls the timing of sleep, but sleep itself is controlled by other brain structures, which we will discuss later. The SCN is shown in Figure 15.2, and you can check Figure 6.2 to see its location.

The SCN is *entrained* to the solar day by cues called *zeitgebers* ("time-givers"). If humans are kept in isolation from all time cues in an underground bunker or a cave, they usually lose their synchrony with the day-night cycle; in many studies, zeitgeber-deprived individuals "drifted" to a day that was about 25 hr long, with a progressively increasing delay in sleep onset (Figure 15.3; Aschoff, 1984). For a long time, researchers believed that alarm clocks and the activity of others were the most important influences that entrain our activity to the 24-hr day, but research points more convincingly to light as the primary zeitgeber.

> "Early to bed and early to rise, makes a man healthy, wealthy, and wise."
>
> —Benjamin Franklin

The *difference* in light intensity between the light and dark periods is important for entraining the day-night cycle. One group of night workers worked under bright lights and slept in complete darkness during the day (*light discrepant*); a second group worked under normal light and slept in the semidarkness that is typical of the day sleeper (*similar light*). The light-discrepant workers scored higher in performance and alertness than the similar-light workers. Their physiological measures also synchronized with the new sleep-wake cycle; for example, their body temperature dropped to its low value around 3:00 p.m., when they were asleep, but the

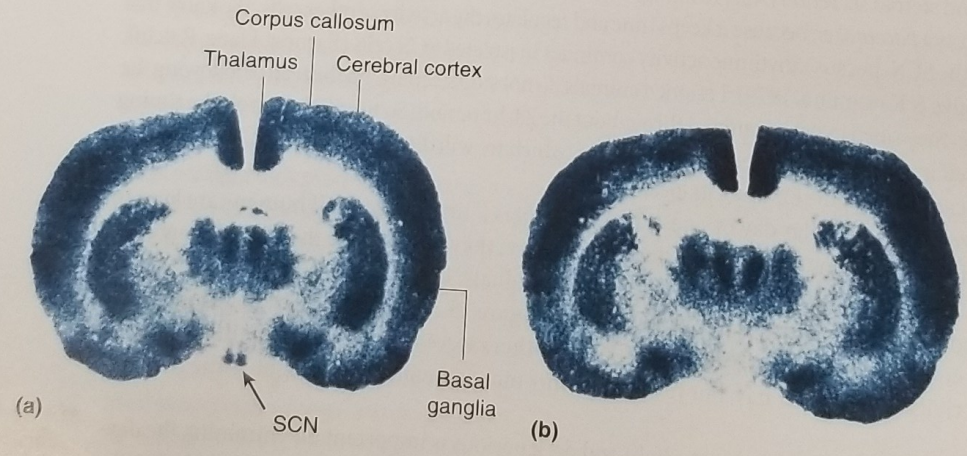
similar-light group's low continued to occur at 3:30 a.m. in spite of being awake and working (Czeisler et al., 1990). If you're thinking the take-home from this study is that you should keep the lights as bright as possible when you're awake, you would be wrong. Compared with dim light, exposure to normal room light for the 8-hr period before bedtime reduces the duration of melatonin release by 90 minutes each night (Gooley et al., 2010). *Melatonin* is a hormone that induces sleepiness. The typically sleep-deprived state in modern society appears to be due in part to living under bright lights and spending evenings in front of a computer.

Just how much we rely on a regular lighting schedule for entrainment was underscored in a study of four Greenpeace volunteers living in isolation during the four-month darkness of the Antarctic winter; their sleep times and physiological measures free-ran on a roughly 25-hr interval, even though they had access to time information and social contact with each other (Kennaway & van Dorp, 1991). According to some observers, it is this "slow-running" clock that makes phase delays (going to sleep later) easier than phase advances (going to sleep earlier). So adjustment after westward travel is easier than after traveling east, and workers who rotate shifts sleep better and produce more if the rotation is to later shifts rather than to earlier ones (Czeisler, Moore-Ede, & Coleman, 1982). Some people seem to be relatively insensitive to the environmental cues that entrain most of us to a 24-hr day and operate on a 25-hr clock under normal circumstances; and like a clock that runs too slowly, their physical and cognitive functioning moves in and out of phase with the rest of the world, resulting in insomnia and impaired functioning.

Why the internal clock would operate on a 25-hr cycle is unclear, especially since animals kept in isolation typically run on a 24-hr cycle (Czeisler et al., 1999). Some researchers believe that it has something to do with the 24.8-hr lunar cycle, which influences the tides and activity cycles of a number of marine species (T. S. Kaiser, Neumann, & Heckel, 2011; Tessmar-Raible, Raible, & Arboleda, 2011). Czeisler and his colleagues (1999) suggested that the 25-hr cycle in isolation studies is no more than an artifact of allowing the individuals to control the room lighting. Bright light late in the day causes the cycle to lengthen, so Czeisler kept the light at a level that was too low to influence the circadian rhythm while people lived on a 28-hr sleep-wake schedule. Under that condition, their body temperature cycle averaged 24.18 hr, which led Czeisler to conclude that the *biological* rhythm is approximately 24 hr long. By the way, if you think a lunar influence on sleep is hard to believe, Swiss researchers have preliminary evidence for it. Near the time of the full moon, volunteers sleeping in a windowless laboratory slept 20 minutes less, produced half as much melatonin, and had 30% less of one stage of sleep than at other times of the month (Cajochen et al., 2013). The researchers believe this observation represents an inherent biological rhythm, but they admit that the results could have been influenced by exposure to brighter evening light before their subjects entered the laboratory.

FIGURE 15.2 The Suprachiasmatic Nucleus.

(a) The nuclei, indicated by the arrows, took up more radioactive 2-deoxy-glucose in the scan because the rat was injected during the "light-on" period of the day; (b) the rat was injected during the "light-off" period.



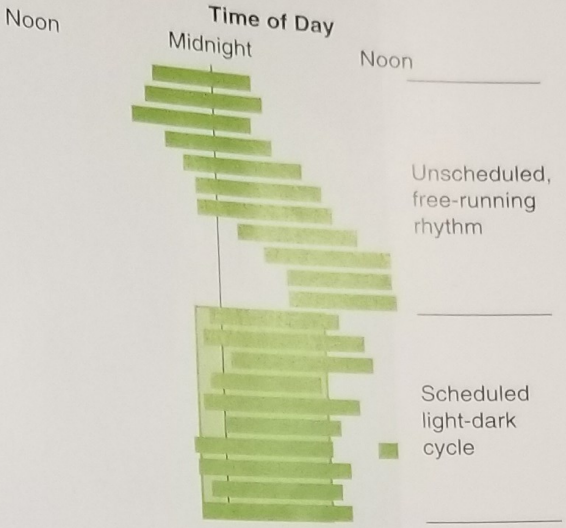
Reprinted with permission from W. J. Schwartz and H. Gainer, "Suprachiasmatic Nucleus: Use of 14C-labeled Deoxyglucose Uptake as a Functional ." *Science, 197*, 1089–1091. Copyright 1977 American Association for the Advancement of Science.

the SCN regulates the pineal gland's secretion of melatonin. ...tonin is often used to combat jet lag and to treat insom... ...shift workers and in the blind (Arendt, Skene, Middleton, ...ker, & Deacon, 1997). Light resets the biological clock by sup... ...ssing melatonin secretion (Boivin, Duffy, Kronauer, & Czeisler, ...). Most totally blind individuals are not entrained to the ...y day and suffer from insomnia in spite of regular schedules ...ping, work, and social contact. These individuals do not experi... ...a decrease in melatonin production when exposed to light; ...ever, totally blind people *without* insomnia do show mela... ...in suppression by light, even though they are unaware of the ...m (Czeisler et al., 1995).

Animal studies explain how some blind individuals are able ...ntrain to the light-dark cycle and, thus, how the rest of us do ...as well. Light information reaches the SCN by way of a direct ...nection from the retinas called the *retinohypothalamic path*... ...However, mice lacking rods and cones still show normal ...ainment and cycling, so the signal must arise from some ...her retinal light receptors (M. S. Freedman et al., 1999; Lucas, ...eedman, Muñoz, Garcia-Fernández, & Foster, 1999). Ganglion ...s ordinarily receive information about light from the recep... ...s, but about 1% of ganglion cells respond to light directly and ...nd neurons into the retinohypothalamic pathway (Berson, ...ma, & Takao, 2002; Hannibal, Hindersson, Knudsen, Georg, ...Fahrenkrug, 2002; Hattar, Liao, Takao, Berson, & Yau, 2002). ...ese ganglion cells contain melanopsin, which is a light-sensi... ...substance, or photopigment (Dacey et al., 2005; Panda et al., ...5; Qiu et al., 2005). The melanopsin is located in their widely ...nching dendrites, which suits the cells for detecting the over... ...level of light, as opposed to contributing to image formation ...(Figure 15.4). A study has confirmed that human retinas have ...elanopsin in some of their ganglion cells (Dkhissi-Benyahya, ...ieux, Hut, & Cooper, 2006).

FIGURE 15.3 Sleep and Wake Periods During Isolation From Time Cues.

Each dark bar indicates the timing and length of sleep during a day. During the unscheduled period (without time cues), the subject's activity assumed a 25-hr rhythm and began to advance around the clock. When light-dark periods were scheduled, he resumed a normal sleep and activity rhythm.



Source: From *Introduction to Psychology, Gateways to Mind and Behavior* (with InfoTrac) 9th edition, by D. Coon, 2001. Reprinted with permission of Wadsworth, a division of Thomson Learning.

However, synchronizing the rhythm does not account for the rhythm itself. The internal clock consists of ...few genes and their protein products (Clayton, Kyriacou, & Reppert, 2001; Hastings, Reddy, & Maywood, ...2003; Shearman et al., 2000); the genes fall into two groups, one group that is turned on while the other is ...turned off. When the genes are on, their particular protein products build up. Eventually, the accumulating ...proteins turn their genes off, and the other set of genes is turned on. This feedback loop provides the approxi... ...mately 24- or 25-hr cycle, which then must be reset each day by light. This process is not limited to neurons ...in the SCN; there are additional clocks, located outside the brain and controlling the activities of the body's ...organs (Hastings et al., 2003). These clocks operate independently of the SCN, but the SCN entrains them to ...the day-night cycle. Feeding is an example of an activity that is controlled independently. According to the ...researchers, local clocks that affect blood pressure and heart activity explain why there is a large increase in ...the risk of heart attack, stroke, and sudden cardiac death after waking in the morning. The clock in the SCN ...does not always operate properly, as we saw in Chapter 14 with some depressed patients.

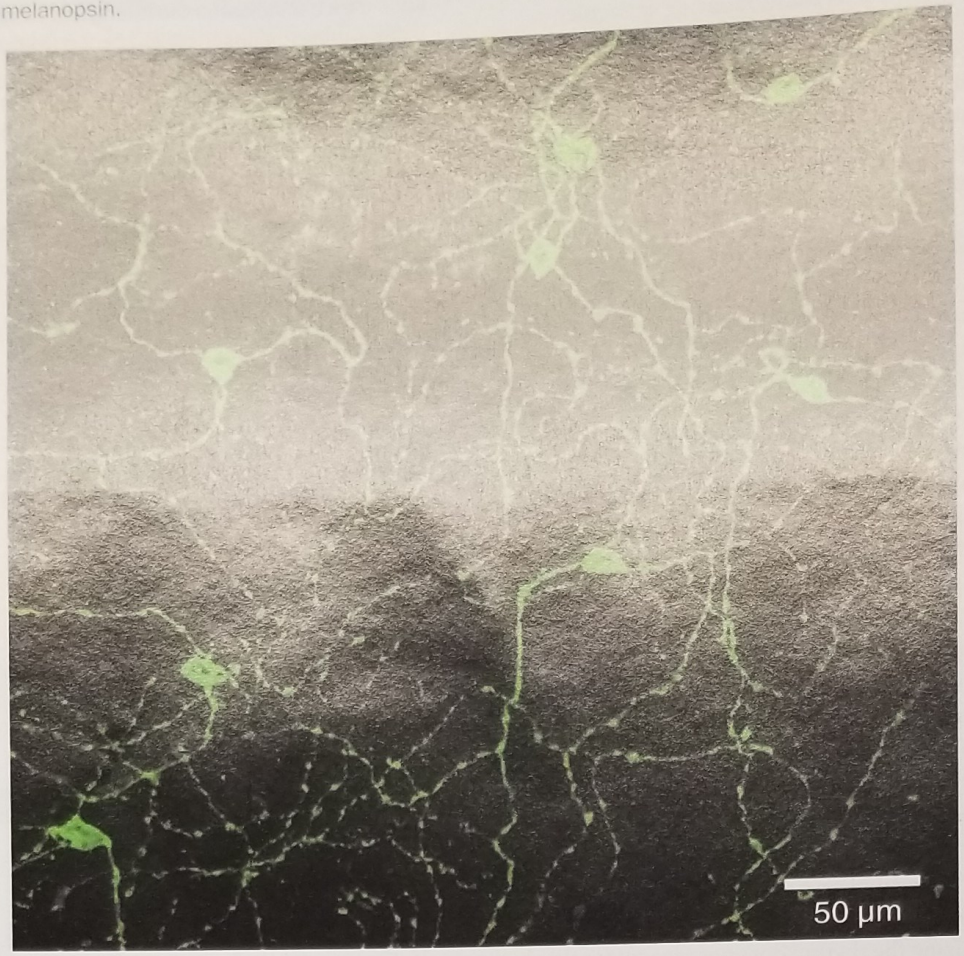
Rhythms During Waking and Sleeping

Riding on the day-long wave of the circadian rhythm are several *ultradian rhythms*, rhythms that are shorter ...than a day in length. Hormone production, urinary output, alertness, and other functions follow regular ...cycles throughout the day. For example, the dip in alertness and performance in the wee hours of the morning ...is mirrored by another in the early afternoon, which cannot be accounted for by postlunch sleepiness, because ...it also occurs in people who skip lunch (Broughton, 1975). Incidentally, this dip coincides with the time of ...siesta in many cultures and a rest period in nonhuman primates. The *basic rest and activity cycle* is a rhythm ...that is about 90–100 minutes (min) long. When people wrote down what they were thinking every 5 min for

? What rhythms occur throughout the day and night?

FIGURE 15.4 Retinal Ganglion Cells Containing Melanopsin.

The cells were labeled with a fluorescent substance that reacts to melanopsin. Notice the widespread dendrites, which contain melanopsin.



Source: From "Melanopsin-Containing Retinal Ganglion Cells: Architecture, Projections, and Intrinsic Photosensitivity," by S. Hattar, H. W. Liao, M. Takao, D. M. Berson, and K. W. Yau, *Science*, 295, pp. 1065–1070. © 2002 American Association for the Advancement of Science (AAAS). Reprinted with permission from AAAS.

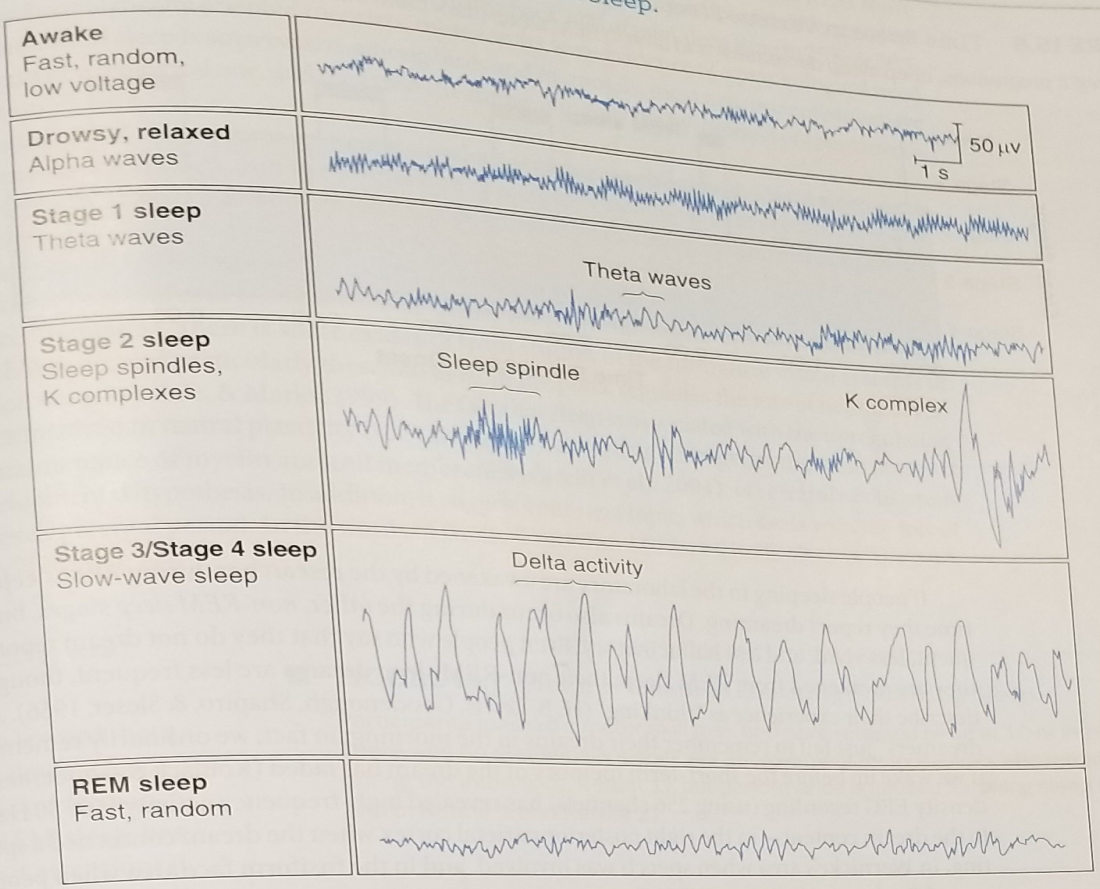
10 hr, the contents showed that they were daydreaming on a 90-min cycle; EEG recordings verified that these were periods of decreased brain activity (Kripke & Sonnenschein, 1973).

The common view of sleep is that it is a cessation of activity that occurs when the body and brain become fatigued. Sleep, however, is an active process. This is true in two respects. First, you will soon see that sleep is a very busy time; a great deal of activity goes on in the brain. Second, sleep is not like a car running out of gas but instead is turned on by brain structures and later turned off by other structures.

The most important measure of sleep activity is the EEG. When a person is awake, the EEG is a mix of *alpha* and *beta* waves. Alpha is activity whose voltage fluctuates at a frequency of 8–12 hertz (Hz) and moderate amplitude; beta has a frequency of 13–30 Hz and a lower amplitude. Beta waves, which are associated with arousal and alertness, are progressively replaced by alpha waves as the person relaxes (Figure 15.5). It may seem strange that the amplitude of the EEG is lower during arousal. Remember that the EEG is the sum of the electrical activity of all the neurons between the two recording electrodes. When a person is cognitively aroused, neurons under the electrodes are mostly desynchronized in their firing as they carry out their separate tasks; with the neurons firing at different times, the EEG has a high frequency, but the amplitude is rather low. As the person relaxes, the neurons have less processing to do and fall into a pattern of synchronized firing. The rate is low, but the cumulative amplitude of the neurons firing at the same time is high.

As the person slips into the light first stage of sleep, the EEG shifts to *theta* waves, with a frequency of 4–7 Hz (Figure 15.5). About 10 min later, Stage 2 begins, indicated by the appearance of *K complexes* and *sleep*

FIGURE 15.5 Electroencephalogram and the Stages of Sleep.

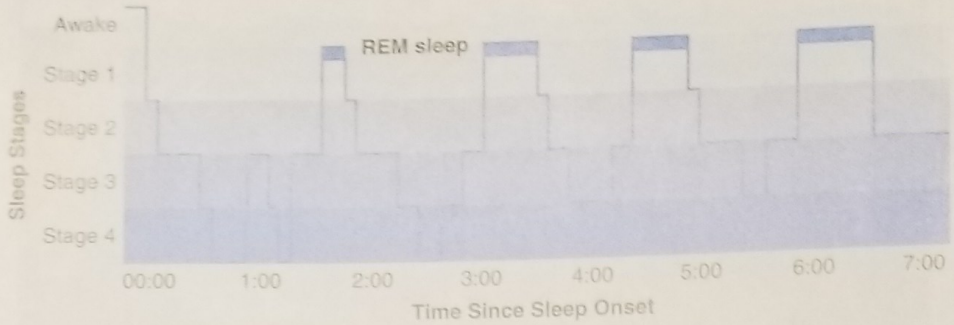


Source: From *Current Concepts: The Sleep Disorders*, by P. Hauri, 1982, Kalamazoo, MI: Upjohn.

spindles. K complexes are sharp, large waves that occur about once a minute; sleep spindles are brief bursts of 12- to 14-Hz waves that appear to serve a gating function, preventing disruptive stimuli from reaching the cortex and waking the sleeper (Dang-Vu, McKinney, Buxton, Solet, & Ellenbogen, 2010). Stages 3 and 4 are known as *slow-wave sleep* and are characterized by large, slow delta waves at a frequency of 1–3 Hz. The person moves around in bed during this period, turning over and changing positions. Sleepwalking, bedwetting, and night terrors, disturbances that are common in children, occur during slow-wave sleep, too. Night terrors are not nightmares but involve screaming and apparent terror, which are usually forgotten in the morning; they are not a sign of a disorder unless they continue beyond childhood. After Stage 4, the sleeper moves rather quickly back through the stages in reverse order. But rather than returning to Stage 1, the sleeper enters rapid eye movement sleep.

Rapid eye movement (REM) sleep is so called because the eyes dart back and forth horizontally during this stage. The EEG returns to a pattern similar to a relaxed waking state, but the person does not wake up; in fact, the sleeper is not easily aroused by noise but does respond to meaningful sounds, such as the sleeper's name. It is easy to see why some researchers call this stage *paradoxical sleep*, because *paradoxical* means "contradictory." During REM sleep, respiration rate and heart rate increase. Males experience genital erection, and vaginal secretion increases in females. In spite of these signs of arousal, the body is very still—in fact, in a state of muscular paralysis or *atonia*. A complete cycle through the stages of sleep—like the daydreaming cycle—takes about 90 min to complete. The night's sleep is a series of repetitions of this ultradian rhythm, although the length of REM sleep periods increases and the amount of slow-wave sleep decreases through the night (Figure 15.6).

FIGURE 15.6 Time Spent in Various Sleep Stages During the Night.
As the night progresses, deep sleep decreases, and time in REM sleep (dark bars) increases.



If people sleeping in the laboratory are awakened by the researcher during REM sleep, about 80% of the time they report dreaming. Dreams also occur during the other, *non-REM sleep* stages, but they are less frequent, less vivid, and less hallucinatory. Even people who say that they do not dream report dreaming when they are awakened from REM sleep; their non-REM sleep dreams are less frequent, though, and they often describe their experience as "thinking" (H. B. Lewis, Goodenough, Shapiro, & Sleser, 1966). Apparently, "non-dreamers" just fail to remember their dreams in the morning; in fact, we ordinarily remember a dream only if we wake up before the short-term memory of the dream has faded (Koulack & Goodenough, 1976). High-density EEG recording (using 256 channels) has revealed high-frequency activity (10–50 Hz) in areas related to the dream content—in the right posterior parietal cortex when the dream concerned a specific spatial setting, in Wernicke's area when speech was involved, and in the fusiform face area when people's faces were a part of the dream (Siclari et al., 2017).

The Functions of Sleep

What are the functions of REM and slow-wave sleep?

The effects of sleep loss leave no doubt about the importance of sleep. You have seen, for example, that the early morning hours are particularly vulnerable times for traffic accidents and major disasters. Cognitive impairment suffers especially; reducing sleep time to 6 hr or 4 hr for 14 days reduced alertness and working memory performance, with deficits equivalent to one night or two nights of total sleep deprivation, respectively (Van Dongen, Maislin, Mullington, & Dinges, 2003). Early research efforts were based on the idea that the main function of sleep is rest and restoration, with the major focus on non-REM sleep. One reason is that after total sleep deprivation, Stage 4 non-REM sleep is recovered before REM sleep (Anders & Roffwarg, 1973). Another reason is that slow-wave sleep increases following exercise; after athletes competed in a 92-kilometer race, slow-wave sleep was elevated for four consecutive nights (Shapiro, Bortz, Mitchell, Bartel, & Jooste, 1981). However, this effect appears to be due to overheating rather than fatigue. The night after people ran on treadmills, slow-wave sleep increased, at the expense of REM sleep; but if they were sprayed with water while they ran, their body temperature increased less than half as much and there was no change in slow-wave sleep (Horne & Moore, 1985). Horne & Harley (1989) believed that the slow-wave sleep increases are more related to the increase in the temperature of the brain than the increase in body temperature; heating only the head and face with a hair dryer was sufficient to increase slow-wave sleep later. According to Horne (1992), slow-wave sleep promotes cerebral recovery, especially in the prefrontal cortex, and there is evidence it restores processes involved in cognitive functioning. M. H. Bonnet and Arand (1996) gave people either caffeine or a placebo before a 3.5-hr nap. The caffeine group had reduced slow-wave sleep during the nap; although they felt more vigorous and no sleepier than the placebo group, they performed less well on arithmetic and vigilance tasks during a subsequent 41-hr work period.

To find out what functions REM sleep serves, researchers deprived volunteers of REM sleep by waking the research participants every time EEG and eye movement recordings indicated that they were entering a REM

period. When this was done the subjects showed a "push" for more REM sleep. They went into REM more frequently as the study progressed and had to be awakened more often; then, on uninterrupted recovery nights, they tended to make up the lost REM by increasing their REM from about 20% of total sleep time to 25% or 30% (Dement, 1960). To psychoanalytically oriented theorists, these results were evidence of a psychological need for dreaming; but because REM sleep is so pervasive among birds and mammals, most neuroscientists believe that any explanation must be a biological one, and they treat dreaming as merely the by-product of spontaneous neural activity in the brain.

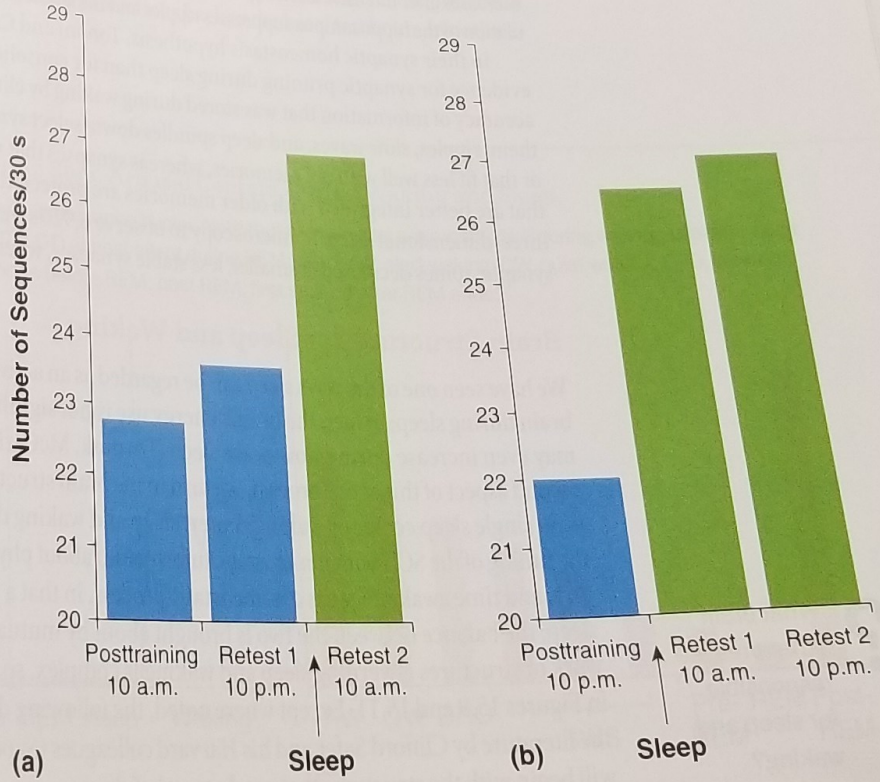
One hypothesis is that REM sleep promotes neural development during childhood. Infant sleep starts with REM rather than non-REM sleep, and the proportion of sleep devoted to REM is around 50% during infancy and decreases through childhood until it reaches an adult level during adolescence (Roffwarg, Muzio, & Dement, 1966). According to this hypothesis, excitation that spreads through the brain from the pons during REM sleep encourages differentiation, maturation, and myelination in higher brain centers, similar to the way spontaneous waves of excitation sweep across the retina during development to help organize its structure (see Chapter 3). There is some evidence from studies of the immature visual systems of newborn cats that REM sleep, and particularly these waves from the pons, regulates the rate of neural development (Shaffery, Roffwarg, Speciale, & Marks, 1999). The fact that sleep is associated with the upregulation of a number of genes involved in neural plasticity (see Chapter 12), as well as other genes that contribute to the synthesis and maintenance of myelin and cell membranes (Cirelli et al., 2004), is certainly consistent with this neurodevelopmental hypothesis. In addition, it suggests our next topic, which deals with the role of sleep beyond the developmental period. In the words of Giulio Tononi and Chiara Cirelli, sleep is "the price we pay for plasticity" (2014, p. 12).

Sleep, Plasticity, and Memory

In Chapter 12, you learned that a period of sleep following learning enhances later performance (Figure 15.7). REM sleep has received the most attention; it increases during the sleep period following learning, and REM sleep deprivation after learning reduces retention (see review in Dujardin, Guerrien, & Leconte, 1990; Karni, Tanne, Rubenstein, Askenasy, & Sagi, 1994; C. Smith, 1995). How much REM sleep increases depends on how well the subject learned (Hennevin, Hars, Maho, & Bloch, 1995). Observation of hippocampal activity after learning suggests why REM sleep is important. Replay during REM sleep of neural activity that occurred during learning (see Chapter 12) is synchronized with spontaneous theta-frequency (4–7 Hz) activity in the hippocampus (Stickgold, Hobson, Fosse, & Fosse, 2001); in other words, the peaks of one wave coincide with the peaks of the other. (You may remember from Chapter 12 that hippocampal theta is necessary for LTP to

FIGURE 15.7 Improvement in Learning Following Sleep.

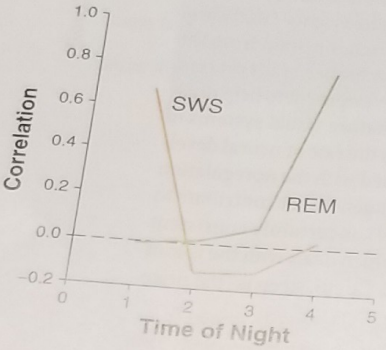
Participants learned a motor skill task and were retested twice at 10-hr intervals. There was no statistically significant improvement for individuals who remained awake during the interval (a, Retest 1), but performance improved following sleep (a, Retest 2, and b, Retest 1 and Retest 2).



Source: Adapted from "Practice With Sleep Makes Perfect: Sleep-Dependent Motor Skill Learning," by M. P. Walker, T. Brakefield, A. Morgan, J. A. Hobson, and R. Stickgold, 2003, Neuron, 35, pp. 205–211.

FIGURE 15.8 Correlation of Slow-Wave and REM Sleep With Overnight Task Improvement.

These graphs show the correlation of slow-wave sleep (SWS) and REM sleep with improvement on a visual discrimination task at the beginning of the next day's practice. They indicate that SWS has more effect during the first quarter of the night, whereas REM is important during the fourth quarter.



Source: Adapted with permission from Stickgold et al., "Sleep, Learning, and Dreams: Off-line Memory Reprocessing," *Science*, 294, pp. 1052–1057. © 2001 American Association for the Advancement of Science. Reprinted with permission from AAAS.

occur.) After four to seven days, the time during which memories become independent of the hippocampus, the replay shifts out of phase with the theta activity, with the peaks of one wave coinciding with the troughs of the other. This suggests that a period of consolidation is followed by one of deleting connections—representing old memories, inaccurate connections, or both.

Some research suggests that consolidation is a multistep process requiring a combination of slow-wave and REM sleep. Overnight improvement on a visual discrimination task in humans was correlated with the percentage of slow-wave sleep during the first quarter of the night *and* the percentage of REM sleep in the last quarter of the night (Figure 15.8; Stickgold, Whidbee, Schirmer, Patel, & Hobson, 2000). Even a 60- to 90-min nap that included both REM and slow-wave sleep produced significant improvement in performance (Mednick, Nakayama, & Stickgold, 2003). According to Ribeiro and his colleagues (2004), neuronal replay is strongest during non-REM sleep, and ample evidence from animal and human studies indicates the importance of non-REM sleep for learning (Hairston & Knight, 2004). For example, applying a 0.75-Hz oscillating current over the frontal and temporal areas or sounding tones timed to coincide with individual EEG waves increases slow-wave activity and improves the recall of word associations learned prior to sleep (L. Marshall, Helgadóttir, Mölle, & Born, 2006; J. L. Ong et al., 2016; Papalambros et al., 2017; Westerberg et al., 2015). Replay during non-REM sleep is accompanied by 200-millisecond bursts of fast-frequency (100–120 Hz) activity known as *ripples*, which are generated by the hippocampus and activate the default mode network, which is otherwise quiescent during non-REM sleep (Kaplan et al., 2016; M. P. Walker & Robertson, 2016). The number of ripples increases after intensive learning, and this increase predicts the success of memory consolidation; electrical stimulation of the hippocampus suppresses ripples and impairs learning.

In their synaptic homeostasis hypothesis, Tononi and Cirelli (2013, 2014) argue that there is far better evidence for synaptic pruning during sleep than for consolidation; they believe this pruning improves the accuracy of information that was stored during waking by eliminating inaccurate connections. According to them, ripples, slow waves, and sleep spindles down-select synapses that were activated rarely during waking or that fit less well with old memories, whereas synapses that were strengthened repeatedly during waking or that are better integrated with older memories are protected. Luisa de Vivo and her colleagues (2017) used three-dimensional electron microscopy to observe 6,920 synapses in mice and confirmed that, during sleep, synaptic spines decreased at smaller, less stable synapses, whereas stronger synapses were spared.

Brain Structures of Sleep and Waking

We have seen one of the ways sleep can be regarded as an active process: A great deal of activity goes on in the brain during sleep; in fact, the brain's energy use is not significantly lower in sleep than during waking, and may even increase during slow-wave sleep (Dworak, McCarley, Kim, Kalinchuk, & Basheer, 2010). For the second aspect of this active process, we turn to the brain structures involved in turning sleep on and off. There is no single sleep center or waking center; sleep and waking depend on a variety of structures that integrate the timing of the SCN with homeostatic information about physical conditions such as fatigue, brain temperature, and time awake. Sleep is a homeostatic process, in that a period of deprivation is followed by a period of sleep; the balance between the two is brought about by mutually inhibiting wake and sleep centers. The network of structures governing sleep and waking is complex, so you will want to trace its connections carefully in Figures 15.9 and 15.11. Except where noted, the following discussion is taken from the thorough review of the literature by Clifford Safer and his Harvard colleagues (Saper, Fuller, Pedersen, Lu, & Scammell, 2010). We will begin with the structures that produce wakefulness.

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Waking and Arousal Controls

The waking network consists of two major pathways (Figure 15.9); we will start with the *brainstem arousal centers*, which send their activating signals to higher levels of the brain. This ascending pathway is itself

made up of two branches. The first consists of *pedunculopontine and laterodorsal tegmental nuclei (PPT/LDT)*, which fire most rapidly during wakefulness and REM sleep and most slowly during non-REM sleep, apparently driving the cortical activation seen outside non-REM sleep. The second branch includes the *locus coeruleus, raphe nuclei, tuberomammillary nucleus,* and *parabrachial nucleus*. A distinction between the two branches is that although they both send projections to the prefrontal cortex, basal forebrain, and lateral hypothalamus, the PPT/LDT also has connections to the thalamus. Figure 15.10 shows how firing varies with sleep and waking in two of these areas.

Although lesions of these brain stem structures produce a loss of wakefulness, both animals and humans recover normal sleep-wake cycles within a period of weeks or months; this underscores the additional contribution of the *forebrain arousal centers*. The *basal forebrain* produces wakefulness and desynchronization of the EEG; disabling the basal forebrain with an anesthetic results in deep non-REM sleep. Similarly, orexin (hypocretin)-releasing neurons in the *lateral hypothalamus* both sustain wakefulness and suppress REM sleep. Like the brain stem centers, the forebrain arousal centers innervate the prefrontal cortex; the prefrontal cortex in turn sends descending projections back to the basal forebrain, hypothalamus, and brain stem.

FIGURE 15.9 Structures of Waking and Arousal.

Several interacting structures and pathways produce waking, maintain arousal during waking, and increase arousal during REM sleep.

Figures Brought to Life

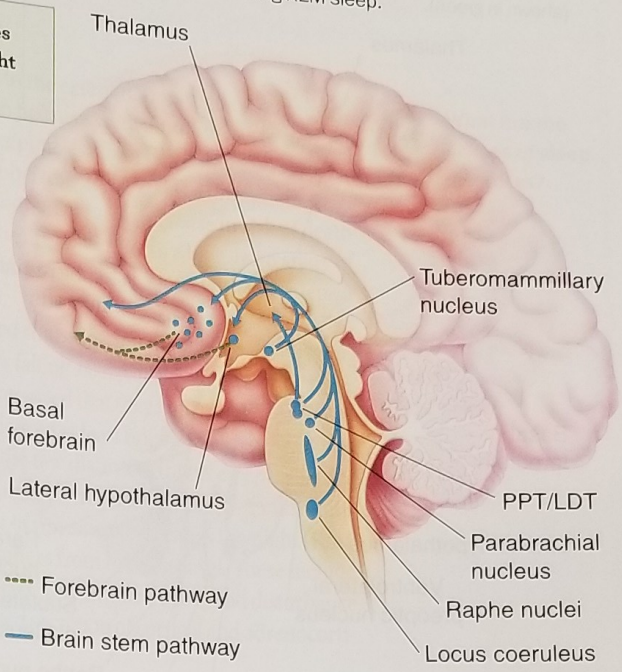
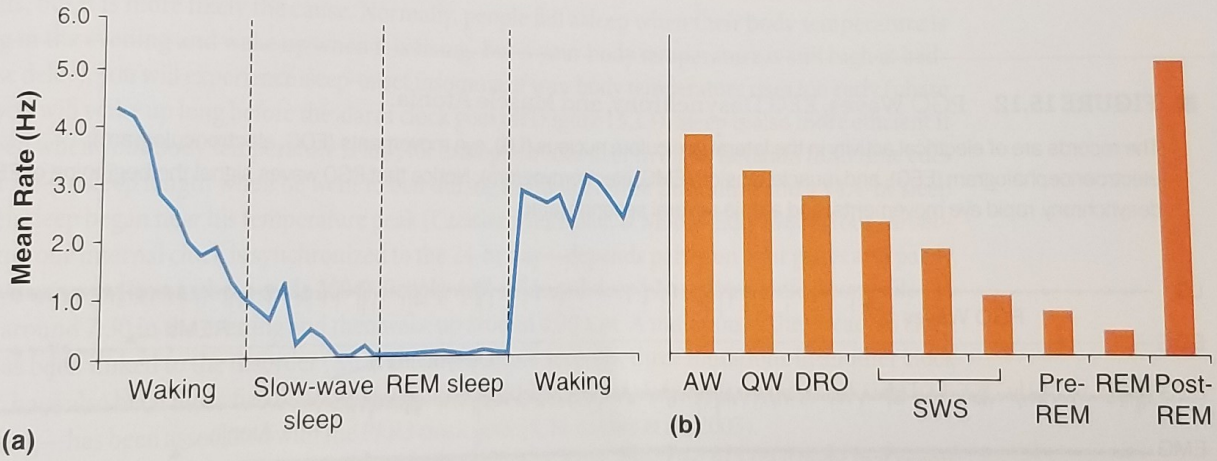


FIGURE 15.10 Firing Rates in Brain Stem Arousal Centers During Waking and Sleep.

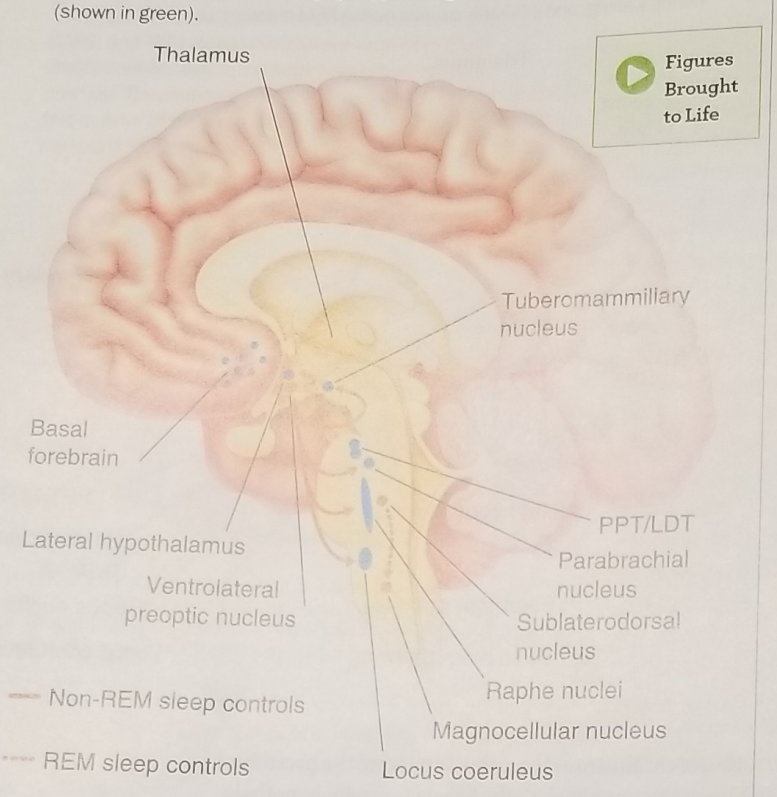
(a) Activity in the locus coeruleus; (b) activity in the raphe nuclei. Note that these nuclei are most active during waking, relatively quiet during non-REM sleep, and (unlike the PPT/LDT) almost silent during REM sleep. AW, alert waking; QW, quiet waking; DRO, drowsy; SWS, slow-wave sleep; pre-REM, 60 seconds before REM; post-REM, first second after REM ends.



Sources: (a) Copyright 1981 by the Society for Neuroscience; (b) From "Activity of Serotonin-Containing Nucleus Centralis Superior (Raphe Medianus) Neurons in Freely Moving Cats," by M. E. Trulson et al., *Experimental Brain Research*, 54, 33–44, fig. 2. © 1984. With kind permission from Springer Science and

■ **FIGURE 15.11** Brain Mechanisms Regulating Sleep.

Sleep is brought about primarily by suppressing activity in arousal structures (shown in green).



Non-REM Sleep Controls

The most important structure in the non-REM sleep network is the *ventrolateral preoptic nucleus (VLPO;* Figure 15.11). Many of the VLPO's neurons fire two to four times faster during non-REM sleep than during wakefulness, and double that again as sleep deepens further. Lesions indicate that the VLPO's core is partly responsible for non-REM sleep and the extended VLPO contributes to REM sleep. The VLPO sends inhibitory signals to the lateral hypothalamus and the second branch of the brain stem arousal network; Saper and his colleagues suggest that the VLPO and the ascending arousal system are the basis for switching between the awake and sleep states. Rats with lesions of the VLPO still sleep about 50% as much as normal rats, which means there must be other sleep centers inhibiting the arousal systems. At least one has been identified; the *parafacial zone* in the medulla contributes to non-REM sleep by sending inhibition to the parabrachial nucleus (Anaclet et al., 2014).

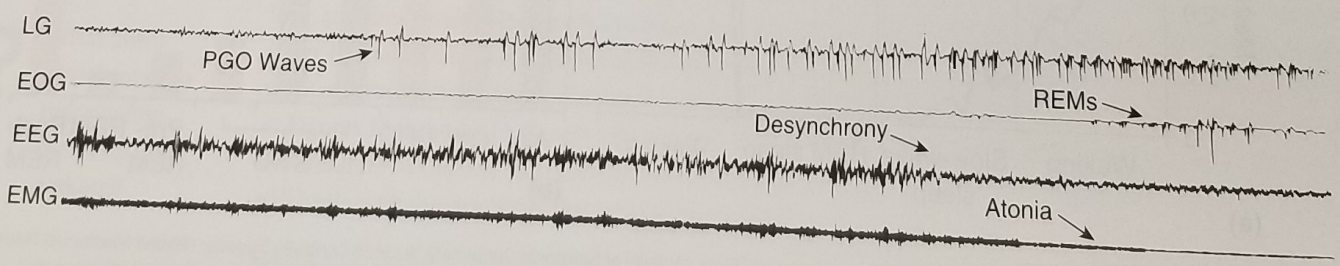
REM Sleep Controls

High-voltage *PGO waves,* so called because of their path of travel from the pons through the lateral geniculate nucleus of the thalamus to the occipital area, begin about 80 seconds before the start of a REM period and apparently are what initiate the EEG desynchrony of REM sleep (Figure 15.12; Mansari, Sakai, & Jouvet, 1989; Steriade, Paré, Bouhassira, Deschênes, & Oakson, 1989). PGO waves are as characteristic of REM sleep as rapid eye movements are. Their arousal of the occipital area may account for the visual imagery of dreaming. PGO waves are synchronized with bursts of firing in PPT/LDT nuclei, while firing in the other arousal centers (locus coeruleus, raphe nucleus, and tuberomammillary nucleus) almost disappears. This suggests that the PPT/LDT might interact with the PGO nuclei to regulate alternation between non-REM and REM sleep.

The most important REM sleep center in the pons is the *sublaterodorsal nucleus (SLD),* which appears to govern switching in and out of REM sleep. Lesioning the SLD reduces REM sleep in rats; in cats it eliminates

■ **FIGURE 15.12** PGO Waves, EEG Desynchrony, and Muscle Atonia.

The records are of electrical activity in the lateral geniculate nucleus (LG), eye movements (EOG, electrooculogram), electroencephalogram (EEG), and muscle tension (EMG, electromyogram). Notice that PGO waves signal the beginning of EEG desynchrony, rapid eye movements, and atonia several seconds later.



Source: Copyright 1989 by the Society for Neuroscience.

output to the magnocellular nucleus in the medulla, which is responsible for the atonia of REM sleep. The cats seemed to be acting out their dreams (assuming that cats dream), and their movements often woke them up (Shouse & Siegel, 1992).

Sleep Disorders

Insomnia

Insomnia is the inability to sleep or to obtain adequate-quality sleep, to the extent that the person feels inadequately rested. Insomnia is important not only as a nuisance but also because sleep duration has important implications for health. In a study of 1.1 million men and women, sleeping less than 6 hr a night was associated with decreased life expectancy (Kripke, Garfinkel, Wingard, Klauber, & Marler, 2002). However, the surprise in the study was that sleeping more than 8.5 hr was associated with as great an increase in risk of death as sleeping less than 4.5 hr. Lack of sleep may also be a factor in the obesity epidemic. In a long-term study of sleep behavior, people who slept less than 8 hr a night had a higher body mass index, along with lower leptin and higher ghrelin levels (Taheri, Lin, Austin, Young, & Mignot, 2004).

The failure to get enough sleep is part of the lifestyle of industrialized countries, but many people who try to get an adequate amount of sleep complain that they have difficulty either falling asleep or staying asleep. In a survey by the National Sleep Foundation (2002), over half the respondents reported that they had trouble sleeping or woke up unrefreshed at least a few nights a week, and a third had experienced at least one symptom of insomnia every night or almost every night in the past year. Insomnia is one of the few disorders that is essentially self-diagnosed, and several studies suggest that the reported frequencies might be misleading. Although insomniacs may overestimate the time required to get to sleep and the amount of time awake through the night (Rosa & Bonnet, 2000), there are several indications that their sleep quality suffers from hyperarousal. These include excess high-frequency EEG during non-REM sleep (Perlis, Smith, Andrews, Orff, & Giles, 2001) and disturbance of the hypothalamic-pituitary-adrenal axis (see Chapter 8), with increased secretion of cortisol and adrenocorticotropic hormone during the night (Vgontzas et al., 2001).

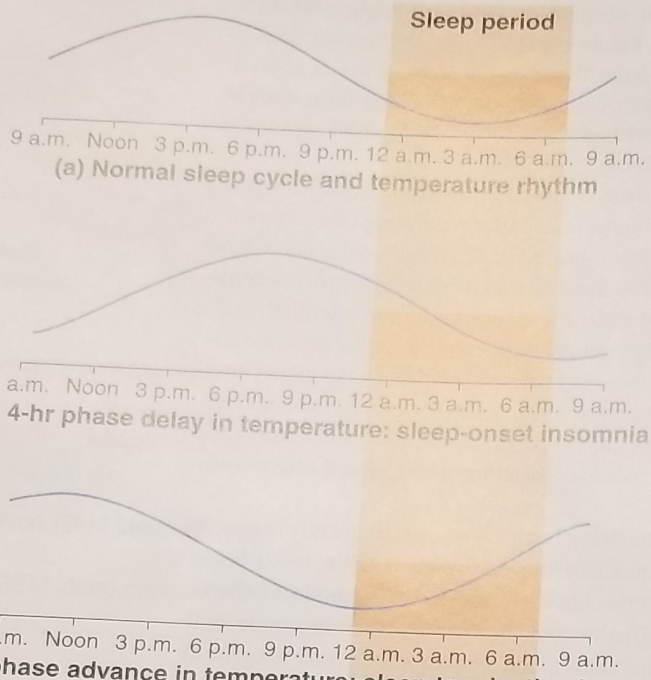
Insomnia can be brought on by a number of factors, such as stress, but it also occurs frequently in people with psychological problems, especially affective disorders (Benca, Obermeyer, Thisted, & Gillin, 1992). Some loss of gray matter in the orbitofrontal cortex and the parietal cortex has been reported in insomniacs (Altena, Vrenken, Van Der Werf, van den Heuvel, & Van Someren, 2010); this could be a cause of their insomnia, or it could reflect the association with psychological disorders. Another study found reduced white matter integrity in several right hemisphere areas, the thalamus, and the corpus callosum; deficiencies in the thalamus and corpus callosum were correlated with the duration of patients' insomnia and with self ratings of depression (S. Li et al., 2016). Another frequent cause is the *treatment* of insomnia; most sleep medications are addictive, so attempts to do without medication or to reduce the dosage produce a rebound insomnia; this can happen after as little as three nights with some benzodiazepines (Kales, Scharf, Kales, & Soldatos, 1979). Insomnia can manifest itself as delayed sleep onset, nighttime waking, or early waking; a disruption of the circadian rhythm is often the culprit (M. Morris, Lack, & Dawson, 1990).

People with sleep difficulties often show a shift in their circadian rhythm; this can be the result of bad sleep habits, but it is more likely the cause. Normally, people fall asleep when their body temperature is decreasing in the evening and wake up when it is rising. But if your body temperature is still high at bedtime (phase delay), you will experience sleep-onset insomnia; if your body temperature rises too early (phase advance) you will wake up long before the alarm clock goes off (Figure 15.13). Sleep is also more efficient if you go to bed when your body temperature is low; for example, a volunteer living in isolation from time cues averaged a 7.8-hr sleep length when he went to bed during his temperature minimum and a 14.4-hr sleep length when sleep began near his temperature peak (Czeisler, Weitzman, & Moore-Ede, 1980). Your *chronotype*—when your internal clock is synchronized to the 24-hr day—depends partly on your genes and partly on your environment (Roenneberg et al., 2004). People with advanced sleep phase disorder feel compelled to go to sleep around 7:30 in the evening and then wake up around 4:30 a.m. A mutation in the circadian clock gene *CKI* has been linked to the disorder (Xu et al., 2005); more recently, three mutations in another clock gene, *PER2*, have also been identified (Chong, Ptáček, & Fu, 2012). Delayed sleep phase syndrome—late bedtime and rising—has been associated with the *PER3* clock gene (S. N. Archer et al., 2003).

It is usually easier for people to delay sleep at night than to rise early, which led to a treatment that seems completely counterintuitive. The patients had a 5- to 15-year history of sleep-onset insomnia so severe that they were not even going to bed until 4:15 a.m., on average. Rather than require them to retire earlier, the researchers had the patients *stay up 3 hr later* each day than the day before. After about a week of this routine—for example,

FIGURE 15.13 Effects of Disrupted Circadian Rhythm on Sleep.

Ordinarily, a person falls asleep while the body temperature is decreasing and awakens as it is rising (a). If body temperature is phase delayed (b), the person has trouble falling asleep; if body temperature is phase advanced (c), the person wakes up early. (Sleep period is the time in bed, whether the person is sleeping or not.)



going to bed at 8 a.m., 11 a.m., 2 p.m., 5 p.m., 8 p.m., and 11 p.m. on successive nights—their average sleep-onset time had shifted from 4:50 a.m. to 12:20 a.m., and their average waking time had shifted from 1:00 p.m. to 7:55 a.m. All five patients were able to give up the sleeping pills they had become dependent on, and improvement was long lasting (Czeisler et al., 1981). Phototherapy is also sometimes used to reset the circadian clock.

Sleepwalking

Some of the sleep disorders are related to specific sleep stages. As we saw earlier, bedwetting, night terrors, and sleepwalking occur during slow-wave sleep. Although sleepwalking is most frequent during childhood, about 3%–8% of adults sleepwalk (A. Dalton, 2005). Kenneth Parks's story in the opening vignette is not unique. The sleepwalking defense was first used in 1846 when Albert Tirrell was acquitted of the murder of his prostitute mistress and the arson of her brothel, and the plea has been successful in a few more recent instances as well (A. Dalton, 2005). Sleepwalking can be triggered by stress, alcohol, and sleep deprivation; Ken's jury was convinced that he was not responsible because he was sleep deprived due to stress over gambling debts and the loss of his job for embezzling; there was a personal and family history of sleepwalking, sleep talking, and bedwetting; and he produced a high level of slow-wave sleep during sleep monitoring (Broughton et al., 1994).

Vulnerability for sleepwalking is at least sometimes genetic. Children of sleepwalkers are 10 times more likely to sleepwalk than children without sleepwalking relatives, and people with a version of a gene that is also implicated in narcolepsy are 3.5 times as likely to sleepwalk as others (Lecendreux et al., 2003). The gene is a member of the human leukocyte antigen (HLA) family, a group of genes that target foreign cells for attack by the immune system, and the authors suspect that cells important in sleep regulation have been attacked by the individual's immune system.

A less known non-REM sleep behavior is sexsomnia, engaging in sexual behavior while asleep. Although usually the worst consequence is embarrassment, the behavior has sometimes led to criminal charges (often leading to acquittal on grounds of nonresponsibility). The prevalence of sexsomnia is uncertain, but 11% of men and 4% of women seeking treatment for sleep disorders reported engaging in sexual behavior while asleep (American Academy of Sleep Medicine, 2010). Patients with sexsomnia didn't differ from the other patients in fatigue, depression, smoking, or caffeine consumption, but they were twice as likely to admit using illicit drugs.

Somewhere in between people who commit mayhem during sleepwalking and those who simply wander about the house are those who suffer from a *sleep-related eating disorder,* the subject of the accompanying Application.

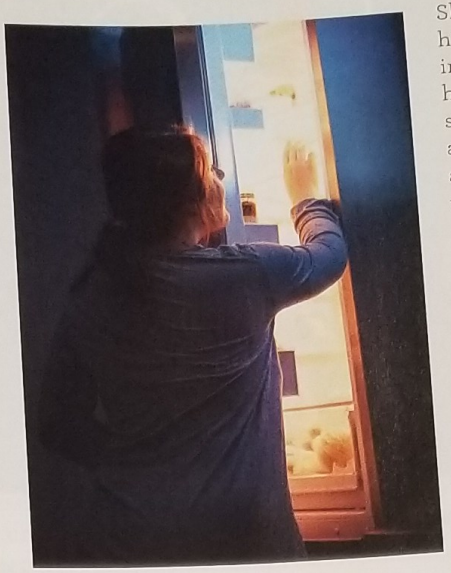
Narcolepsy

When stabilization of the sleep switch fails, the result is *narcolepsy,* a disorder in which individuals fall asleep suddenly during the daytime and go directly into REM sleep. Another symptom of narcolepsy is *cataplexy,* in which the person has a sudden experience of one component of REM sleep, atonia, and falls to the floor paralyzed but fully awake. People with narcolepsy do not sleep more than others; rather, the boundaries are lost between sleep and waking (Nobili et al., 1996). Dogs also develop the disorder, and the study of canine narcolepsy has identified a mutated form of the gene that is responsible for the orexin receptor (Figure 15.14; Lin et al., 1999).

APPLICATION

In the Still of the Night

Shirley Koecheler raids the refrigerator at night (Black & Robertson, 2010). She would like to quit because she's gaining weight, but she isn't aware she's doing it until she wakes up in the morning to a crumb-filled bed and an uncomfortably full stomach. She even had her husband hide the Easter candy, but the next morning she found the wrappers from the chocolate bunnies in the wastebasket. Shirley's 24-year-old daughter Amy is also a sleep eater and has been since she was a toddler; the difference is that she doesn't gain weight. Anna Ryan, like Shirley, started sleep eating in adulthood; she didn't even know about the nighttime kitchen forays that added 60 pounds to her weight in a year and a half until she went to a sleep clinic to find out why she was exhausted every morning. Sleep eaters, usually women, ordinarily pass up healthy snacks for high-calorie junk food; they have also been known to eat soap, glue, frozen pizza, paper, and even egg shells (Epstein, 2010).



Source: iStock/Artfoliophoto.

Sleep-related eating likely has multiple causes. In some instances, the individuals have other sleep disorders, such as sleep apnea, or a history of substance abuse. Some individuals who take pharmacological sleep aids (e.g., Ambien or benzodiazepines such as zolpidem) have reported drug-induced sleep-related eating, although the clinical features of these individuals seem to differ from those who have sleep-related eating as a primary diagnosis (Komada et al., 2016).

A reliable and effective treatment has not yet been found. Amy has responded well to a drug used to prevent seizures; she still sleep eats occasionally, but it's no longer the problem it once was. It took Anna and her doctor months of trial and error to find a combination of drugs that works, but now she sleeps through the night and is losing weight.

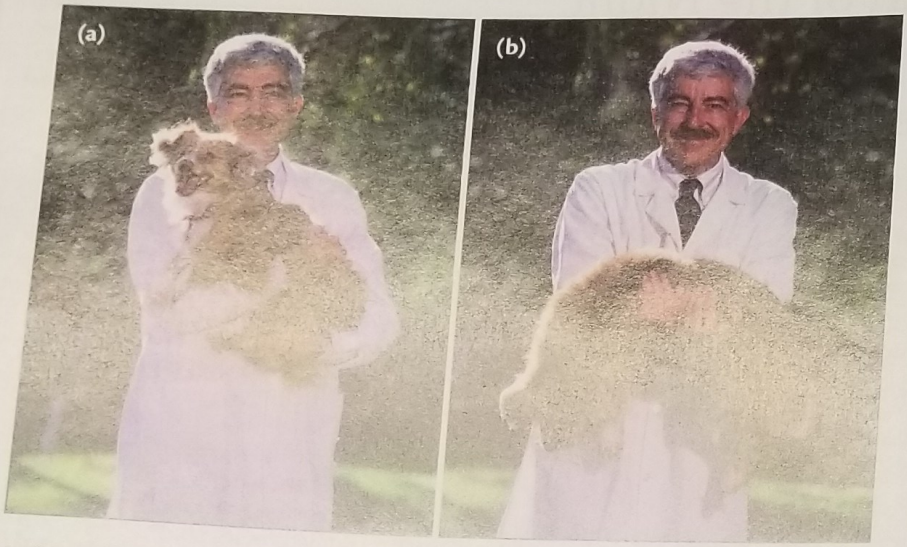
Other researchers studied the effect of orexin as a feeding stimulant in mice by disabling both copies of the gene responsible for producing orexin, but what they observed was more interesting than eating behavior (Chemelli et al., 1999). Occasionally, the mice would suddenly collapse, often while walking around or grooming; the mice were narcoleptic! Most narcoleptic humans (those with cataplexy) turned out to have the same deficiency as the mice; they had low or undetectable levels of orexin, due to a loss of orexin-secreting neurons in the hypothalamus (Higuchi et al., 2002; Kanbayashi et al., 2002). The neurons are destroyed by an autoimmune reaction, which in some cases can be traced to an allele of the *HLA* immune system gene (Hallmayer et al., 2009). Narcolepsy's concordance of 25%–31% in identical twins (Mignot, 1998) leaves plenty of room for environmental influence. Identifying the nongenetic causes has been difficult, but one appears to be the H1N1 influenza (swine flu) virus (De la Herran-Arita et al., 2013; Han et al., 2011). The onset of narcolepsy tends to be seasonal, nearly seven times more frequent shortly after winter; it also increased threefold following the 2009 H1N1 epidemic in China, and lab study has shown that the virus can trigger the immune reaction.

REM Sleep Behavior Disorder

An apparent opposite of cataplexy is *REM sleep behavior disorder;* affected individuals are uncharacteristically physically active during REM sleep, often to the point of injuring themselves or their bed partners. A study of 93 patients, 87% of whom were male, found that 32% had injured themselves and 64% had assaulted their spouses

FIGURE 15.14 Cataplexy in a Dog.

Sleep researcher William Dement holds Tucker before (a) and during (b) an attack of cataplexy. Tucker is paralyzed but awake.



Source: Courtesy of Stanford University Center for Narcolepsy.

(E. J. Olson, Boeve, & Silber, 2000). A 67-year-old man had tied himself to his bed with a rope at night for 6 years because he had a habit of leaping out of bed and landing on furniture or against the wall. One night, he was awakened by his wife's yelling because he was choking her; he was dreaming that he was wrestling a deer to the ground and was trying to break its neck (Schenck, Milner, Hurwitz, Bundlie, & Mahowald, 1989). REM sleep behavior disorder is often associated with a neurological disorder, such as Parkinson's disease or a brain stem tumor (E. J. Olson et al., 2000). Lewy bodies have been found in patients' brains, and two thirds of patients develop Parkinson's about 10 years later (Boeve et al., 2003). These findings have contributed to the hypothesis that Parkinson's disease is preceded by the development of Lewy bodies in the medulla, where inhibition of the magnocellular nucleus ordinarily produces atonia; the Lewy bodies then progress upward through the brain before reaching the substantia nigra years later, when the full-blown disease appears (Braak et al., 2003).

Sleep as a Form of Consciousness

At the beginning of this discussion, we said that sleep is neither entirely conscious nor unconscious. Francis Crick (1994), who shared a Nobel Prize for the discovery of DNA's structure in 1962 before turning to neuroscience and the study of consciousness, believed that we are in a state of diminished consciousness during REM sleep and that we are unconscious during non-REM sleep. Certainly there are some elements of consciousness in the dream state, particularly in people who are *lucid dreamers.* You have probably had the occasional experience of realizing during a bad dream that it is not actually real and will end soon. That kind of experience is common for lucid dreamers—they are often aware during a dream that they are dreaming. People can be trained to become aware of their dreaming and to signal to the researcher when they are dreaming by pressing a handheld switch (Salamy, 1970). They can even learn to *control* the content of their dreams; they may decide before sleeping what they will dream about, or they may interact with characters in their dream (Gackenbach & Bosveld, 1989). This ability tells us that the sleeping person is not necessarily as detached from reality as we have thought. This point is further illustrated by sleepwalkers, who have driven cars; wandered the streets; brandished weapons (Schenck et al., 1989); and strangled, stabbed, and beaten people to death, all presumably during non-REM sleep.

When you are asleep, are you unconscious?

So it is not clear where or whether the transition from consciousness to nonconsciousness occurs during sleep. The idea of a dividing line is blurred even further by reports that surgical patients can sometimes remember the surgical staff's conversations while they were anesthetized, and they show some memory later for verbal material presented at the time of surgery (Andrade, 1995; Bonebakker et al., 1996). Whether you draw the line of consciousness between waking and sleeping or between REM and non-REM sleep or between sleep and coma depends more on your definition of consciousness than on any clear-cut distinctions between these conditions. Perhaps it is better to think of sleep as a different state of consciousness along a continuum of consciousness. We can then concentrate on what the differences between waking and sleeping tell us about consciousness rather than worrying about classifications.

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lack of wonders,
ack of wonder.

–J. B. S. Haldane

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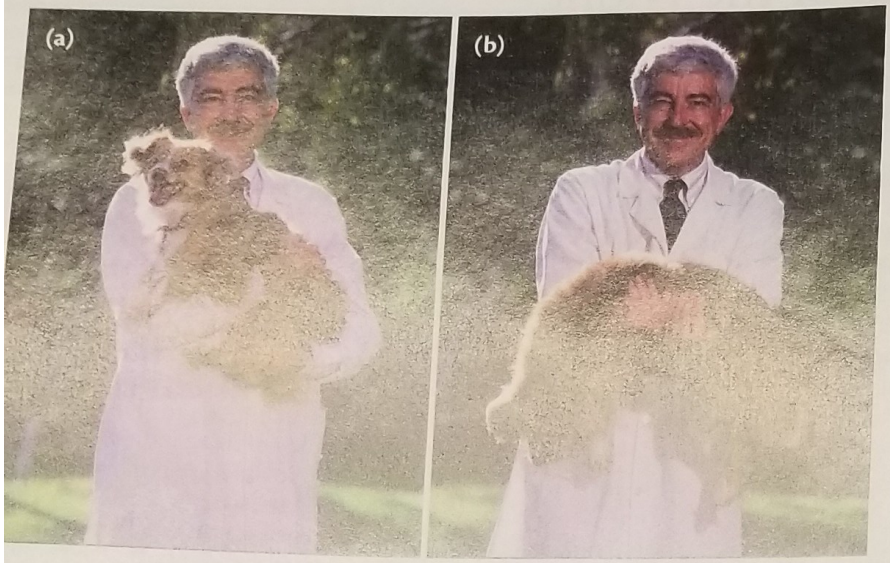

Courtesy of Stanford University Center for Narcolepsy.

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So it is not clear where or whether the transition from consciousness to nonconsciousness occurs during sleep. The idea of a dividing line is blurred even further by reports that surgical patients can sometimes remember the surgical staff's conversations while they were anesthetized, and they show some memory later for verbal material presented at the time of surgery (Andrade, 1995; Bonebakker et al., 1996). Whether you draw the line of consciousness between waking and sleeping or between REM and non-REM sleep or between sleep and coma depends more on your definition of consciousness than on any clear-cut distinctions between these conditions. Perhaps it is better to think of sleep as a different state of consciousness along a continuum of consciousness. We can then concentrate on what the differences between waking and sleeping tell us about consciousness rather than worrying about classifications.