Wakefulness and Sleep

CHAPTER OUTLINE

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- Setting and Resetting the Biological Clock
- Mechanisms of the Biological Clock
- In Closing: Sleep–Wake Cycles

MODULE 9.2 Stages of Sleep and Brain Mechanisms
- Sleep and Other Interruptions of Consciousness
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- Brain Function in REM Sleep
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MODULE 9.3 Why Sleep? Why REM? Why Dreams?
- Functions of Sleep
- Functions of REM Sleep
- Biological Perspectives on Dreaming
- In Closing: Our Limited Self-Understanding
- Interactive Exploration and Study

MAIN IDEAS

1. The brain generates a wake–sleep cycle of approximately 24 hours even in an unchanging environment.
2. Sleep progresses through various stages that differ in brain activity, heart rate, and other aspects. A stage known as paradoxical or REM sleep is light in some ways and deep in others.
3. Areas in the brainstem and forebrain control arousal and sleep. Localized brain damage can produce prolonged sleep or wakefulness.
4. Because sleep depends on inhibition of brain activity, sometimes one brain area is awake while another is asleep, as in the case of sleepwalking.
5. Sleep can be impaired in many ways.
6. We need sleep and REM sleep, although much about their functions remains uncertain.

Anyone deprived of sleep suffers. But if life evolved on another planet with different conditions, could animals evolve life without a need for sleep? Imagine a planet that doesn’t rotate on its axis. Some animals evolve adaptations to live in the light area, others in the dark area, and still others in the twilight zone separating light from dark. There would be no need for any animal to alternate active periods with inactive periods on any fixed schedule and perhaps no need for prolonged inactive periods. If you were the astronaut who discovered these sleepless animals, you might be surprised.

Now imagine that astronauts from that planet set out on their first voyage to Earth. Imagine their surprise to discover animals like us with long inactive periods resembling death. To someone who hadn’t seen sleep before, it would seem mysterious indeed. For the purposes of this chapter, let’s adopt their perspective and ask why animals as active as we are spend one third of our lives doing so little.

OPPOSITE: Rock hyraxes at a national park in Kenya.
You are probably not amazed to learn that your body spontaneously generates its own rhythm of wakefulness and sleep. Psychologists of an earlier era strongly resisted that idea. When behaviorism dominated experimental psychology during the mid-1900s, many psychologists believed that every behavior could be traced to external stimuli. For example, alternation between wakefulness and sleep must depend on something in the outside world, such as changes in light or temperature. Research as early as that of Curt Richter (1922) implied that the body generates its own cycles of activity and inactivity, but it took a huge amount of research to convince the skeptics. The idea of self-generated rhythms was a major step toward viewing animals as active producers of behaviors.

## Endogenous Cycles

An animal that produced its behavior entirely in response to current stimuli would be at a serious disadvantage. Animals often need to anticipate changes in the environment. For example, migratory birds start flying toward their winter homes before their summer territory becomes too cold. A bird that waited for the first frost would be in trouble. Similarly, squirrels begin storing nuts and putting on extra layers of fat in preparation for winter long before food becomes scarce.

Animals' readiness for a change in seasons comes partly from internal mechanisms. Changes in the light–dark pattern of the day tell a migratory bird when to fly south for the winter, but what tells it when to fly back north? In the tropics, the temperature and amount of daylight are nearly the same throughout the year. Nevertheless, a migratory bird flies north at the right time. Even if it is released, it flies north (Gwinner, 1986). Evidently, the bird generates a rhythm that prepares it for seasonal changes. We refer to that rhythm as an endogenous circannual rhythm. (Endogenous means ‘generated from within.’ Circannual comes from the Latin words circum, for “about,” and annum, for “year.”)

Animals also produce endogenous circadian rhythms that last about a day. (Circadian comes from circum, for “about,” and dies, for “day.”) If you go without sleep all night—as most college students do, sooner or later—you feel sleepier and sleepier as the night goes on, but as morning arrives, you feel more alert, not less. The light from the sun helps you feel less sleepy, but also your urge to sleep depends partly on the time of day, not just how long you have been awake (Babkoff, Caspy, Mikulincer, & Sing, 1991).

Figure 9.1 represents the activity of a flying squirrel kept in total darkness for 25 days. Each horizontal line represents one 24-hour day. A thickening in the line represents a period of activity. Even in this unchanging environment, the animal generates a consistent rhythm of activity and sleep. Depending on the individual and the details of the procedure, the self-generated cycle may be slightly shorter than 24 hours, as in Figure 9.1, or slightly longer (Carpenter & Grossberg, 1984).

Humans also generate wake–sleep rhythms, and we find it difficult to sleep on anything far from a 24-hour schedule. We can modify it a little. If we ever send astronauts to Mars, they will have to adjust to the Martian day, which lasts about 24 hours and 39 minutes of Earth time. Researchers have found that people can adjust to that schedule without much difficulty (Scheer, Wright, Kronauer, & Czeisler, 2007). Circadian rhythms may be the least of our problems if we travel to Mars. However, more severe departures from a 24-hour schedule pose difficulties. Naval personnel on U.S. nuclear-powered submarines are cut off from sunlight for months at a time, living under faint artificial light. In many cases, they live on a schedule of 6 hours of work alternating with 12 hours of rest. Even though they sleep (or try to sleep) on this 18-hour schedule, their bodies generate rhythms of alertness and body chemistry that average about 24.3 to 24.4 hours (Kelly et al., 1999).

Circadian rhythms affect much more than just waking and sleeping. We have circadian rhythms in our eating and drinking, urination, secretion of hormones, sensitivity to drugs, and other variables. For example, although we ordinarily think of human body temperature as 37°C, normal temperature fluctuates over the course of a day from
a low near 36.7° C during the night to almost 37.2° C in late afternoon (Figure 9.2). We also have circadian rhythms in mood. In one study, young adults recorded their mood every two hours throughout the day. Although the results varied among individuals, most showed increases in positive mood (happiness) from waking until late afternoon, and then a slight decline from then to bedtime. In a follow-up study, the same investigators kept young adults awake for 30 consecutive hours, starting at either 10 a.m. or 5 p.m., in a laboratory setting with constant levels of light and temperature. Regardless of whether people started this procedure at 10 a.m. or 5 p.m., most reported their most pleasant mood around 5 p.m. and their least pleasant mood at around 5 a.m. (Murray et al., 2009). These results suggest a biologically driven circadian rhythm in our emotional well-being (Figure 9.3).

Circadian rhythms differ among individuals. Some people ("morning people," or "larks") awaken early, quickly become productive, and become less alert as the day progresses. Others ("evening people," or "owls") warm up more slowly, both literally and figuratively, reaching their peak in the late afternoon or evening. They tolerate staying up all night better than morning people do (Taillard, Philip, Coste, Sagaspe, & Bioulac, 2003).

Not everyone falls neatly into one extreme or the other, of course. A convenient way to compare people is to ask, "On holidays and vacations when you have no obligations, what time is the middle of your sleep?" For example, if you sleep from 1 a.m. until 9 a.m. on those days, your middle is 5 a.m. As Figure 9.4 shows, people differ by age. As a child, you almost certainly went to bed early and woke up early. As you entered adolescence, you started staying up later and waking up later, when you had the opportunity. The mean preferred time of going to sleep gets later and later until about age 20 and then starts a gradual reversal (Roenneberg et al., 2004).

Do people older than 20 learn to go to bed earlier because they have jobs that require them to get up early? Maybe, but two facts point instead to a biological explanation. First, in Figure 9.4, note how the shift continues gradually over decades. If people were simply adjusting to their jobs, we might expect a sudden shift in the early 20s and then steadiness until retirement. Second, a similar trend occurs in rats: Older rats reach their best performance shortly after awakening, whereas younger rats tend to improve performance as the day progresses (Winocur & Hasher, 1999, 2004).

STOP & CHECK
1. What evidence indicates that humans have an internal biological clock?

ANSWER
1. People who have lived in an environment with a light-dark schedule much different from a 24-hour day fall asleep on about a 24-hour basis and sleep on about a 24-hour basis. To follow this schedule and instead become well-rested, they need to adjust their sleep schedule. This is consistent with the idea that humans have an internal biological clock that regulates their sleep-wake cycle.
During 30 hours in an unchanging laboratory environment, the average young adult reported the most pleasant mood in the late afternoon or early evening, and the least pleasant mood around 5 to 7 a.m. The pattern was similar for those who started the procedure in the morning (above) or in the evening (below). (From Murray, G., Nicholas, C. L., Kleiman, J., Dwyer, R., Carrington, M. J., Allen, N. B., et al. (2009). Nature’s clocks and human mood: The circadian system modulates reward motivation. Emotion, 9, 705–716.)

**FIGURE 9.3 Reported positive mood over time**

**FIGURE 9.4 Age differences in circadian rhythms**

People reported the time of the middle of their sleep, such as 3 a.m. or 5 a.m., on days when they had no obligations. (Reprinted from T. Roenneberg et al., “A Marker for the End of Adolescence,” Current Biology, 14, R1038–R1039.) Figure 1, copyright 2004, with permission from Elsevier.)
9.1 Rhythms of Waking and Sleeping

Setting and Resetting the Biological Clock

Our circadian rhythms generate a period close to 24 hours, but they are not perfect. We readjust our internal workings daily to stay in phase with the world. Sometimes, we misadjust them. On weekends, when most of us are freer to set our own schedules, we expose ourselves to lights, noises, and activity at night and then awaken late the next morning. By Monday morning, when the clock indicates 7 a.m., the biological clock within us may say 5 a.m., and we stagger off to work or school without much pep (Moore-Ede, Czeisler, & Richardson, 1983).

Although circadian rhythms persist without light, light is critical for resetting them. Without something to reset your circadian rhythm, it would gradually drift away from the correct time. The stimulus that resets the circadian rhythm is referred to by the German term zeitgeber (TSITE-gay-ber), meaning “time-giver.” Light is the dominant zeitgeber for land animals (Rusak & Zucker, 1979). (The tides are important for many marine animals.) In addition to light, other zeitgebers include exercise (Eastman, Hoese, Youngstedt, & Liu, 1995), arousal of any kind (Gritton, Sutton, Martinez, Sarter, & Lee, 2009), meals, and the temperature of the environment (Refinetti, 2000). Social stimuli—that is, the effects of other people—are weak zeitgebers, unless they induce exercise or other vigorous activity (Mistlberger & Skene, 2004). These additional zeitgebers merely supplement or alter the effects of light. On their own, their effects are weak. For example, people who are working in Antarctica during the constant darkness of an Antarctic winter try to maintain a 24-hour rhythm, but they drift away from it. Different people generate different rhythms, until they find it more and more difficult to work together (Kennaway & Van Dorp, 1991). Astronauts in Earth orbit face a special problem: As they orbit the Earth, a 45-minute period of daylight alternates with 45 minutes of darkness. If they retreat from the flight deck to elsewhere in the spacecraft, they have constant dim light. As a result, they are never fully alert during their wakeful periods and they sleep poorly during their rest periods (Dijk et al., 2001). On long trips, many of them experience depression, irritability, and impaired performance (Mallis & DeRoshia, 2004). These additional zeitgebers merely supplement or alter the effects of light. On their own, their effects are weak. For example, people who are working in Antarctica during the constant darkness of an Antarctic winter try to maintain a 24-hour rhythm, but they drift away from it. Different people generate different rhythms, until they find it more and more difficult to work together (Kennaway & Van Dorp, 1991). Astronauts in Earth orbit face a special problem: As they orbit the Earth, a 45-minute period of daylight alternates with 45 minutes of darkness. If they retreat from the flight deck to elsewhere in the spacecraft, they have constant dim light. As a result, they are never fully alert during their wakeful periods and they sleep poorly during their rest periods (Dijk et al., 2001). On long trips, many of them experience depression, irritability, and impaired performance (Mallis & DeRoshia, 2004).

Even when we try to set our wake-sleep cycles by the clock, sunlight has its influence. Consider what happens when we shift to daylight savings time in spring. You set your clock to an hour later, and when it shows your usual bedtime, you dutifully go to bed, even though it seems an hour too early. The next morning, when the clock says it is 7 a.m. and time to get ready for work, your brain registers 6 a.m. Most people remain inefficient and ill-rested for days after the shift to daylight savings time. The adjustment is especially difficult for people who were already sleep-deprived, including most college students (Lahti et al., 2006; Monk & Aplin, 1980).

Particularly impressive evidence for the importance of sunlight comes from a study in Germany. The sun time at the eastern end of Germany differs by about half an hour from that at the western edge, even though everyone is on the same clock time. Researchers asked adults for their preferred times of awakening and going to sleep and determined for each person the midpoint of those values. (For example, if on weekends and holidays you prefer to go to bed at 12:30 a.m. and awaken at 8:30 a.m., your sleep midpoint is 4:30 a.m.) Figure 9.5 shows the results. People at the eastern edge have a sleep midpoint about 30 minutes earlier than those at the west, corresponding to the fact that the sun rises earlier at the eastern edge (Roenneberg, Kumar, & Merrow, 2007). The data shown here apply to people in towns and cities with populations under 300,000. People in larger cities show a less consistent trend, presumably because they spend more time indoors with less exposure to the sun.

What about blind people, who need to set their circadian rhythms by zeitgebers other than light? The results vary. Some do set their circadian rhythms by noise, temperature, meals, and activity. However, others who are not sufficiently sensitive to these secondary zeitgebers produce circadian rhythms that are a little longer than 24 hours. When their cycles are in phase with the clock, all is well, but when they drift out of phase, they experience insomnia at night and sleepiness during the day (Sack & Lewy, 2001).

FIGURE 9.5 Sun time competes with social time
On days when people have no obligation to awaken at a particular time, they awaken about half an hour earlier at the eastern edge of Germany than at the western edge. Points along the y axis represent the midpoint between the preferred bedtime and the preferred waking time. Data are for people living in towns and cities with populations under 300,000. (From Roenneberg, T., et al. (2007). “The human circadian clock entrains to sun time.” Current Biology, 17, R44–R45. Reprinted by permission of the Copyright Clearance Center.)
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STOP & CHECK

2. Why do people at the eastern edge of Germany awaken earlier than those at the western edge on their weekends and holidays?

ANSWER

Jet Lag

A disruption of circadian rhythms due to crossing time zones is known as jet lag. Travelers complain of sleepiness during the day, sleeplness at night, depression, and impaired concentration. All these problems stem from the mismatch between internal circadian clock and external time (Haimov & Arendt, 1999). Most people find it easier to adjust to crossing time zones going west than east. Going west, we stay awake later at night and then awaken late the next morning, already partly adjusted to the new schedule. We phase-delay our circadian rhythms. Going east, we phase-advance to sleep earlier and awaken earlier (Figure 9.6). Most people find it difficult to go to sleep before their body’s usual time and difficult to wake up early the next day.

Adjusting to jet lag is often stressful. Stress elevates blood levels of the adrenal hormone cortisol, and many studies have shown that prolonged elevations of cortisol damage neurons in the hippocampus, a brain area important for memory. One study examined flight attendants who had spent the previous 5 years making flights across 7 or more time zones—such as Chicago to Italy—with mostly short breaks (fewer than 6 days) between trips. Most of these flight attendants had smaller than average volumes of the hippocampus and surrounding structures, and they showed some memory impairments (Cho, 2001). These results suggest a danger from repeated adjustments of the circadian rhythm, although the problem here could be air travel itself. (A good control group would have been flight attendants who flew long north–south routes.)

Shift Work

People who sleep irregularly—such as pilots, medical interns, and shift workers in factories—find that their duration of sleep depends on when they go to sleep. When they have to sleep in the morning or early afternoon, they sleep only briefly, even if they have been awake for many hours (Frese & Harwich, 1984; Winfree, 1983).

People who work on a night shift, such as midnight to 8 a.m., sleep during the day. At least they try to. Even after months or years on such a schedule, many workers adjust incompletely. They continue to feel groggy on the job, they sleep poorly during the day, and their body temperature continues to peak when they are sleeping in the day instead of while they are working at night. In general, night–shift workers have more accidents than day–shift workers.

Working at night does not reliably change the circadian rhythm because most buildings use artificial lighting in the range of 150–180 lux, which is only moderately effective in resetting the rhythm (Boivin, Duffy, Kronauer, & Czeisler, 1996). People adjust best to night work if they sleep in a very dark room during the day and work under very bright lights at night, comparable to the noonday sun (Czeisler et al., 1990).

Mechanisms of the Biological Clock

How does the body generate a circadian rhythm? Curt Richter (1967) introduced the concept that the brain generates its own rhythms—a biological clock—and he reported that the biological clock is insensitive to most forms of interference.
Blind or deaf animals generate circadian rhythms, although they slowly drift out of phase with the external world. The circadian rhythm remains surprisingly steady despite food or water deprivation, X-rays, tranquilizers, alcohol, anesthesia, lack of oxygen, most kinds of brain damage, or the removal of endocrine organs. Even an hour or more of induced hibernation often fails to reset the biological clock (Gibbs, 1983; Richter, 1975). Evidently, the biological clock is a hardy, robust mechanism.

The Suprachiasmatic Nucleus (SCN)

The biological clock depends on part of the hypothalamus, called the suprachiasmatic (soo-pruh-ki-az-MAT-ik) nucleus, or SCN. It gets its name from its location just above (“supra”) the optic chiasm (Figure 9.7). The SCN provides the main control of the circadian rhythms for sleep and body temperature (Refrinetti & Menaker, 1992), although several other brain areas generate local rhythms (Granados-Fuentes, Tseng, & Herzog, 2006). After damage to the SCN, the body’s rhythms become erratic.

The SCN generates circadian rhythms itself in a genetically controlled, unlearned manner. If SCN neurons are disconnected from the rest of the brain or removed from the body and maintained in tissue culture, they continue to produce a circadian rhythm of action potentials (Earnest, Liang, Ratcliff, & Cassone, 1999; Inouye & Kawamura, 1979). Even a single isolated SCN cell can maintain a circadian rhythm, although interactions among cells sharpen the accuracy of the rhythm (Long, Jutras, Connors, & Burwell, 2005; Yamaguchi et al., 2003).

A mutation in one gene causes hamsters’ SCN to produce a 20-hour instead of 24-hour rhythm (Ralph & Menaker, 1988). Researchers surgically removed the SCN from adult hamsters and transplanted SCN tissue from hamster fetuses into the adults. When they transplanted SCN tissue from fetuses with a 20-hour rhythm, the recipients produced a 20-hour rhythm. When they transplanted tissue from fetuses with a 24-hour rhythm, the recipients produced a 24-hour rhythm (Ralph, Foster, Davis, & Menaker, 1990). That is, the rhythm followed the pace of the donors, not the recipients. Again, the results show that the rhythms come from the SCN itself.

How Light Resets the SCN

The SCN is located just above the optic chiasm. Figure 9.7 shows the position in the human brain. The relationship is similar in other mammals. A small branch of the optic nerve, known as the retinohypothalamic path, extends directly from the retina to the SCN. Axons of that path alter the SCN’s settings.

Most of the input to that path, however, does not come from normal retinal receptors. Mice with genetic defects that destroy nearly all their rods and cones nevertheless reset their biological clocks in synchrony with the light (Freedman et al., 1999; Lucas, Freedman, Muñoz, García-Fernández, & Foster, 1999). Also, consider blind mole rats (Figure 9.8). Their eyes are covered with folds of skin and fur. They have neither eye muscles nor a lens with which to focus an image. They have fewer than 900 optic nerve axons compared with 100,000 in hamsters. Even a bright flash of light evokes no startle response and no measurable change in brain activity. Nevertheless, light resets their circadian rhythms (de Jong, Hendriks, Sanyal, & Nevo, 1990).

The surprising explanation is that the retinohypothalamic path to the SCN comes from a special population of retinal ganglion cells that have their own photopigment, called melanopsin, unlike the ones found in rods and cones (Hannibal, Hindersson, Knudsen, Georg, & Fahrenkrug, 2001; Lucas, Douglas, & Foster, 2001). These special ganglion cells receive some input from rods and cones (Gooley et al., 2010; Güler et al., 2008), but even if they do not receive that input, they respond directly to light (Berson, Dunn, & Takao, 2002). These special ganglion cells are located mainly near the nose, not evenly throughout the retina (Visser, Beersma, & Daan, 1999). (That is, they see toward the periphery.) They respond to light slowly and turn off slowly when the light ceases (Berson et al., 2002). Therefore, they respond to the overall average amount of light, not to instantaneous changes in light. The average intensity over a period of minutes or hours is, of course, exactly the information the SCN needs to gauge the time of day.

Note a couple of consequences: First, many people who are blind because of damage to the rods and cones nevertheless have enough input to the melanopsin-containing ganglion cells to entrain their waking and sleeping cycle to the local pattern of sunlight. Second, it was formerly puzzling that bright

STOP & CHECK

3. What evidence strongly indicates that the SCN produces the circadian rhythm itself?

ANSWER

The SCN contains neurons that produce a circadian rhythm followed by the rest of the body. Also, when hamsters receive SCN tissue from fetuses with a 20-hour rhythm, the recipients produce a 20-hour rhythm. Even a bright flash of light evokes no startle response and no measurable change in brain activity. Nevertheless, light resets their circadian rhythms (de Jong, Hendriks, Sanyal, & Nevo, 1990).
light aggravates migraine headaches even for many blind people. The explanation is that the melanopsin-containing ganglion cells send input to the posterior thalamus, which is part of the pathway producing pain in migraines (Noseda, et al., 2010). Someone with no input to the visual cortex, and therefore no conscious vision, can nevertheless have light-sensitive excitation in the thalamus.

STOP & CHECK

4. How does light reset the biological clock?

ANSWER: They do not receive input from rods or cones. Their cells that respond to light by themselves, even from axons comprising that part of the retina specialized for light, convey information about light to the SCN. This part of the optic nerve, the retinohypothalamic tract, conveys this information to the SCN.

4. A blind mole rat

Although blind mole rats are blind in other regards, they reset their circadian rhythms in response to light.
The Biochemistry of the Circadian Rhythm

The suprachiasmatic nucleus produces the circadian rhythm, but how? Research on production of the circadian rhythm began with insects. Studies on the fruit fly Drosophila found several genes responsible for a circadian rhythm (X. Liu et al., 1992; Sehgal, Ousley, Yang, Chen, & Schotland, 1999). Two of these genes, known as period (abbreviated per) and timeless (tim), produce the proteins PER and TIM. The concentration of these two proteins, which promote sleep and inactivity, oscillates over a day, based on feedback interactions among several sets of neurons. Early in the morning, the messenger RNA levels responsible for producing PER and TIM start at low concentrations. As they increase during the day, they increase synthesis of the proteins, but the process takes time, and so the protein concentrations lag hours behind, as shown in Figure 9.9. As the PER and TIM protein concentrations increase, they feed back to inhibit the genes that produce the messenger RNA molecules. Thus, during the night, the PER and TIM concentrations are high, but the messenger RNA concentrations are declining (Nitabach & Taghert, 2008). By the next morning, PER and TIM protein levels are low, the flies awaken, and the cycle is ready to start again. Because the feedback cycle takes about 24 hours, the flies generate a circadian rhythm even in an unchanging environment. However, in addition to the automatic feedback, light activates a chemical that breaks down the TIM protein, thereby increasing wakefulness and synchronizing the internal clock to the external world (Ashmore & Sehgal, 2003).

Why do we care about flies? The reason is that analyzing the mechanism in flies told researchers what to look for in humans and other mammals. Mammals have three versions of the PER protein and several proteins closely related to TIM and the others found in flies (Reick, Garcia, Dudley, & McKnight, 2001; Zheng et al., 1999). Mutations in the genes producing PER proteins lead to alterations of sleep schedules. People with a particular PER mutation have been found to have a circadian rhythm shorter than 24 hours, as if they were moving about a time zone west every day (C. R. Jones et al., 1999). They consistently get sleepy early in the evening and awaken early in the morning (Toh et al., 2001; Xu et al., 2005). Most people look forward to days when they can stay up late. People with the altered gene look forward to times when they can go to bed early. Most people with this sleep abnormality suffer from depression (Xu et al., 2005). As we see again in Chapter 15, sleep impairments and depression are closely linked. Another PER mutation has been identified that is more common but less intrusive. People with this gene are normal in most regards except that their alertness deteriorates substantially if they are deprived of a good night’s sleep (Dijk & Archer, 2010).

STOP & CHECK

5. How do the proteins TIM and PER relate to sleepiness in Drosophila?

ANSWER

The proteins TIM and PER remain low during most of the day and begin to increase toward evening. They reach high levels at night, promoting sleep. They also feed back to inhibit the genes that produce them, so their level declines toward morning. Thus, the proteins TIM and PER remain low during most of the day and begin to increase toward evening. They reach high levels at night, promoting sleep. They also feed back to inhibit the genes that produce them, so their level declines toward morning.

Melatonin

The SCN regulates waking and sleeping by controlling activity levels in other brain areas, including the pineal gland (PIN-ce-al; Figure 9.7), an endocrine gland located just posterior to the thalamus (Aston-Jones, Chen, Zhu, & Oshinsky, 2001; von Gall et al., 2002). The pineal gland releases the hormone melatonin, which influences both circadian and circannual rhythms (Butler et al., 2010). The pineal gland secretes melatonin mostly at night, making us sleepy at that time. When people shift to a new time zone and start following a
new schedule, they continue to feel sleepy at their old times until the melatonin rhythm shifts (Dijk & Cajochen, 1997). People who have pineal gland tumors sometimes stay awake for days at a time (Haimov & Lavie, 1996). Melatonin secretion starts to increase about 2 or 3 hours before bedtime. Taking a melatonin pill in the evening has little effect on sleepiness because the pineal gland produces melatonin at that time anyway. However, people who take melatonin at other times become sleepy within 2 hours (Haimov & Lavie, 1996). Melatonin pills are sometimes helpful when people travel across time zones or for other reasons need to sleep at an unaccustomed time.

Melatonin also feeds back to reset the biological clock through its effects on receptors in the SCN (Gillette & McArthur, 1996). A moderate dose of melatonin (0.5 mg) in the afternoon phase-advances the clock. That is, it makes the person get sleepy earlier in the evening and wake up earlier the next morning. A single dose of melatonin in the morning has little effect (Wirz-Justice, Werth, Renz, Müller, & Kräuchi, 2002), although repeated morning doses can phase-delay the clock, causing the person to get sleepy later than usual at night and awaken later the next morning.

### Module 9.1 In Closing

Sleep—Wake Cycles

Unlike an electric appliance that stays on until someone turns it off, the brain periodically turns itself on and off. Sleepiness is not a voluntary or optional act. We have biological mechanisms that prepare us to wake at certain times and sleep at other times, even if we would prefer different schedules.

### Summary

1. Animals, including humans, have circadian rhythms—internally generated rhythms of activity and sleep lasting about 24 hours, even in an unchanging environment. It is difficult to adjust to a sleep schedule much different from 24 hours. 266
2. Some people are most alert early in the morning, and others become more alert later in the day. On average, people show their greatest preference for staying awake late and sleeping late the next morning when they are about 20 years old. 267
3. Although the biological clock continues to operate in constant light or constant darkness, the onset of light resets the clock. Even when people set their waking and sleeping times by the clock, the timing of sunrise strongly influences their circadian rhythm. 268
4. It is easier for most people to follow a cycle longer than 24 hours (as when traveling west) than to follow a cycle shorter than 24 hours (as when traveling east). 270
5. If people wish to work at night and sleep during the day, the best way to shift the circadian rhythm is to have bright lights at night and darkness during the day. 270
6. The suprachiasmatic nucleus (SCN), a part of the hypothalamus, generates the body’s circadian rhythms for sleep and temperature. 271
7. Light resets the biological clock by a branch of the optic nerve that extends to the SCN. Those axons originate from a special population of ganglion cells that respond directly to light in addition to receiving some input from rods and cones. 272
8. The genes controlling the circadian rhythm are almost the same in mammals as in insects. Circadian rhythms result from a feedback cycle based on genes that produce the proteins PER and TIM, and the ability of those proteins to inhibit the genes that produce them. 273
9. The SCN controls the body’s rhythm partly by directing the release of melatonin by the pineal gland. The hormone melatonin increases sleepiness; if given at certain times of the day, it can also reset the circadian rhythm. 274

### Key Terms

Terms are defined in the module on the page number indicated. They’re also presented in alphabetical order with definitions in the book’s Subject Index/Glossary, which begins on page 561. Interactive flashcards and crossword puzzles are among the online resources available to help you learn these terms and the concepts they represent.

- endogenous circadian rhythms 266
- endogenous circannual rhythm 266
- jet lag 270
- melatonin 273
- pineal gland 273
- suprachiasmatic nucleus (SCN) 271
- zeitgeber 269
THOUGHT QUESTIONS

1. Why would evolution have enabled blind mole rats to synchronize their SCN activity to light, even though they cannot see well enough to make any use of the light?

2. If you travel across several time zones to the east and want to use melatonin to help reset your circadian rhythm, at what time of day should you take it? What if you travel west?
Stages of Sleep and Brain Mechanisms

Suppose you buy a new radio. After you play it for 4 hours, it suddenly stops. You wonder whether the batteries are dead or whether the radio needs repair. Later, you discover that this radio always stops after playing for 4 hours but operates again a few hours later even without repairs or a battery change. You begin to suspect that the manufacturer designed it this way, perhaps to prevent you from listening to the radio all day. Now you want to find the device that turns it off whenever you play it for 4 hours. You are asking a new question. When you thought that the radio stopped because it needed repairs or new batteries, you did not ask which device turned it off.

Similarly, if we think of sleep as something like wearing out a machine, we do not ask which part of the brain produces it. But if we think of sleep as a specialized state evolved to serve particular functions, we look for the mechanisms that regulate it.

Sleep and Other Interruptions of Consciousness

Let’s start with some distinctions. Sleep is a state that the brain actively produces, characterized by decreased response to stimuli. In contrast, coma (KOH-muh) is an extended period of unconsciousness caused by head trauma, stroke, or disease. It is possible to awaken a sleeping person but not someone in a coma. A person in a coma has a low level of brain activity throughout the day, and little or no response to stimuli, including those that are ordinarily painful. Any movements that occur are purposeless and not directed toward anything. Typically, someone in a coma either dies or begins to recover within a few weeks.

Someone in a vegetative state alternates between periods of sleep and moderate arousal, although even during the more aroused state, the person shows no awareness of surroundings. Breathing is more regular, and a painful stimulus produces at least some autonomic responses of increased heart rate, breathing, and sweating. The person does not speak, respond to speech, or show any purposeful activity. However, people in this state probably have some cognitive activity (Guérit, 2005). A minimally conscious state is one stage higher, with occasional, brief periods of purposeful actions and a limited amount of speech comprehension. A vegetative or minimally conscious state can last for months or years.

Brain death is a condition with no sign of brain activity and no response to any stimulus. Physicians usually wait until someone has shown no sign of brain activity for 24 hours before pronouncing brain death, at which point most people believe it is ethical to remove life support.

I Stages of Sleep

Nearly every scientific advance comes from new or improved measurements. Researchers did not even suspect that sleep has different stages until they accidentally measured them. The electroencephalograph (EEG), as described in Chapter 4, records an average of the electrical potentials of the cells and fibers in the brain areas nearest each electrode on the scalp (Figure 9.10). If half the cells in some area increase their electrical potentials while the other half decrease, they cancel out. The EEG record rises or falls when most cells do the same thing at the same time. You might compare it to a record of the noise in a crowded sports stadium: It shows only slight fluctuations until some event gets everyone yelling at once. The EEG enables brain researchers to compare brain activity at different times during sleep.

Figure 9.11 shows data from a polysomnograph, a combination of EEG and eye-movement records, for a college student during various stages of sleep. Figure 9.11a presents a period of relaxed wakefulness for comparison. Note the steady series of alpha waves at a frequency of 8 to 12 per second. Alpha waves are characteristic of relaxation, not of all wakefulness.

In Figure 9.11b, sleep has just begun. During this period, called stage 1 sleep, the EEG is dominated by irregular, jagged, low-voltage waves. Overall brain activity is less than in relaxed wakefulness but higher than other sleep stages. As Figure 9.11c shows, the most prominent characteristics of stage 2 are sleep spindles and K-complexes. A sleep spindle consists of 12- to 14-Hz waves during a burst that lasts at least half a second. Sleep spindles result from oscillating interactions between cells in the thalamus and the cortex. A K-complex is a
sharp wave associated with temporary inhibition of neuronal firing (Cash et al., 2009).

In the succeeding stages of sleep, heart rate, breathing rate, and brain activity decrease, while slow, large-amplitude waves become more common (Figures 9.11d and e). By stage 4, more than half the record includes large waves of at least a half-second duration. Stages 3 and 4 together constitute slow-wave sleep (SWS).

Slow waves indicate that neuronal activity is highly synchronized. In stage 1 and in wakefulness, the cortex receives a great deal of input, much of it at high frequencies. Nearly all neurons are active, but different populations of neurons are active at different times. Thus, the EEG is full of short, rapid, choppy waves. By stage 4, however, sensory input to the cerebral cortex is greatly reduced, and the few remaining sources of input can synchronize many cells.

STOP & CHECK

6. What do long, slow waves on an EEG indicate?

**ANSWER**

Long, slow waves indicate a low level of activity, with much synchrony of response among neurons.

II Paradoxical or REM Sleep

Many discoveries occur when researchers stumble upon something by accident and then notice that it might be important. In the 1950s, French scientist Michel Jouvet was trying to test the learning abilities of cats after removal of the cerebral cortex. Because decorticate mammals don’t do much, Jouvet recorded slight movements of the muscles and EEGs from the hindbrain. During certain periods of apparent sleep, the cats’ brain activity was relatively high, but their neck muscles were completely relaxed. Jouvet (1960) then recorded the same phenomenon in normal, intact cats and named it paradoxical

![FIGURE 9.11 Polysomnograph records from a college student](https://example.com/figure911.png)

For each of these records, the top line is the EEG from one electrode on the scalp. The middle line is a record of eye movements. The bottom line is a time marker, indicating 1-second units. Note the abundance of slow waves in stages 3 and 4. (Records provided by T. E. LeVere)
sleep because it is deep sleep in some ways and light in others. (The term paradoxical means “apparently self-contradictory.”)

Meanwhile, in the United States, Nathaniel Kleitman and Eugene Aserinsky were observing eye movements of sleeping people as a means of measuring depth of sleep, assuming that eye movements would stop during sleep. At first, they recorded only a few minutes of eye movements per hour because the recording paper was expensive and they did not expect to see anything interesting in the middle of the night anyway. When they occasionally found periods of eye movements in people who had been asleep for hours, the investigators assumed that something was wrong with their machines. Only after repeated careful measurements did they conclude that periods of rapid eye movements occur during slow-wave sleep (Dement, 1990)

They called these periods rapid eye movement (REM) sleep (Aserinsky & Kleitman, 1955; Dement & Kleitman, 1957a) and soon realized that REM sleep was synonymous with what Jouvet called paradoxical sleep. Researchers use the term REM sleep when referring to humans but often prefer the term paradoxical sleep for nonhumans because many species lack eye movements.

During paradoxical or REM sleep, the EEG shows irregular, low-voltage fast waves that indicate increased neuronal activity. In this regard, REM sleep is light. However, the postural muscles of the body, including those that support the head, are more relaxed during REM than in other stages. In this regard, REM sleep is deep sleep. REM sleep is also associated with erections in males and vaginal moistening in females. Heart rate, blood pressure, and breathing rate are more variable in REM than in stages 2 through 4. In short, REM sleep combines deep sleep, light sleep, and features that are difficult to classify as deep or light. Consequently, it is best to avoid the terms deep and light sleep.

In addition to its steady characteristics, REM sleep has intermittent characteristics such as facial twitches and eye movements, as shown in Figure 9.11f. The EEG record is similar to that for stage 1 sleep, but notice the difference in eye movements. The stages other than REM are known as non-REM (NREM) sleep.

When you fall asleep, you start in stage 1 and slowly progress through stages 2, 3, and 4 in order, although loud noises or other intrusions can interrupt the sequence. After about an hour of sleep, you begin to cycle back from stage 4 through stages 3, 2, and then REM. The sequence repeats, with each cycle lasting about 90 minutes. (Some people have inferred that because a cycle lasts 90 minutes, you need to sleep at least 90 minutes to get any benefit. No evidence supports that claim.)

Early in the night, stages 3 and 4 predominate. Toward morning, REM occupies an increasing percentage of the time. Figure 9.12 shows typical sequences. The amount of REM depends on time of day more than how long you have been asleep. That is, if you go to sleep later than usual, you still increase your REM at about the same time that you would have ordinarily (Czeisler, Wetzman, Moore-Ede, Zimmerman, & Knauer, 1980).

Shortly after the discovery of REM, researchers believed it was almost synonymous with dreaming. William Dement and Nathaniel Kleitman (1957b) found that people who were awakened during REM reported dreams 80% to 90% of the time. Later research, however, found that people also some-

FIGURE 9.12 Sleep stages on three nights
Columns indicate awake (A) and sleep stages 2, 3, 4, and REM. Deflections in the line at the bottom of each chart indicate shifts in body position. Note that stage 4 sleep occurs mostly in the early part of the night’s sleep, whereas REM sleep becomes more prevalent toward the end. (Based on Dement & Kleitman, 1957a)
times report dreams when awakened from NREM sleep. REM dreams are more likely than NREM dreams to include striking visual imagery and complicated plots, but not always. Some people continue to report dreams despite an apparent lack of REM (Solms, 1997). In short, REM and dreams usually overlap, but they are not the same thing.

A cut through the midbrain decreases arousal by damaging the reticular formation, a structure that extends from the medulla into the forebrain. Some neurons of the reticular formation have axons ascending into the brain, and some have axons descending into the spinal cord. Those with axons descending into the spinal cord form part of the medial tract of motor control, as discussed in Chapter 8. In 1949, Giuseppe Moruzzi and H. W. Magoun proposed that those with ascending axons are well suited to regulate arousal. The term reticular (based on the Latin word rete, meaning “net”) describes the widespread connections among neurons in this system. One part of the reticular formation that contributes to cortical arousal is known as the pontomesencephalon (Woolf, 1996). (The term derives from pons and mesencephalon, or “midbrain.”) These neurons receive input from many sensory systems and generate spontaneous activity of their own. Their axons extend into the forebrain, as shown in Figure 9.13, releasing acetylcholine and glutamate, which excite cells in the hypothalamus, thalamus, and basal forebrain. Consequently, the pontomesencephalon maintains arousal during wakefulness and increases it in response to new or challenging tasks (Kinomura, Larsson, Gulyás, & Roland, 1996). Stimulation of the pontomesencephalon awakens a sleeping individual or increases alertness in one already awake, shifting the EEG from long, slow waves to short, high-frequency waves (Munk, Roelfsema, König, Engel, & Singer, 1996). However, subsystems within the pontomesencephalon control different sensory modalities, so a stimulus sometimes arouses one part of the brain more than others (Guillery, Feig, & Lozsádi, 1998). The locus coeruleus (LOW-kus ser-ROO-lee-us; literally, “dark blue place”), a small structure in the pons, is usually inactive, especially during sleep, but it emits bursts of impulses in response to meaningful events, especially those that produce emotional arousal (Sterpenich et al., 2006). Axons from the locus coeruleus release norepinephrine widely throughout the cortex, so this tiny area has a huge influence. Anything that stimulates the locus coeruleus strengthens the storage of recent memories (Clayton & Williams, 2000) and increases wakefulness (Berridge, Stellick, & Schmeichel, 2005).

The hypothalamus has several axon pathways that influence arousal. One pathway releases the neurotransmitter histamine (J.-S. Lin, Hou, Sakai, & Jouvet, 1996), which produces excitatory effects throughout the brain (Haas & Panula, 2003). Cells releasing histamine are active during arousal and alertness. As you might guess, they are less active when you are getting ready for sleep and when you have just awakened in the morning (K. Takahashi, Lin, & Sakai, 2006). Antihistamines, often used for allergies, counteract this transmitter and produce drowsiness. Antihistamines that do not cross the blood–brain barrier avoid that side effect.

Another pathway from the hypothalamus, mainly from the lateral and posterior nuclei of the hypothalamus, releases a peptide neurotransmitter called either orexin or hypocretin. For simplicity, this text will stick to the term orexin, but if you find the term hypocretin in other reading, it means the same thing.

### Brain Structures of Arousal and Attention

After a cut through the midbrain separates the forebrain and part of the midbrain from all the lower structures, an animal enters a prolonged state of sleep for the next few days. Even after weeks of recovery, the wakeful periods are brief. We might suppose a simple explanation: The cut isolated the brain from the sensory stimuli that come up from the medulla and spinal cord. However, if a researcher cuts each individual tract that enters the medulla and spinal cord, thus depriving the brain of the sensory input, the animal still has normal periods of wakefulness and sleep. Evidently, the midbrain does more than just relay sensory information; it has its own mechanisms to promote wakefulness.
thing. The axons releasing orexin extend to the basal forebrain and other areas, where they stimulate neurons responsible for wakefulness (Sakurai, 2007). Orexin is not necessary for waking up, but it is for staying awake. That is, most adult humans stay awake for roughly 16–17 hours at a time, even when nothing much is happening. Staying awake depends on orexin, especially toward the end of the day (Lee, Hassani, & Jones, 2005). A study of squirrel monkeys found that orexin levels rose throughout the day and remained high when the monkeys were kept awake beyond their usual sleep time. As soon as the monkeys went to sleep, the orexin levels dropped (Zeitzer et al., 2003). Mice lacking orexin fail to sustain activities, such as running in a running wheel, and therefore fall asleep at times when normal mice would remain alert (Anaclet et al., 2009). Drugs that block orexin receptors increase sleep (Brisbare-Roch et al., 2007), and procedures that increase orexin (e.g., a nasal spray of orexin) lead to increased wakefulness and alertness (Deadwyler, Porrino, Siegel, & Hampson, 2007; Prober, Rihel, Ohah, Sung, & Schier, 2006).

Other pathways from the lateral hypothalamus regulate cells in the basal forebrain (an area just anterior and dorsal to the hypothalamus). Basal forebrain cells provide axons that

**FIGURE 9.13 Brain mechanisms of sleeping and waking**

Green arrows indicate excitatory connections. Red arrows indicate inhibitory connections. Neurotransmitters are indicated where they are known. (Based on J.-S. Lin, Hou, Sakai, & Jouvet, 1996; Robbins & Everitt, 1995; Szymusiak, 1995)
extend throughout the thalamus and cerebral cortex (Figure 9.13). Some of these axons release acetylcholine, which is excitatory and tends to increase arousal (Mesulam, 1995; Szymusiak, 1995). Acetylcholine is released during wakefulness and REM sleep, but not during slow-wave sleep (Hassani, Lee, Henny, & Jones, 2009). During wakefulness, its release sharpens attention—that is, it increases the accurate, reliable detection of sensory stimuli (Goard & Dan, 2009).

Table 9.1 summarizes the effects of some key brain areas on arousal and sleep.

**STOP & CHECK**

9. Why do most antihistamines make people drowsy?

10. What would happen to the sleep–wake schedule of someone who lacked orexin?

### ANSWERS

9. Antihistamines cause drowsiness because they block the action of histamine, which has excitatory effects on the brain and tends to increase arousal.

10. Someone who lacks orexin would experience shorter periods of wakefulness and sleep. Orexin is released during REM sleep, so a person without orexin would have less REM sleep, potentially leading to numerous symptoms associated with REM sleep deprivation.

### Sleep and the Inhibition of Brain Activity

Figure 9.13 shows axons (in red) that lead to the release of GABA, the brain’s main inhibitory transmitter. GABA is responsible for sleep. During sleep, body temperature and metabolic rate decrease slightly, as does the activity of neurons, but by less than we might expect. Spontaneously active neurons continue to fire at close to their usual rate, and neurons in the brain's sensory areas continue to respond to sounds and other stimuli. Nevertheless, we are unconscious. The reason is that GABA inhibits synaptic activity. When a neuron is active, the increased GABA levels cut the activity short and prevent axons from spreading stimulation to other areas (Massimini et al., 2005). Connections from one brain area to another become weaker (Esser, Hill, & Tononi, 2009). When stimulation doesn’t spread, you don’t become conscious of it. (Chapter 14 elaborates on that point.)

Because sleep depends on GABA-mediated inhibition, it can be local within the brain (Krueger et al., 2008). That is, you might have substantial inhibition in one brain area and not so much in another. Ordinarily, different brain areas wake up or go to sleep at almost the same time, but not necessarily. The most extreme case of this principle occurs in dolphins and other aquatic mammals. At night, they need to be alert enough to surface for a breath of air. They have evolved the ability to sleep on one side of the brain at a time. That is, the two hemispheres take turns sleeping, always leaving one awake enough to control swimming and breathing (Rattenborg, Amlaner, & Lima, 2000).

Thinking of sleep as a local phenomenon helps make sense of some otherwise puzzling phenomena. Take, for instance, sleepwalking. Almost by definition, a sleepwalker is awake in one part of the brain and asleep in another. Another example is lucid dreaming. During lucid dreaming, someone is dreaming but aware of being asleep and dreaming. Evidently some brain area is more awake than usual during dreaming. Another example: Have you ever had the experience of waking up but finding that you can’t move your arms or legs? During REM sleep, cells in the pons send messages that inhibit the motor neurons that control the body’s large muscles. A cat with damage to those cells moves around awkwardly during REM sleep, as if it were acting out its dreams (Morrison, Sanford, Ball, Mann, & Ross, 1995) (Figure 9.14). Ordinarily, when you awaken from a REM period, those cells in the pons shut off and you regain muscle control. But occasionally most of the brain wakes up while the pons remains in REM. The result is your experience of being temporarily unable to move—a very unsettling experience, if you don’t understand it.

### TABLE 9.1  Brain Structures for Arousal and Sleep

<table>
<thead>
<tr>
<th>Structure</th>
<th>Neurotransmitter(s) It Releases</th>
<th>Effects on Behavior</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pontomesencephalon</td>
<td>Acetylcholine, glutamate</td>
<td>Increases cortical arousal</td>
</tr>
<tr>
<td>Locus coeruleus</td>
<td>Norepinephrine</td>
<td>Increases information storage during wakefulness, suppresses REM sleep</td>
</tr>
<tr>
<td>Basal forebrain</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Excitatory cells</td>
<td>Acetylcholine</td>
<td>Excites thalamus and cortex; increases learning, attention; shifts sleep from NREM to REM</td>
</tr>
<tr>
<td>Inhibitory cells</td>
<td>GABA</td>
<td>Inhibits thalamus and cortex</td>
</tr>
<tr>
<td>Hypothalamus (parts)</td>
<td>Histamine</td>
<td>Increases arousal</td>
</tr>
<tr>
<td>(parts)</td>
<td>Orexin</td>
<td>Maintains wakefulness</td>
</tr>
<tr>
<td>Dorsal raphe and pons</td>
<td>Serotonin</td>
<td>Interrupts REM sleep</td>
</tr>
</tbody>
</table>

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Brain Function in REM Sleep

Researchers interested in the brain mechanisms of REM decided to use a PET scan to determine which areas increased or decreased in activity during REM. Although that research might sound simple, a PET scan requires injecting a radioactive chemical. Imagine trying to give sleepers an injection without awakening them. Further, a PET scan yields a clear image only if the head remains motionless during data collection. If the person tosses or turns even slightly, the image is worthless.

To overcome these difficulties, researchers in two studies persuaded young people to sleep with their heads firmly attached to masks that did not permit any movement. They also inserted a cannula (plastic tube) into each person’s arm so that they could inject radioactive chemicals at various times during the night. So imagine yourself in that setup. You have a cannula in your arm and your head is locked into position. Now try to sleep.

Because the researchers foresaw the difficulty of sleeping under these conditions (!), they had their participants stay awake the entire previous night. Someone who is tired enough can sleep even under trying circumstances. (Maybe.)

Now that you appreciate the heroic nature of the procedures, here are the results. During REM sleep, activity increased in the pons (which triggers the onset of REM sleep) and the limbic system (which is important for emotional responses). Activity decreased in the primary visual cortex, the motor cortex, and the dorsolateral prefrontal cortex but increased in parts of the parietal and temporal cortex (Braun et al., 1998; Maquet et al., 1996).

REM sleep is associated with a distinctive pattern of high-amplitude electrical potentials known as PGO waves, for pons-geniculate-occipital (Figure 9.15). Waves of neural activity are detected first in the pons, shortly afterward in the lateral geniculate nucleus of the thalamus, and then in the occipital cortex (D. C. Brooks & Bizzi, 1963; Laurent, Cespuglio, & Jouvet, 1974). Each animal maintains a nearly constant amount of PGO waves per day. During a prolonged period of REM deprivation in laboratory animals, PGO waves begin to emerge during sleep stages 2 to 4 and even during wakefulness, often in association with strange behaviors, as if the animal were hallucinating. At the end of the deprivation period, when an animal is permitted to sleep without interruption, the REM periods have an unusually high density of PGO waves.

REM sleep apparently depends on a relationship between the neurotransmitters serotonin and acetylcholine. Injections of the drug carbachol, which stimulates acetylcholine synapses, quickly move a sleeper into REM sleep (Baghdoyan, Spotts, & Snyder, 1993). Note that acetylcholine is important for both wakefulness and REM sleep, states of brain arousal. Serotonin and norepinephrine interrupt REM sleep (Boutrel, Franc, Hen, Hamon, & Adrien, 1999; Singh & Mallick, 1996).

FIGURE 9.14. A cat with a lesion in the pons, wobbling about during REM sleep

Cells of an intact pons send inhibitory messages to the spinal cord neurons that control the large muscles. (From Morrison, A. R., Sanford, L. D., Ball, W. A., Mann, G. L., & Ross, R. J., “Stimulus-elicited behavior in rapid eye movement sleep without atonia,” Behavioral Neuroscience, 109, 972–979, 1995. Published by APA and reprinted by permission.)

STOP & CHECK

11. What would happen to the sleep–wake schedule of someone who took a drug that blocked GABA?

12. Someone who has just awakened sometimes speaks in a loose, unconnected, illogical way. How could you explain this finding?

ANSWERS

11. Someone who took a drug that blocks GABA would remain awake. (Tranquilizers put people to sleep by facilitating GABA.)

12. People often awaken from a REM period, because REM is abundant toward morning when people usually awaken. Different brain areas don’t wake up all at once. Shortly after awakening, parts of the brain may still be in a REM-like state, and people often experience strange dreams. People who take drugs that block GABA often experience these dreams as well.
9.2 Stages of Sleep and Brain Mechanisms

Sleep Disorders

How much sleep is enough? Different people need different amounts. Most adults need about 7 1/2 to 8 hours of sleep per night, but some have been known to get by with less than 3 hours per night, without unpleasant consequences (H. S. Jones & Oswald, 1968; Meddis, Pearson, & Langford, 1973). Among people who ordinarily get the more typical 7 1/2 to 8 hours of sleep, some are better than others at withstanding a temporary lack of sleep. People who tolerate sleep deprivation relatively well are usually “evening people,” who like to waken late and stay up late. They tend to show greater than average levels of brain arousal, as indicated by fMRI (Caldwell et al., 2005).

The best gauge of insomnia—inadequate sleep—is how someone feels the following day. If you feel tired during the day, you are not sleeping enough at night. Causes of insomnia include noise, uncomfortable temperatures, stress, pain, diet, and medications. Insomnia can also be the result of epilepsy, Parkinson’s disease, brain tumors, depression, anxiety, or other neurological or psychiatric conditions. Some children suffer insomnia because they are milk-intolerant, and their parents, not realizing the intolerance, give them milk to drink right before bedtime (Horne, 1992). One man suffered insomnia until he realized that he dreaded going to sleep because he hated waking up to go jogging. After he switched his jogging time to late afternoon, he slept without difficulty. In short, try to identify the reasons for your sleep problems before you try to solve them.

Some cases of insomnia relate to shifts in circadian rhythms (MacFarlane, Cleghorn, & Brown, 1985a, 1985b). Ordinarily, people fall asleep while their temperature is declining and awaken while it is rising, as in Figure 9.16a. Someone whose rhythm is phase-delayed, as in Figure 9.16b, has trouble falling asleep at the usual time, as if the hypothalamus thinks it isn’t late enough (Morris et al., 1990). Someone whose rhythm is phase-advanced, as in Figure 9.16c, falls asleep easily but awakens early.

Another cause of insomnia is, paradoxically, the use of tranquilizers as sleeping pills. Although tranquilizers help people fall asleep, repeated use causes dependence and an inability to sleep without the pills (Kales, Scharf, & Kales, 1978). Similar problems arise when people use alcohol to get to sleep.

Sleep Apnea

One type of insomnia is sleep apnea, impaired ability to breathe while sleeping. People with sleep apnea have breathless periods of a minute or so from which they awaken gasping for breath. They may not remember all their awakenings, although they certainly notice the consequences—sleepiness during the day, impaired attention, depression, and sometimes heart problems. People with sleep apnea have multiple brain areas that appear to have lost neurons, and consequently, they show deficiencies of learning, reasoning, attention, and impulse control (Beebe & Gozal, 2002; Macey et
al., 2002). These correlational data do not tell us whether the brain abnormalities led to sleep apnea or sleep apnea led to the brain abnormalities. However, research with rodents suggests the latter: Mice that are subjected to frequent periods of low oxygen (as if they hadn’t been breathing) lose some neurons and impair others, especially in areas responsible for alertness (Zhu et al., 2007). Sleep impairments may be responsible for cognitive loss not only in people with sleep apnea but also in some with Alzheimer’s disease.

Sleep apnea results from several causes, including genetics, hormones, and old-age deterioration of the brain mechanisms that regulate breathing. Another cause is obesity, especially in middle-aged men. Many obese men have narrower than normal airways and have to compensate by breathing frequently or vigorously. During sleep, they cannot keep up that rate of breathing. Furthermore, their airways become even narrower than usual when they adopt a sleeping posture (Mezzanotte, Tangel, & White, 1992).

People with sleep apnea are advised to lose weight and avoid alcohol and tranquilizers (which impair the breathing muscles). Medical options include surgery to remove tissue that obstructs the trachea (the breathing passage) or a mask that covers the nose and delivers air under enough pressure to keep the breathing passages open (Figure 9.17).

**Narcolepsy**

Narcolepsy, a condition characterized by frequent periods of sleepiness during the day (Aldrich, 1998), strikes about 1 person in 1,000. It sometimes runs in families, but many cases emerge in people with no affected relatives. No gene for narcolepsy has been identified, and many people with narcolepsy have no close relatives with the disease. Narcolepsy has four main symptoms, although not every patient has all four. Each of these symptoms can be interpreted as an intrusion of a REM-like state into wakefulness:

1. Gradual or sudden attacks of sleepiness during the day.
2. Occasional cataplexy—an attack of muscle weakness while the person remains awake. Cataplexy is often triggered by strong emotions, such as anger or great excitement. (One man suddenly collapsed during his own wedding ceremony.)
3. Sleep paralysis—an inability to move while falling asleep or waking up. Other people may experience sleep paralysis occasionally, but people with narcolepsy experience it more frequently.
4. Hypnagogic hallucinations—dreamlike experiences that the person has trouble distinguishing from reality, often occurring at the onset of sleep.

**FIGURE 9.17** A Continuous Positive Airway Pressure (CPAP) mask

The mask fits snugly over the nose and delivers air at a fixed pressure, strong enough to keep the breathing passages open.
The cause relates to the neurotransmitter orexin. People with narcolepsy lack the hypothalamic cells that produce and release orexin (Thanickal et al., 2000). Why they lack them is unknown, but one possibility is an autoimmune reaction, in which the immune system attacks part of the body—in this case, cells with orexin (Hallmayer et al., 2009). Recall that orexin is important for maintaining wakefulness. Consequently, people lacking orexin alternate between short waking periods and short sleepy periods, instead of staying awake throughout the day. Dogs that lack the gene for orexin receptors have symptoms much like human narcolepsy, with frequent alternations between wakefulness and sleep (L. Lin et al., 1999). The same is true for mice that lack orexin (Hara, 2001; Mochizuki et al., 2004).

As discussed in Chapter 8, people with Huntington’s disease have widespread damage in the basal ganglia. In addition, most lose neurons in the hypothalamus, including the neurons that make orexin. As a result, they have problems staying awake during the day and difficulty staying asleep at night (Morton et al., 2005).

Theoretically, we might imagine combating narcolepsy with drugs that restore orexin. Perhaps eventually, such drugs will become available. Currently, the most common treatment is stimulant drugs, such as methylphenidate (Ritalin), which enhance dopamine and norepinephrine activity.

**STOP & CHECK**

**14. What is the relationship between orexin and narcolepsy?**

**ANSWER**

Dogs of sleepiness during the day, loss of orexin, decreased orexin, characterized by people of normal living either orexin or the effect. Orexin is important for staying awake. Therefore, orexin is the key to understanding narcolepsy.

---

**Periodic Limb Movement Disorder**

Another sleep disorder is periodic limb movement disorder, characterized by repeated involuntary movement of the legs and sometimes the arms (Edinger et al., 1992). Many people, perhaps most, experience an occasional involuntary kick, especially when starting to fall asleep. Leg movements are not a problem unless they become persistent. In some people, mostly middle-aged and older, the legs kick once every 20 to 30 seconds for minutes or hours, mostly during NREM sleep. Frequent or especially vigorous leg movements may awaken the person or his or her partner. In some cases, tranquilizers help suppress the movements (Schenck & Mahowald, 1996).

**REM Behavior Disorder**

For most people, the major postural muscles are relaxed and inactive during REM sleep. However, people with REM behavior disorder move around vigorously during their REM periods, apparently acting out their dreams. They frequently dream about defending themselves against attack, and they may punch, kick, and leap about. Most of them injure themselves or other people and damage property (Olson, Boeve, & Silber, 2000).

REM behavior disorder occurs mostly in older people, especially older men with brain diseases such as Parkinson’s disease (Olson et al., 2000). Presumably, the damage includes the cells in the pons that send messages to inhibit the spinal neurons that control large muscle movements.

**Night Terrors and Sleepwalking**

Night terrors are experiences of intense anxiety from which a person awakens screaming in terror. A night terror is more severe than a nightmare, which is simply an unpleasant dream. Night terrors occur during NREM sleep and are more common in children than adults. Dream content, if any, is usually simple, such as a single image.

Sleepwalking runs in families and occurs mostly in children. Most people who sleepwalk, and many of their relatives, have one or more additional sleep difficulties, such as chronic snoring, disorders of sleep breathing, bedwetting, and night terrors (Cao & Guilleminault, 2010). The causes of sleepwalking are not well understood, but it is more common when people are sleep deprived or under unusual stress (Zadra & Pilon, 2008). It is most common during stage 3 or 4 sleep early in the night and is usually not accompanied by dreaming. (It does not occur during REM sleep, when the large muscles are completely relaxed.) Sleepwalking is usually harmless but not always. One teenage girl walked out of her house, climbed a crane, and went back to sleep on a support beam. Fortunately, a pedestrian saw her and called the police. Sleepwalkers have been known to eat, rearrange furniture, fall off balconies, and drive cars—while disregarding lanes and traffic lights. Unlike wakeful actions, the deeds of sleepwalkers are poorly planned and not remembered. Evidently, parts of the brain are awake and other parts are asleep (Gunn & Gunn, 2007). Incidentally, contrary to common sayings, it is not dangerous to awaken a sleepwalker. It is not particularly helpful either, but it is not dangerous.

An analogous condition is sleep sex or “sexsomnia,” in which sleeping people engage in sexual behavior, either with a partner or by masturbation, and do not remember it afterward. Sexsomnia poses a threat to romances and marriages. As one woman said, “After getting married a few years ago, my husband told me I was masturbatıng in my sleep. I was mortified, thinking back to all the slumber parties as a girl, and then when I was older and my little sister stayed the night at my house! How many others might have witnessed and not said anything? My new marriage is on the rocks, and I’m having such good sex in my sleep, I have NO desire while I’m awake. This is killing my relationship with my husband.” (Mangan, 2004, p. 290)
Chemists divide the world into different elements, biologists divide life into different species, and physicians distinguish one disease from another. Similarly, psychologists try to recognize the most natural or useful distinctions among types of behavior or experience. The discovery of different stages of sleep was a major landmark in psychology because researchers found a previously unrecognized distinction that is both biologically and psychologically important. It also demonstrated that external measurements—in this case, EEG recordings—can be used to identify internal experiences. We now take it largely for granted that an electrical or magnetic recording from the brain can tell us something about a person’s experience, but it is worth pausing to note what a surprising discovery that was in its time.

**SUMMARY**

1. During sleep, brain activity decreases, but a stimulus can awaken the person. Someone in coma cannot be awakened. A vegetative state or minimally conscious state can last months or years, during which the person shows only limited responses. Brain death is a condition without brain activity or responsiveness of any kind. 276
2. Over the course of about 90 minutes, a sleeper goes through stages 1, 2, 3, and 4 and then returns through stages 3 and 2 to a stage called REM. REM is characterized by rapid eye movements, more brain activity than other sleep stages, complete relaxation of the trunk muscles, irregular breathing and heart rate, penile erection or vaginal lubrication, and an increased probability of vivid dreams. 276
3. REM sleep or paradoxical sleep is a condition marked by more cortical activity than other sleep, complete relaxation of the body’s postural muscles, and an increased probability of dreaming. 277
4. The brain has multiple systems for arousal. The pontomesencephalon and parts of the hypothalamus control various cell clusters in the basal forebrain that send axons releasing acetylcholine throughout much of the forebrain. 279
5. The locus coeruleus is active in response to meaningful events. It facilitates attention and new learning; it also blocks the onset of REM sleep. 279
6. Orexin is a peptide that maintains wakefulness. Cells in the lateral and posterior nuclei of the hypothalamus release this peptide. 279
7. During sleep, enhanced release of GABA limits neuronal activity and blocks the spread of activation. Sometimes this suppression is stronger in one brain area than another. That is, sleep can occur in one brain area and not another at a given time. 281
8. REM sleep is associated with increased activity in a number of brain areas, including the pons and limbic system. Activity decreases in the prefrontal cortex, the motor cortex, and the primary visual cortex. 282
9. REM sleep begins with PGO waves, which are waves of brain activity transmitted from the pons to the lateral geniculate to the occipital lobe. 282
10. People with sleep apnea have long periods without breathing while they sleep. Many have indications of neuronal loss, probably as a result of decreased oxygen while they sleep. 283
11. People with narcolepsy have attacks of sleepiness during the day. Narcolepsy is associated with a deficiency of the peptide neurotransmitter orexin. 284

**KEY TERMS**

Terms are defined in the module on the page number indicated. They’re also presented in alphabetical order with definitions in the book’s Subject Index/Glossary, which begins on page 561. Interactive flashcards and crossword puzzles are among the online resources available to help you learn these terms and the concepts they represent.

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coma 276  
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K-complex 276  
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THOUGHT QUESTION

When cats are deprived of REM sleep, longer periods of deprivation—up to about 25 days—are associated with greater rebound of REM when they can sleep uninterrupted. However, REM deprivation for more than 25 days produces no additional rebound. Speculate on a possible explanation. (Hint: Consider what happens to PGO waves during REM deprivation.)
Why Sleep? Why REM? Why Dreams?

FUNCTIONS OF SLEEP

Sleep serves many functions. During sleep, we rest our muscles, decrease metabolism, rebuild proteins in the brain (Kong et al., 2002), reorganize synapses, and strengthen memories (Sejnowski & Destexhe, 2000). People who don’t get enough sleep have trouble concentrating and become more vulnerable to illness, especially mental illness (Wulff, Gatti, Wettstein, & Foster, 2010). We all have moments when our attention lapses and we fail to notice important stimuli. Those periods are longer and more frequent after a sleepless night. Furthermore, people who have had enough sleep notice their lapses and jar themselves into increased arousal. People who are sleep-deprived fail to do so (Chee et al., 2008). Inadequate sleep is a major cause of accidents by workers and poor performance by college students. Driving while sleep deprived is comparable to driving under the influence of alcohol (Falleti, Maruff, Collie, Darby, & McStephen, 2003).

People working in Antarctica during the winter sleep poorly and feel depressed (Palinkas, 2003). Even one night of sleeplessness activates the immune system (Matsumoto et al., 2001). That is, you react to sleep deprivation as if you were ill. With more prolonged sleep deprivation, people report dizziness, tremors, and hallucinations (Dement, 1972; L. C. Johnson, 1969).

Clearly, we need to sleep. Is there, however, one primary or original reason?

SLEEP AND ENERGY CONSERVATION

Even if we identified what seems to be the most important function of sleep for humans today, it might not be the function for which sleep originally evolved. By analogy, consider computers: People use computers today to write papers, send e-mail, search the Internet, play video games, store and display photographs, play music, and find a date. Someone who didn’t know the history might not guess that computers were built originally for mathematical calculations.

Similarly, sleep probably started with a simple function to which evolution added others later. Even bacteria have circadian rhythms of activity and inactivity (Mihalcescu, Hsing, & Leibler, 2004). What benefit of sleep applies to species with little or no nervous system?

A likely hypothesis is that sleep’s original function—and still an important one—is to save energy (Kleitman, 1963; Siegel, 2009; Webb, 1974). Nearly every species is more efficient at some times of day than at others. Those with good vision are more efficient in the day. Those that rely on olfaction instead of vision are more efficient at night, when their predators cannot see them. Sleep conserves energy during the inefficient times, when activity would be wasteful and possibly dangerous. NASA’s Rover spacecraft, built to explore Mars, had a mechanism to make it “sleep” at night to conserve its batteries. During sleep, a mammal’s body temperature decreases by 1° or 2° C, enough to save a significant amount of energy. Muscle activity decreases, saving more energy. Animals increase their sleep duration during food shortages, when energy conservation is especially important (Berger & Phillips, 1995).

Sleep is therefore in some ways analogous to hibernation. Hibernation is a true need. A ground squirrel that is prevented from hibernating can become as disturbed as a person who is prevented from sleeping. However, the function of hibernation is simply to conserve energy while food is scarce.

If one of the main functions of sleep is to shut down activity at times of relative inefficiency, we might expect to find little or no sleep in species that are equally effective at all times of day. Indeed, that expectation appears to be confirmed. Certain fish have evolved for life in a cave where “day” and “night” have
no meaning, because light is always absent and temperature is virtually constant. Observers report that these fish apparently never sleep (Kavanau, 1998).

Several other species turn off their need for sleep under certain circumstances (Siegel, 2009). After a dolphin or whale gives birth, both mother and baby stay awake 24 hours a day for the first couple of weeks while the baby is especially vulnerable. Neither shows any sign of harm from sleep deprivation. Migratory birds face a different kind of problem. During a week or two in fall and spring, they forage for food during the day and do their migratory flying at night. (Flying at night makes sense, because it is cooler then.) That schedule leaves little time for sleep. They apparently decrease their need for sleep at this time. If a bird is kept in a cage during the migration season, it flutters around restlessly at night, sleeping only one third its usual amount. It compensates to some extent with many brief periods of drowsiness (less than 30 seconds each) during the day (Fuchs, Haney, Jechura, Moore, & Bingman, 2006). Still, it is getting very little sleep, while remaining alert and performing normally on learning tasks. If the same bird is deprived of sleep during other seasons of the year, its performance suffers (Rattenborg et al., 2004). Exactly how a bird or a mother dolphin decreases its sleep need is unknown, but the fact that it is possible fits with the idea that sleep is primarily a way to conserve energy, rather than a way to fulfill a function that one could not fulfill in other ways.

Animal species vary in their sleep habits in ways that make sense if we ask how many hours the animal needs to be awake, and therefore how long it can afford to spend conserving energy. Migratory birds need to eat for many hours per day get less sleep than carnivores (meat eaters) that can satisfy their nutritional needs with a single meal. Animals that need to be on the alert for predators get little sleep, whereas the predators themselves sleep easily. Insect-eating bats are active in the early evening, when moths and similar insects are most abundant, and then they sleep the rest of the day (Figure 9.18).

Here’s another bit of miscellaneous trivia about animal sleep: Swifts are small, dark birds that chase insects. They get all the nutrition and water they need from the insects. When a baby European swift first takes off from its nest, how long would you guess its first flight lasts, until it comes to land again?

The answer: up to 2 years. Except during treacherous storms, it doesn’t come down until it is old enough to mate and build a nest. In the meantime, it spends both days and nights in the air. At night it heads into the wind, sticks out its wings, glides, and presumably sleeps—although confirming sleep would require measuring the EEG of a small bird in flight. It picks an altitude where the air is not too cold, accepts the risk of being blown a great distance, and awakens the next morning to resume its chase of flying insects (Bäckman & Alerstam, 2001).
CHAPTER 9
Wakefulness and Sleep

Sleep and Memory

Another apparent function of sleep is improved memory. Young adults deprived of a night’s sleep show deficits on memory tasks (Yoo, Hu, Gujar, Jolesz, & Walker, 2007). In contrast, if people learn something and then go to sleep, or even take a nap, their memory often improves beyond what it was before the sleep (Hu, Stylos-Allan, & Walker, 2006; Korman et al., 2007). That is, we see not just an absence of forgetting but also a gain of memory. The amount of improvement varies from one study to another and from one type of learning task to another (Cai & Rickard; Cai, Shuman, Gorman, Sage, & Anagnostaras, 2009; Doyon et al., 2009). Still, it appears that reviewing something right before you go to sleep is an excellent idea.

Sleep enhances memory of some events more than others. In one study, people viewed 50 objects making a sound (such as a cat meowing) in various locations on a computer screen. Then they took a nap, during which they heard some of those sounds again. After the nap, they were tested on their memory for the location of each object on the screen. They showed enhanced memory for the objects whose sounds they heard during the nap (Rudy, Voss, Westerberg, & Paller, 2009). Evidently the sounds reminded them of the information, and the brain then processed that information again.

Sleep also helps people reanalyze their memories: In one study, people who had just practiced a complex task were more likely to perceive a hidden rule (an “aha” experience) after a period of sleep than after a similar period of wakefulness (Wagner, Gais, Haider, Verleger, & Born, 2004). Another study found that a nap that included REM sleep enhanced performance on certain kinds of creative problem solving (Cai, Mednick, Harrison, Kanady, & Mednick, 2009).

How does sleep enhance memory? Researchers recorded activity in the hippocampus during learning, and then recorded from the same locations during sleep, using microelectrodes within cells for laboratory animals and electrodes on the scalp for humans. The results: Patterns that occurred during sleep resembled those that occurred during learning, except that they were more rapid during sleep. Furthermore, the amount of hippocampal activity during sleep correlated highly with the subsequent improvement in performance (Derégnaucourt, Mitra, Fehér, Pytte, & Tchernichovski, 2005; Euston, Tatsuno, & McNaughton, 2007; Huber, Ghilardi, Massimini, & Tononi, 2004; Ji & Wilson, 2007; Maquet et al., 2000; Peigneux et al., 2004). These results suggest that the brain replays its daily experiences during sleep. However, further research found that the sleeping brain replays its experience backward as often as forward, and that it sometimes replays less common experiences more often than

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**FIGURE 9.18 Hours of sleep per day for various species**

Generally, predators and others that are safe when they sleep tend to sleep a great deal. Animals in danger of being attacked while they sleep spend less time asleep. (© Cengage Learning 2013)
more common ones (Gupta, van der Meer, Touretzky, & Redish, 2010). Also, the hippocampus replays recently learned patterns during quiet waking periods, not just during sleep (Karlsson & Frank, 2009). So the role of hippocampal replay during sleep is less clear than it once appeared to be.

One way for sleep to strengthen memory is by weeding out the less successful connections. In Chapter 13, we shall examine the phenomenon of long-term potentiation, the ability of new experiences to strengthen synaptic connections. Suppose that every time you learn something, your brain strengthened certain synapses without making adjustments elsewhere. As you learned more and more, you would have more and more brain activity. By middle age, your brain might be burning with constant activity. To prevent runaway overactivity, your brain compensates for strengthening some synapses by weakening others, mostly during sleep (Liu, Faraguna, Cirelli, Tononi, & Gao, 2010; Vyazovskiy, Cirelli, Pfister-Genskow, Faraguna, & Tononi, 2008). Weakening synapses during sleep emphasizes the ones that were strengthened during wakefulness.

Another aspect of sleep’s contribution to memory relates to sleep spindles. Recall that sleep spindles are waves of activity, about 12–14 Hz, that occur mostly during stage 2 sleep. They indicate an exchange of information between the thalamus and cerebral cortex. In both rats and humans, sleep spindles increase in number after new learning (Eschenko, Mölle, Born, & Sara, 2006). Most people are fairly consistent in their amount of spindle activity from one night to another, and the amount of spindle activity correlates more than .7 with nonverbal tests of IQ (Fogel, Nader, Cote, & Smith, 2007). Who would have guessed that brain waves during sleep could predict IQ scores?

STOP & CHECK

17. Does sleep improve memory by strengthening or weakening synapses?

ANSWER

The evidence so far points to weakening the less relevant synapses as opposed to strengthening them. Who wants to be extra relevant? Synaptic pruning during sleep emphasizes the strengthened synapses during the day.

Functions of REM Sleep

An average person spends about one third of his or her life asleep and about one fifth of sleep in REM, totaling about 600 hours of REM per year. Presumably, REM serves a biological function. But what is it?

One way to approach this question is to compare the people or animals with more REM to those with less. REM sleep is widespread in mammals and birds, indicating that it is part of our ancient evolutionary heritage. Some species, however, have far more than others. As a rule, the species with the most total sleep hours also have the highest percentage of REM sleep (J. M. Siegel, 1995). Cats spend up to 16 hours a day sleeping, much or most of it in REM sleep. Rabbits, guinea pigs, and sheep sleep less and spend little time in REM.

Figure 9.19 illustrates the relationship between age and REM sleep for humans. The trend is the same for other mammalian species. Infants get more REM and more total sleep than adults do, confirming the pattern that more total sleep predicts a higher percentage of REM sleep. Among adult humans, those who sleep 9 or more hours per night have the highest percentage of REM sleep, and those who sleep 5 or fewer hours have the lowest percentage. This pattern implies that although REM is no doubt important, NREM is more tightly regulated. That is, the amount of NREM varies less among individuals and among species.

One hypothesis is that REM is important for memory storage, especially for weakening the inappropriate connections (Crick & Mitchison, 1983). REM and non-REM sleep may be important for consolidating different types of memories. Depriving people of sleep early in the night (mostly non-REM sleep) impairs verbal learning, such as memorizing a list of words, whereas depriving people of sleep during the second half of the night (more
REM) impairs consolidation of learned motor skills (Gais, Plihal, Wagner, & Born, 2000; Plihal & Born, 1997).

However, many people take antidepressant drugs that severely decrease REM sleep, without incurring memory problems (Rasch, Pomer, Diekelmann, & Born, 2009). Research on laboratory animals indicates that these drugs sometimes even enhance memory (Parent, Habib, & Baker, 1999).

Another hypothesis sounds odd because we tend to imagine a glamorous role for REM sleep: David Maurice (1998) proposed that REM just shakes the eyeballs back and forth enough to get sufficient oxygen to the corneas of the eyes. The corneas, unlike the rest of the body, get oxygen directly from the surrounding air. During sleep, because they are shielded from the air, they deteriorate slightly (Hoffmann & Curio, 2003). They do get some oxygen from the fluid behind them (see Figure 6.2), but when the eyes are motionless, that fluid becomes stagnant. Moving the eyes increases the oxygen supply to the corneas. According to this view, REM is a way of arousing a sleeper just enough to shake the eyes back and forth, and the other manifestations of REM are just byproducts. This idea makes sense of the fact that REM occurs mostly toward the end of the night’s sleep, when the fluid behind the eyes would be the most stagnant. It also makes sense of the fact that individuals who spend more hours asleep devote a greater percentage of sleep to REM. (If you don’t sleep long, you have less need to shake up the stagnant fluid.) However, as mentioned, many people take antidepressants that restrict REM sleep. They are not known to suffer damage to the cornea.

18. What kinds of individuals get more REM sleep than others? (Think in terms of age, species, and long versus short sleepers.)

ANSWER

18a. REM sleep is more typical of the young than the old, of individuals who get more sleep than those who get little, and of species that sleep much of the day. A comparison of newborns with the elderly shows that they get about the same amount of REM sleep at about the same times of day. In fact, 2- or 3-month-old infants display sleep patterns similar to those of college students. The amount of REM sleep decreases as individuals age. The details are lost as we age, and the details fade quickly.

**The Activation-Synthesis Hypothesis**

According to the activation-synthesis hypothesis, a dream represents the brain’s effort to make sense of sparse and distorted information. Dreams begin with periodic bursts of spontaneous activity in the pons—the PGO waves previously described—that activate some parts of the cortex but not others. The cortex combines this haphazard input with whatever other activity was already occurring and does its best to synthesize a story that makes sense of the information (Hobson & McCarley, 1977; Hobson, Pace-Schott, & Stickgold, 2000; McCarley & Hoffman, 1981). Sensory stimuli, such as sounds in the room, occasionally get incorporated into a dream, although usually they do not (Nir & Tononi, 2010).

Consider how this theory handles a couple of common dreams. Most people have occasional dreams of falling or flying. Well, while you are asleep, you lie flat, unlike your posture for the rest of the day. Your brain in its partly aroused condition feels the vestibular sensation of your position and interprets it as falling or flying. Have you ever dreamed that you were trying to move but couldn’t? Most people have. An interpretation based on the activation-synthesis theory is that during REM sleep (which accompanies most dreams), your motor cortex is inactive and your major postural muscles are virtually paralyzed. That is, when you are dreaming, you really can’t move, you feel your lack of movement, and thus, you dream of failing to move.

One criticism is that the theory’s predictions are vague. If we dream about falling because of the vestibular sensations from lying down, why don’t we always dream of falling? If we dream we can’t move because our muscles are paralyzed during REM sleep, why don’t we always dream of being paralyzed?

**The Clinico-Anatomical Hypothesis**

An alternative view of dreams has been labeled the clinico-anatomical hypothesis because it was derived from clinical studies of patients with various kinds of brain damage (Solms, 1997, 2000). Like the activation-synthesis theory, this theory emphasizes that dreams begin with arousing stimuli that are generated within the brain combined with recent memories and any information the brain is receiving from the senses. However, the clinico-anatomical hypothesis puts less emphasis on the pons, PGO waves, or REM sleep. It regards dreams as thinking that takes place under unusual conditions, similar to mind-wandering during everyday life (Domhoff, 2011).

One of those conditions is that the brain is getting little information from the sense organs, and the primary visual and auditory areas of the cortex have lower than usual activity, so other brain areas are free to generate images without constraints or interference. Also, the primary motor cortex is suppressed, as are the motor neurons of the spinal cord, so arousal cannot lead to action. Activity is suppressed in the prefrontal cortex, which is important for working memory (memory of very recent events). Consequently, we not only forget most dreams after we awaken, but we also lose track of what has been happening within a dream, and sudden scene changes are common. We also lose a sense of volition—that is, planning (Hobson, 2009). It seems that events just happen, without any intention on our part.
Meanwhile, activity is relatively high in the inferior (lower) part of the parietal cortex, an area important for visuospatial perception. Patients with damage here have problems binding body sensations with vision. They also report no dreams. Fairly high activity is also found in the areas of visual cortex outside V1. Those areas are presumably important for the visual imagery that accompanies most dreams. Finally, activity is high in the hypothalamus, amygdala, and other areas important for emotions and motivations (Gvilia, Turner, McGinty, & Szymusiak, 2006).

So the idea is that either internal or external stimulation activates parts of the parietal, occipital, and temporal cortex. The arousal develops into a hallucinatory perception, with no sensory input from area V1 to override it. This idea, like the activation-synthesis hypothesis, is hard to test because it does not make specific predictions about who will have what dream and when.

STOP & CHECK

19. What is a key point of disagreement between the activation-synthesis hypothesis and the clinico-anatomical hypothesis?

ANSWER

The activation-synthesis hypothesis and the clinico-anatomical hypothesis differ in their emphasis on the importance of the pons.
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