

## *Sleep*

*Sleep* may be defined as a recurring state characterized by (1) reduced awareness of and interaction with the external environment, (2) reduced motility and muscular activity, and (3) partial or complete cessation of voluntary behavior and self-consciousness (Anch, Browman, Mitler, & Walsh 1988). Several stages of sleep can be distinguished by their patterns of physiological correlates, though it is not possible to identify an exact moment of sleep onset in terms of physiological correlates (Dement 1976).

Sleep is the most important and unique of the altered states of consciousness, for several reasons. It involves dramatic changes in behavior and subjective experience, which are correlated with changes in physiological response patterns. Disruptions of the sleep-wake cycle are related to fatigue, performance problems, and physical and psychological disorders. And last but not least, dreams during sleep are some of the most dramatic, emotional, and bizarre experiences of our lives. Some psychologists believe that dreaming serves important psychological functions. Understanding the dreaming process is important for understanding how the human mind/brain system works.

This chapter will concentrate on sleep, with an emphasis on the different stages of sleep and their physiological correlates, the question of the function of sleep in general and its different stages in particular, and sleep disorders. Dreaming will be discussed in detail in the next three chapters.

## SLEEP-WAKE CYCLES

Under normal conditions, sleep occurs in a cyclic manner, termed a *circadian rhythm* (*circa*, about; *-dies*, day). Diurnal animals sleep at night and are awake and active during daylight hours, whereas the opposite is true of nocturnal animals. For both diurnal and nocturnal animals, the sleep-wake cycle is synchronized with the day-night cycle. As the day-night cycle changes with the seasons, the animals' sleep-wake cycles change also. In ancient times and in primitive societies, human sleep-wake cycles were synchronized with day-night cycles. In modern urban societies, however, the cycle is more likely to be synchronized with clock time, such that people stay up well past nightfall, and tend to wake up at about the same time each morning throughout the year, regardless of how light it is outside.

Synchronization with an external timer—either the day-night cycle or a clock—is not the only reason for sleep-wakefulness cycles. A biological clock, located in the brain, is also involved. The biological clock is *entrained* to an external timer (usually the day-night cycle), such that the internal clock tends to maintain the sleep-wakefulness cycle even when external time cues have been removed. Laboratory experiments with animals have shown that when they are kept in constant light or darkness they continue to have regular sleep-wake cycles, though the cycles tend to drift gradually out of phase with the outside day-night cycle. In natural settings, animals' internal clocks reset daily as the day-night cycle changes with the seasons, thus maintaining their clocks' entrainment to the day-night cycle.

Biological clocks have been demonstrated in humans, too, in experiments in which people lived alone for several months in caves or laboratory apartments where all cues to the day-night cycle and clock time were eliminated. The subjects could control the electric lights in their isolation apartment. They were instructed to sleep whenever they felt like doing so, but to try to avoid naps and limit themselves to one sleep period per day (based on their own estimate of a "day"). The result was that most subjects soon fell into a consistent sleep-waking cycle of about twenty-five hours. That is, they went to bed an hour later each day, so that if they had gone to bed at 11 p.m. on the first night, ten days later they were going to bed at 9 a.m. Also, average sleep time increased from eight hours to eight and a half hours (Coleman 1986). Under such conditions, various physiological cycles (including temperature and blood levels of various biochemicals) that are normally synchronized with each other and with the sleep-wake cycle become desynchronized (see Anch et al. 1988).

The internal clock is the reason for *jet lag*, the feelings of fatigue and malaise that occur when people travel across several time zones in a relatively short period of time. It takes a few days (up to two weeks) for the internal clock to become entrained to the day-night cycle or local clock time in the new time zone. A similar problem occurs when people have to work on night shifts, such as at a factory or hospital. Some researchers have speculated that it should be possible for people to make a good adjustment to night-shift work if they would stay on the same sleep-wake cycle (awake at night, sleep in the daytime) consistently for several weeks. But in fact, most data show that night-shift workers rarely make a complete adjustment to

night work. They sleep poorly in the daytime and are sleepy and fatigued at night. Their physiological cycles do not adjust to the night-work cycle. Probably one reason it is hard to make a complete adjustment to night work is that night workers usually revert to the normal day-wake, night-sleep cycle on weekends, so they can participate in normal social activities with friends and family. Also, work shifts are usually rotated every week or so. Thus, night workers never have a chance for their internal sleep-wake clocks to become entrained to the night-work cycle. This is a serious problem, since night-shift workers not only feel stressed and fatigued at night, but also have higher rates of accidents and health problems than day-shift workers (see Anch et al. 1988; Coleman 1986).

## STAGES OF SLEEP

Sleep is not a uniform state. There are several different types or stages of sleep that occur in a cyclic pattern during a sleep period.

The most important discovery in the history of sleep and dream research—the discovery of REM sleep—came about by accident (Aserinsky & Kleitman 1953).<sup>1</sup> In 1952 Dr. Nathaniel Kleitman at the University of Chicago became interested in the slow rolling eye movements that accompany sleep onset. Kleitman and his research assistant, physiology graduate student Eugene Aserinsky, decided to study these eye movements in a subject throughout a full night of sleep. Aserinsky's task was to monitor the eye movements as they were recorded on a polygraph. To his surprise, after the subject had been sleeping for awhile he started making relatively large, rapid, binocularly coordinated eye movements. These movements occurred periodically through the night, separated by intervals with slow rolling movements. This discovery was unexpected because it had been thought that sleep was a time of general depression or inhibition of the central nervous system—not the sort of state likely to produce rapid, coordinated eye movements. Meanwhile, medical student William C. Dement joined the research team, and started measuring brain waves and other physiological variables during sleep. It was soon noticed that during periods of rapid eye movements the brain waves showed a distinctive pattern, and there was a change in breathing (Dement 1976).

The rapid eye movements were particularly interesting and puzzling. What could be their cause? The researchers speculated that they might be related to dreaming. For example, perhaps they occurred as a result of sleepers "watching" movements in their dreams. They tested the hypothesis of a relationship to dreaming by systematically awakening ten subjects and interrogating them about their thoughts, sometimes during rapid eye movement (REM sleep) periods, and sometimes during periods of ocular quiescence (non-REM, or NREM sleep). The subjects reported dreams after almost all of the REM awakenings, but rarely after NREM awakenings. The relationship between REM sleep and dreaming was so strong that the authors concluded: "This method furnishes the means of determining the incidence and duration of periods of dreaming" (Aserinsky & Kleitman 1953, p. 274).

The discovery of REM sleep had several important results. It demon-

strated that sleep is not a uniform state, and further, that the distinctive subjective experience of dreaming occurs in conjunction with a distinctive physiological state. And most important, this discovery opened new worlds of possibilities for the experimental study of dreaming. It became possible to know when a sleeper is likely to be dreaming, and thus to collect dream reports immediately after awakening the subject, when dream recall is at its best. The discovery of REM inspired a surge of interest in laboratory research on the psychology and physiology of sleep and dreaming.

### Physiological Criteria for REM and NREM Sleep

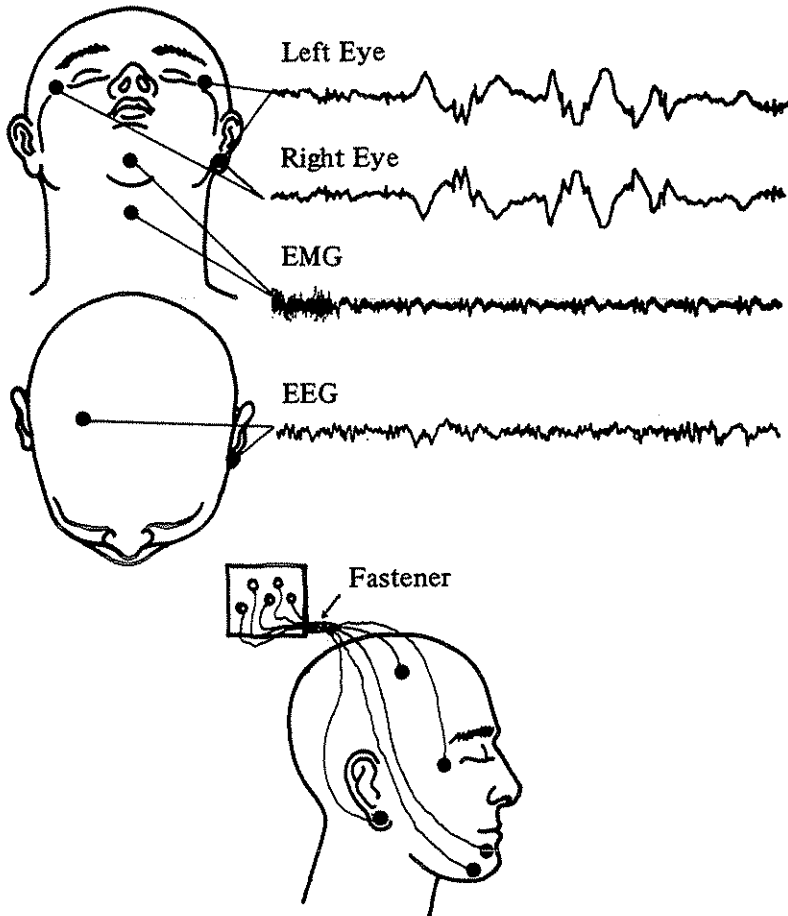
For most of our purposes it will be sufficient to distinguish between REM sleep versus NREM sleep though, as we will see in a later section, NREM sleep can be subdivided into four different stages. REM and NREM sleep are defined in terms of a systematic pattern of three physiological responses.

The three defining criteria include brain waves measured by the electroencephalogram (EEG), conjugate eye movements measured by the electrooculogram (EOG), and chin muscle tension measured by the electromyogram (EMG). These physiological responses involve small changes in the electrical potential of brain tissue and muscles. They are detected by means of small silver electrodes (less than 1 cm in diameter) that are taped to the subject's skin. (The electrodes are shaped like a shallow cup, and the cup is filled with an electrode jelly to ensure good contact with the skin.)

Figure 10.1 shows standard locations for EEG, EOG, and EMG electrodes in sleep research. The earlobe electrode is a reference point for the EEG and EOG voltage measurements. In some experiments additional physiological measurements are taken, such as measures of breathing, heartbeat, body temperature, or penile tumescence (erection). Also, EEG measures may be taken from several different scalp locations, and additional EOG electrodes may be used to record vertical eye movements (the standard arrangement distinguishes only lateral eye movements). Wires from the electrodes are collected in a "pony tail" and attached to a jackbox on the wall. On the other side of the wall, in an adjacent room, wires from the jackbox are connected to a machine called a *polygraph* or *polysomnograph*. The polygraph amplifies the voltage fluctuations and records them on moving chart paper (and also in computer data files, in modern research).

Figure 10.1 also shows polygraph recordings of a transition from NREM to REM sleep. During NREM sleep the eyes are not completely still, but they tend to drift slowly back and forth. At the transition from NREM to REM sleep, the EOG lines show a change from relatively quiescent eyes to frequent, relatively large conjugate lateral eye movements (REMs). ("Conjugate" means that the eyes move together.) The fact that the EOG tracings move in opposite directions has to do with the fact that the electrical polarity of the electrode depends upon whether the eyes are moving toward it or away from it. Thus, if the eyes move to the right, then the right electrode becomes more positive and the left more negative, and vice versa.

REM sleep gets its name from the rapid eye movements that are characteristic of this stage of sleep, and which do not occur in other stages of sleep. However, while rapid eye movements are the distinctive behavioral charac-



**FIGURE 10.1.** Standard electrode placements for sleep recordings. This is the minimum setup required for identifying different stages of sleep. The polygraph tracings show the transition from NREM to REM sleep. The tracings move horizontally over time; thus, they are read from left (earlier time) to right (later time). Greater response amplitudes (in microvolts) are shown by greater ranges of vertical movement of the tracings. The transition from NREM to REM sleep is indicated by three physiological events: (1) EEG, an increase in brain wave activity; (2) EOG, an increase in eye movements; and (3) EMG, a decrease in chin muscle tension. The standard chart speed is 10 mm per second, so a full 8 hours of sleep requires 288 meters (944 feet) of chart paper. [From Dement, W. C. (1976). *Some Must Watch While Some Must Sleep*. New York: Norton. Copyright by William C. Dement and the Stanford Alumni Association.]

teristic of REM period sleep, the eyes do not necessarily move continuously throughout a REM period. There are often intervals of several seconds (and occasionally up to a minute or more) without eye movements during a REM period.

The EMG tracing shows that in the transition from NREM to REM sleep

the chin muscles relax further, that is, there is a decreased magnitude and rate of voltage oscillations in the EMG.

The EEG is a rather crude measure, since it is the average electrical voltage of thousands of neurons located on the surface of the cortex several millimeters below the scalp electrode. Nonetheless, the EEG is sensitive enough to distinguish different brain wave patterns in wakefulness and sleep. In the transition from NREM to REM the EEG shows an increase in brain wave activity, that is, an increased frequency of voltage oscillations, along with a decrease in mean brain wave amplitude. (The difference may be seen more clearly in Figure 10.2.) In general, EEG frequency and amplitude are inversely correlated, such that slower frequency goes with increased amplitude (especially in deep NREM sleep stages, Figure 10.2). Slower-frequency/higher-amplitude tracings are associated with an increase in the synchronization of firings of cortical neurons under the scalp electrode.

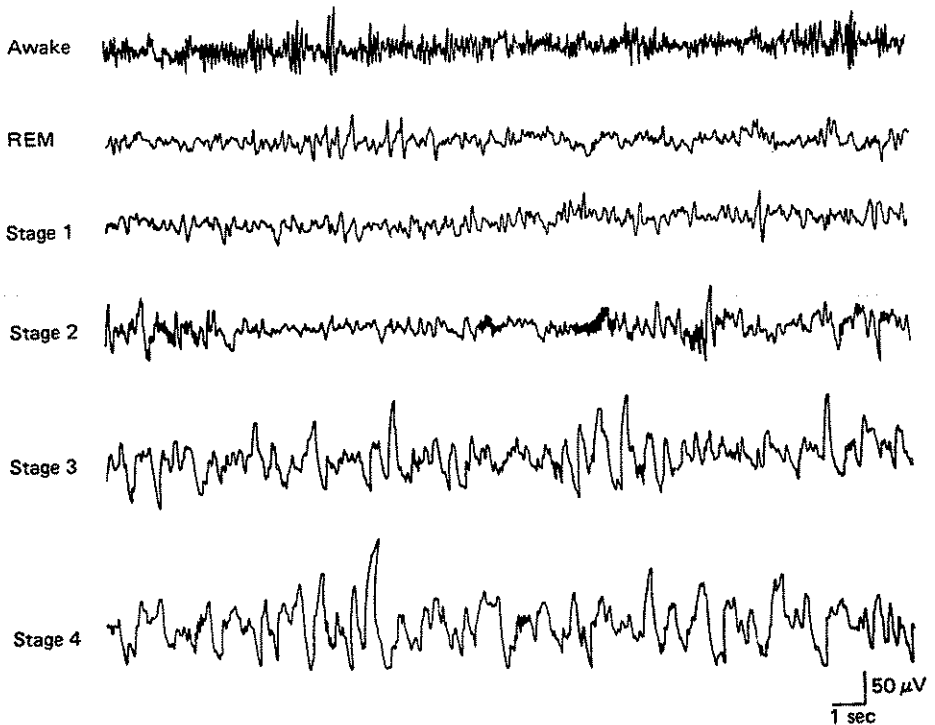
It should be emphasized that REM sleep is defined by the *pattern* of three physiological correlates, rather than by only one of them. Table 10.1 summarizes the patterns of psychophysiological correlates for waking and for REM and NREM sleep. (In the table the + and - symbols indicate relatively more or relatively less, respectively.) You can see that no single indicator is sufficient to distinguish between all three of these major states of consciousness.<sup>2</sup>

### The Structure of Sleep

**Sleep stages.** For a more complete description of sleep stages we need to distinguish between four NREM stages. Figure 10.2 shows typical EEG tracings for relaxed wakefulness, NREM stages 1, 2, 3, and 4, and REM sleep. In relaxed wakefulness, with the eyes closed, the EEG tracing shows a predominance of *alpha waves*, fairly synchronous brain waves occurring at a rate of about ten cycles per second (8–13 Hz). The transition from wakefulness to sleep is shown in Figure 10.6.

When we fall asleep we first enter NREM Stage 1, and then progress successively through NREM stages 2, 3, and 4, then back up through stages 3 and 2 to REM sleep. NREM Stage 1 is characterized by relatively low-amplitude, fast waves of mixed frequencies. In Stage 2 the mean brain wave frequency is somewhat slower, and mean amplitude somewhat greater, than in Stage 1. Also, in Stage 2 distinctive sleep spindles and K-complexes appear in the EEG (see Figure 10.2). The characteristic feature of Stage 3 is the emergence of rhythmic but very slow (0.5 to 2 Hz), high-voltage delta waves. Stage 4 is defined as the occurrence of delta waves that exceed 75 microvolts in amplitude in over 50 percent of the EEG tracing. Thus, Stages 2 and 4 are distinctive, while Stage 3 (about 20–50 percent delta waves) is a transition stage between Stages 2 and 4.

The EEG pattern during REM sleep is superficially similar to that during Stage 1 NREM in that both involve predominantly low-voltage, high-frequency, desynchronized waves of mixed frequencies (including occasional alpha). However, a more fine-grained analysis shows that REM EEG also includes distinctive “saw-tooth” waves of moderate amplitude and



**FIGURE 10.2.** EEG in different stages of sleep. The recordings were made at the standard chart speed of 10 mm per second, with a 10 mm vertical deflection of the pen representing a 50 microvolt change in electrical potential. The first line was recorded during relaxed wakefulness, and shows mostly alpha waves of about 10 cycles per second (Hz). Going from NREM Stage 1 to NREM Stage 4, the general trend is toward slower and higher amplitude waves. The Stage 1 NREM tracing shows low amplitude, fast waves of mixed frequencies. The Stage 2 NREM tracing shows two features characteristic of that stage: several *sleep spindles*, which are the bursts of 12–14 Hz waves lasting about a second and appearing against a background of generally slower waves; and a *K complex*, which is the isolated high voltage spike in the right half of the recording. In Stage 3 NREM very slow ( $\frac{1}{2}$  to 2 Hz), high-amplitude delta waves begin to appear. The stage is identified as Stage 4 NREM when over 50 percent of the tracing consists of delta waves exceeding 75 microvolts in amplitude. The REM tracing is largely similar to NREM stage 1, except for the occurrence of bursts of fairly uniform “saw-tooth” waves in REM stage (Anch et al. 1988; Dement 1976.) [From Rechtschaffen, A. & Kales, A. (1968). *A Manual of Standardized Terminology, Techniques and Scoring System for Sleep Stages of Human Sleep Subjects*. Washington, DC: U.S. Government Printing Office.]

about 2–3 Hz. Saw-tooth waves typically occur in bursts of two or three at a time, and often precede or coincide with rapid eye movements.

**REM cycles.** During the course of a night’s sleep of seven or eight hours, normal adults go through several cycles of NREM and REM sleep. Figure 10.3 shows a typical sequence of stages. This figure illustrates several points about a night’s sleep for young adults: (1) The first REM period does

**TABLE 10.1 Physiological Correlates of Waking, REM Sleep, and Non-REM Sleep States\***

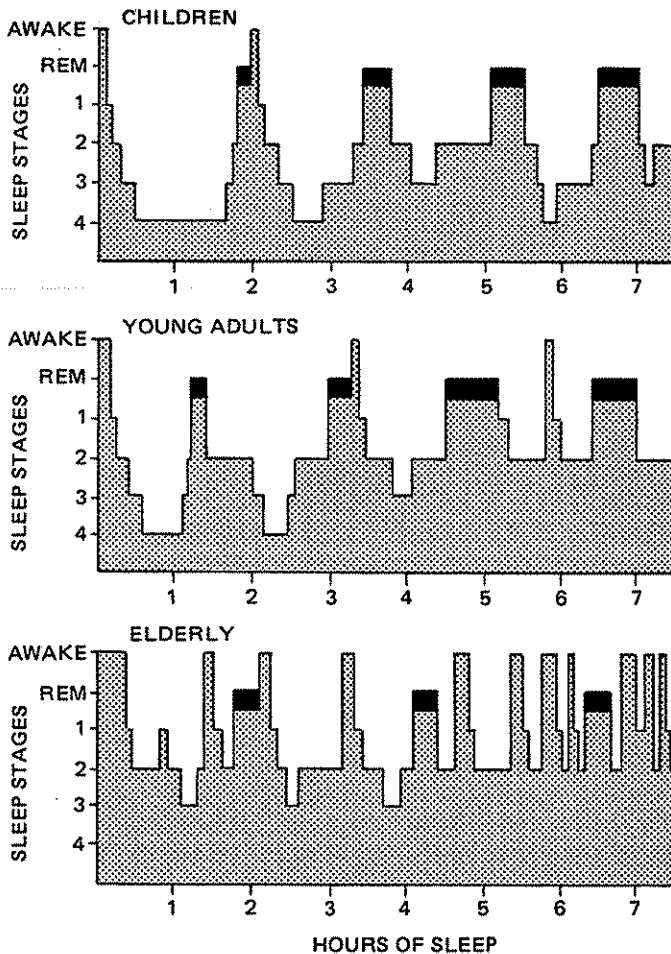
	DEFINING FEATURES			OTHER FEATURES		
	EEG	EOG	EMG	Extero-ception	Vestibular System Activation	Autonomic Arousal
	Brain Waves	Eye Move-ments	Chin Muscle Tension			
<i>State</i>						
Waking	Fast, low amplitude	+	+	+	+	+
REM Sleep	Fast, low amplitude	+	-	-	+	+
NREM Sleep	Slow, high amplitude	-	+	-	-	-

\*Within each response system the "+" and "-" symbols indicate the relative amounts of activity in the different states. The pattern of EEG, EOG, and EMG responses taken together are the defining physiological features by which the relaxed waking state, REM sleep, and non-REM (NREM) sleep are identified. Exteroception, vestibular activation, and autonomic arousal are other state correlates, though they are not used as defining features.

not occur until over an hour after sleep onset. (2) The duration of the first REM period is typically rather brief (5 to 10 minutes), but REM duration tends to increase in later cycles, averaging about 22 minutes in later cycles (though with considerable variability about the mean). (3) The first sleep cycle is defined as the time from sleep onset until the end of the first REM period. Subsequent cycles are defined as the time from the end of one REM period until the end of the next REM period. After the first cycle, subsequent cycles average about 90 to 100 minutes in length. (4) NREM Stages 3 and 4 are confined almost entirely to the first four hours of sleep; after that NREM time is almost entirely devoted to Stage 2. (5) Toward the end of the night it is common to awaken briefly from either NREM or REM sleep (Anch et al. 1988; Dement 1976).

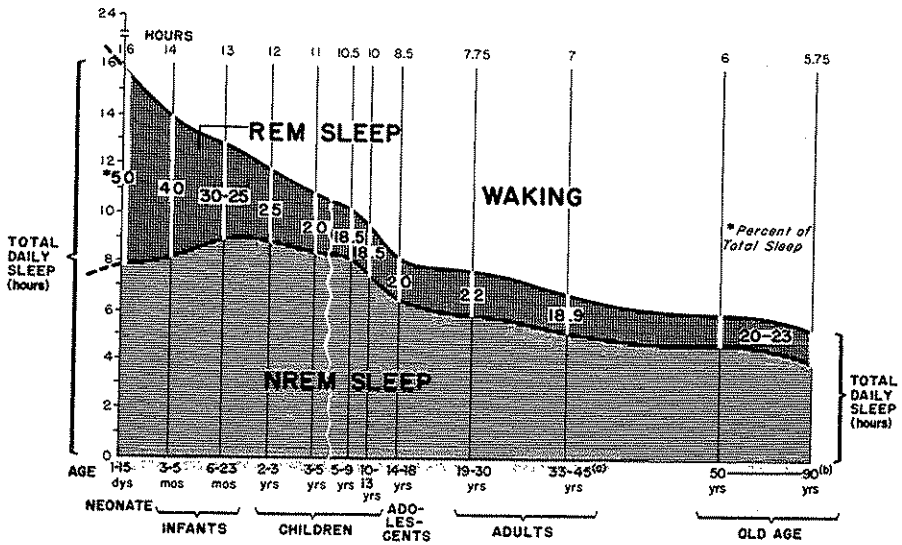
**Infants and animals.** Newborn human infants sleep about 16 to 18 hours per day, and spend at least 50 percent of this time in REM sleep. (Premature infants may spend as much as 75 percent of their sleep time in REM.) Figure 10.4 shows that as people mature the percent of sleep-time spent in REM decreases gradually to about 25% at age 3, to 20% in adolescents and adults (Roffwarg, Muzio, & Dement 1966). Nor is REM sleep unique to humans. It occurs in all mammals (except the echidna), but not in reptiles, and probably not in birds (Hartmann 1967). Much of what we know about the brain mechanisms involved in sleep comes from research on cats, whose sleep stages are quite similar to those of humans except that the mean cycle





**FIGURE 10.3.** Cycles of sleep stages in a typical night of sleep for three age groups. In the young adult the first REM period does not begin until about an hour after sleep onset. After that, REM periods begin about every 90 to 100 minutes, on the average. Going from child to young adult to elderly, the amount of NREM Stage 4 decreases, while the frequency of brief awakenings increases. [From Kales, A. & Kales, J. D. (1974). *Sleep disorders: Recent findings in the diagnosis and treatment of disturbed sleep. New England Journal of Medicine*, 290, 487-99. By permission of the publisher.]

length for cats is only about 40 minutes, with REM periods of 5 to 10 minutes duration (Hobson & McCarley 1977). Adult cats spend about 17 percent of their sleep in REM state, but in newborn kittens nearly 100 percent of sleep is REM sleep. Whether nonhuman mammals dream is unknown, since they have no way of telling us.



**FIGURE 10.4.** Changes in amounts of REM and NREM sleep with increasing age. Total sleep time decreases from infancy to old age. From infancy to about 10–13 years the decrease is in the amount of REM sleep. From 10–13 years onward the amount of REM stays fairly constant (about 1½ hours), whereas the amount of NREM continues to decrease. The percent of sleep time spent in REM stage is also indicated; it decreases from about 50 percent for infants to about 20 percent for adolescents and adults. [From Roffwarg, H. P., Muzio, J. N., & Dement, W. C. (1966). Ontogenetic development of the human sleep-dream cycle. *Science*, 152, 604–19. Copyright 1966 by the AAAS. Revised by the authors since original publication.]

### Other Physiological and Behavioral Correlates of Sleep Stages

Besides the three defining criteria of REM versus NREM sleep—EEG, EOG, and EMG—there are several other physiological and behavioral correlates of sleep stages (Anch et al. 1988; Rechtschaffen 1973).

**Exteroception.** It is impossible to determine the exact instant of sleep onset solely by means of an EEG recording. According to Dement, “The essential difference between wakefulness and sleep is the loss of awareness. Sleep onset occurs at the exact instant when a meaningful stimulus fails to elicit its accustomed response” (1976, p. 27). For example, if you speak a person’s name softly, then he or she would normally respond when awake, but not when asleep. Of course, if you shout the name, the sleeper will awaken. *Auditory arousal thresholds* are higher during sleep than during waking, and they are higher during Stages 3 and 4 than during other stages of sleep (Zimmerman 1970). (A higher arousal threshold means that it takes a louder noise to awaken the sleeper.)

Other sensory thresholds, such as smell and touch, are also higher during sleep than waking. Dement (1976) described a dramatic example of the sudden loss of visual responsiveness during sleep onset:

A sleepy subject lies in bed with his eyes taped open (which can be achieved, believe it or not, with relatively little discomfort). A very bright strobe light is placed about six inches in front of his face and is flashed into his eyes at the rate of about once every second or two. A microswitch is taped to his finger, which he is instructed to press every time he sees a flash. A simple task. How can he possibly avoid seeing the flash? The subject will press and press. Suddenly he stops. If we immediately ask why, he will be surprised. The light exploded right into his widely open eyes, yet he was totally unaware. In one second, he was awake, seeing, hearing, responding—in the very next second, he was functionally blind and asleep (pp. 27–28).

Though sensory thresholds are reduced during sleep, the sleeping brain can still detect stimuli, as is shown by the fact that stimuli during sleep can elicit physiological responses, such as evoked potentials in brain waves and heart rate changes. Meaningful stimuli (such as emotionally significant names) elicit different physiological reactions than meaningless stimuli (see McDonald et al. 1975). The arousal threshold is lower for a meaningful stimulus, such as a baby's cry, than it is for meaningless stimuli. Also meaningful stimuli during sleep are more likely than meaningless stimuli to affect the contents of dreams (see Chapter 11). All of these findings show that the sleeping brain is capable of discriminating between different external stimuli, even though the sleeping person is not consciously aware of the stimuli. Sensory discrimination ability is typically better in REM than Stage 2 NREM, and worst in Stage 4. (See reviews in Anch et al. 1988; Arkin and Antrobus 1978.) However, carefully controlled studies have generally failed to show that people can learn verbal materials, such as foreign language vocabularies, during either REM or other sleep stages. (See review by Aarons 1976.)

**Muscle tension and movement.** In general, the muscles become more relaxed, and spinal reflexes (such as knee jerk) are reduced, in the transition from waking to NREM sleep. Muscle tension throughout the body is usually lower during REM than NREM sleep, and reduced chin EMG is one of the defining features of REM sleep. Paradoxically, aperiodic twitches of face and finger muscles occur more often during REM than in deeper sleep. Also the frequency of body movements is higher during REM than NREM sleep, with the rate of body movements progressively decreasing from waking to NREM Stage 1, REM, and NREM Stages 2, 3, and 4, respectively (Wilde-Frenz & Schulz 1983).<sup>5</sup> Thus, while reduced muscle tension is a *tonic* (long term, sustained) characteristic of REM sleep, muscle twitches and body movements can occur as *phasic* (brief, sporadic) events (Pivik 1978). The EEG often suggests a brief awakening during the body movements, although the individual is typically unresponsive at that time and does not remember the movement (Dement 1976). Sleepwalking (somnambulism)—which occurs during Stage 4—is considered to be a sleep disorder. (I will discuss sleepwalking further in the section on sleep disorders.)

**Vestibular system activation.** The vestibular system is responsible for our *equilibratory sense*, which includes sensing the orientation of our head and maintaining our balance, and sensing the acceleration of our body through space. This is accomplished by the vestibular apparatus, which is located in

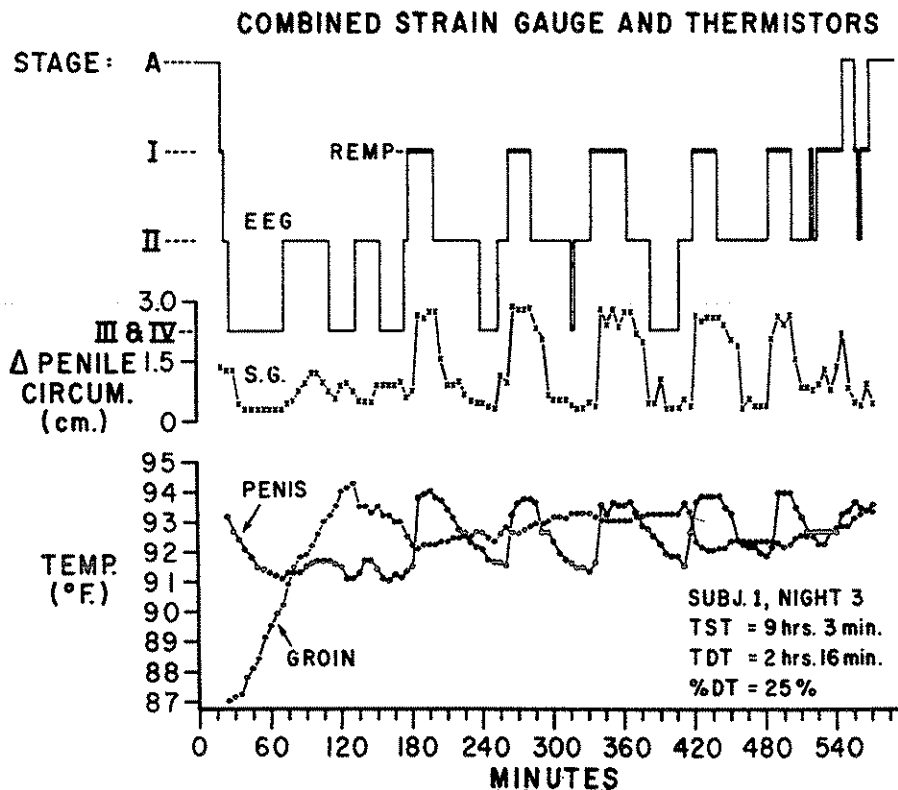
the inner ear, next to the cochlea, on each side of the head. The motion of fluid in the apparatus bends hair cells, which send a unique pattern of neural activity to the brain to indicate head position and movement. During REM sleep, neural activity in the vestibular system increases, even though the sleeper is perfectly still. As we will see in Chapter 12 on theories of dreaming, Hobson and McCarley (1977) have speculated that this spontaneous vestibular activity during REM sleep can account for the sensation of movement during dreams.

**Autonomic responses.** Heart rate, respiration rate, and systolic blood pressure decrease during the transition from waking to NREM sleep. However, these responses increase somewhat—about 6 percent—in REM compared to NREM sleep (Snyder et al. 1964). More dramatic is the increase in *variability* of these measures, about 55 percent. In other words, there are more momentary, phasic changes in autonomic responses during REM than during NREM sleep.

Heart rate, respiration rate, and blood pressure are controlled by the autonomic nervous system, and increases in these responses—such as during emotional arousal—indicate increased activation of the sympathetic part of the autonomic nervous system. It has been speculated that there is an association between autonomic arousal in REM and the emotionality of dreams. However, the situation is made more complicated when we consider the Galvanic skin response (GSR). The GSR (measured from the palm or fingers) indicates decreased electrical resistance in the skin (or conversely, increased electrical conductivity), related to increased neural activity in the sweat glands. The GSR is a component of the orienting reaction to novel or emotionally significant stimuli, and it is a standard measure on “lie-detector” tests. If REM is a more autonomically aroused state than NREM, then we would expect more GSRs in REM than NREM. In fact, the opposite is the case. Spontaneous GSRs occur so often during NREM Stages 3 and 4, and with such vigor, that they have been called “GSR storms” (Rechtschaffen 1973).

The reasons for the variability in heart rate and respiratory rate during REM, and for the paradoxical prevalence of GSRs in Stages 3 and 4 rather than REM, are not clearly understood. However, the data suggest that, contrary to an earlier hypothesis, it would be incorrect to characterize REM sleep as a state of tonically increased sympathetic arousal (Rechtschaffen 1973). Furthermore, autonomic responses, such as GSR and heart rate, are not necessarily reliable correlates of emotional dream events (Pivik 1978).

**Penile erections.** Throbbing penile erections occur during REM sleep in normal males, including infants and children as well as adults (see Figure 10.5). Fisher, Gross, and Zuch (1965) measured erections during uninterrupted sleep in seventeen normal adult males, using several methods including a strain gauge (which measures changes in diameter), phalloglethysmograph (which measures surface blood flow), and direct observation. Other studies have used a sort of pneumatic donut to measure erections. Fisher et al. found that 60 percent of the REM periods were accompanied by full erections, 35 percent by partial or fluctuating erections, and only 5 percent by no



**FIGURE 10.5.** Penile erection cycle during sleep, for a representative subject. The upper graph shows changes in EEG sleep stages; REM (REMP) stage is indicated by the darker horizontal lines. The middle graph shows penis circumference as measured by a strain gauge; increases of 2.5 cm or more indicate full erection. Note that erections occur during REM periods. The bottom graph shows groin and penis temperatures. [From Fisher, G., Gross, J. & Zuch, J. (1965). Cycle of penile erections synchronous with dreaming (REM) sleep: Preliminary report. *Archives of General Psychiatry*, 12, 29-45. Copyright 1965, American Medical Association.]

erection. Erections tended to begin about the same time as REM, though sometimes they began a few minutes before REM onset. Maximum erection was usually attained about 5 minutes after REM onset, and once the maximum was attained it tended to be maintained throughout the REM period. The erections were maintained for a while after the end of the REM period, with full detumescence not occurring until 12 minutes after the end of REM, on the average. Thus, the common experience among males of waking up with an erection is due to the persistence of the erection from the last REM period before awakening. Ejaculation of seminal fluid ("wet dreams") sometimes occurs during REM erections.

Fisher et al. (1965) found no evidence of erections during NREM sleep, except for erections sometimes beginning in Stage 2 shortly before REM onset. Also, they found that REM erections occurred independently of the subject's recency of sexual gratification. Other studies have shown that REM

erections are not necessarily associated with sexy REM dreams (Karacan et al. 1966). Thus, REM erections are elicited by the physiological events of REM sleep, rather than by REM dream images. REM erections are so commonplace for normal males that they can be used to aid in diagnosing the cause of chronic impotency (the persistent failure to get an erection when it is desired for sexual intercourse). Absence of REM erections would indicate that the impotency problem has some organic basis. On the other hand, if the patient gets erections during REM, then his failure to get them during sexual play probably has a psychological basis.

Genital arousal occurs during REM sleep in women, too, though not in such an obvious way as in men. Research with electrophysiological recording devices has shown that during REM women have increased vaginal blood flow, and erections of the clitoris, both responses being typical of sexual arousal.

**Other measures.** Cortical blood flow and temperature increase during REM, as one might expect from the increased cortical activation during REM. And while the cortex may, in a sense, be more quiet during NREM than REM, the sleeper may be noisier during NREM: most snoring occurs during NREM sleep (Dement 1976). (See Anch et al. 1988, for more details and reference citations on all varieties of physiological responses during sleep.)

### The Physiological Basis of Sleep Cycles

Two types of sleep cycles are of interest: the cycle of sleep versus wakefulness, and the REM-NREM cycle within sleep periods. Both types of cycles are produced by biological clocks in the brain. Neurophysiologists have used a variety of experimental procedures with animals (such as rats, cats, and monkeys) to discover the location of the biological clock or clocks. The procedures include ablation (destroying a localized part of the brain), disconnection (cutting the connecting neurons between two parts of the brain), electrical stimulation of neurons, and measurement of spontaneous electrical activity in localized cell groups during sleep. More recently, identification of specific neurotransmitter chemicals in different brain circuits, and stimulation of synapses by different chemicals, have also been employed. For obvious reasons, it has not been possible to do most of these experimental procedures with humans. However, the similarity of sleep in other mammals is sufficiently similar to that of humans to make it seem likely that the findings from animal research are also applicable to humans.

**Sleep-wakefulness cycle.** Recent research suggests that the sleep-wakefulness cycle is controlled primarily by a biological clock located in the *suprachiasmatic nucleus* (SCN) of the hypothalamus (located just above the optic chiasm, where the optic nerves divide; see Figure 5.1). The SCN clock is normally entrained to the day-night cycle, though the clock continues to operate in constant light or darkness, with a tendency to drift toward a somewhat shorter or longer cycle (depending on the species). The SCN gets its own visual input from the eyes, as is shown by the fact that brain lesions or

disconnections posterior to the chiasm, which produce blindness, do not prevent entrainment to light-dark cycles (Anch et al. 1988).

**REM-NREM cycle.** Neural circuits in the *pontine reticular formation*, located in the pons section of the brain stem, are particularly important for controlling the REM-NREM cycle within sleep periods. Earlier it was proposed that the clock is located in the pontine reticular formation (Hobson & McCarley 1977). More recent evidence suggests that the clock is a widely distributed network of cells with reciprocal excitatory and inhibitory interactions, though the pontine reticular system is an important part of this network (Hobson 1988; Hobson, Lydic, & Baghdoyan 1986).

Hobson and McCarley (1977) concluded that the reticular system is responsible for directly eliciting several of the physiological features of REM sleep, including: (1) Tonic activation of the cortex, which produces the high level of brain-wave activity characteristic of REM sleep. (2) Inhibition of spinal cord motoneurons, which blocks muscle movements despite high levels of activity in the motor cortex. (Phasic muscle movements seem to involve occasional “breakthrough” of excitation against the inhibitory processes.) (3) Phasic activation of the oculomotor system to produce eye movements. (4) Phasic activation of the vestibular system. (In addition to possibly producing spurious sensations of movement, the vestibular activation also causes some of the eye movements. This occurs because one of the normal functions of the vestibular system is to coordinate eye movements with head movements.) (5) Blockade of exteroceptive sensory inputs to the cortex.

An implication of the evidence that the physiological correlates of REM sleep are directly elicited by a brain-stem mechanism is that there is no need to try to explain them as responses to dream images. (In Chapter 12 on theories of dreaming I will discuss Hobson and McCarley’s provocative idea that the opposite is the case: their Activation-Synthesis Hypothesis proposes that dreams are the brain’s response to the physiological events of REM sleep.)

## THE FUNCTION OF SLEEP

It seems obvious that the sleep-wake cycle must have evolved because sleep has some sort of adaptive function. Presumably, we need to spend a certain amount of time sleeping each day. But why? A negative answer—that we feel crummy if we don’t sleep—is not sufficient. We need a positive answer to the question: What is the function—or the adaptive value—of sleep? Surprisingly, despite decades of research on sleep, scientists cannot give a definite answer to this fundamental question.

The most obvious explanation of the need for sleep is that it serves some *restorative function*. Presumably, the body needs to rest each day in order to allow a period of time for physiological growth and repair functions to operate. But if rest for restoration were the only reason for sleep, then it should be sufficient to spend eight hours per day in physical relaxation, without actually sleeping. Yet, it is almost impossible for people to remain awake overnight while physically resting. It is easier to stay awake if we are

engaged in some sort of physical activity. Clearly, physical rest is no substitute for sleep. Yet there is no clear evidence that sleep is essential for any particular physiological restorative function.

But sleep is more than a physiological state. It is also a kind of behavior. Taking a behavioral viewpoint, and looking at humans and other animals in the wild state, we could define sleep as the behavior of lying relatively still and temporarily suspending reactivity to the environment.

According to the *adaptive behavior hypothesis*, sleep has a clear adaptive value. For primates and other diurnal (active in daytime) animals, nighttime sleep ensures that they are quiet and inconspicuous at night, when nocturnal predators are out hunting for them. Presumably, prey animals with a tendency to sleep at night (in a safe place, such as a burrow, tree, or cave) are more likely to survive and leave offspring than those that are awake and active at night. Also, sleep is an *instinctive behavior*, according to these criteria: (1) it has a basis in heredity; (2) it is developmental in nature; (3) there is a positive motivation to do it; and (4) it shows regularities in response to the environment while maintaining some degree of flexibility. From the viewpoint of the adaptive behavior hypothesis, behavior is primary, and physiology is secondary, in the evolution of sleep. That is, the physiological sleep state evolved because it controls the behavior of sleep (Webb 1975).

Several comments on the adaptive behavior hypothesis are in order. First, while it seems appropriate to primates and other diurnal animals that tend to sleep through the night, it may not be applicable to animals with other sleep patterns (Allison & Cicchetti 1976; Zepelin & Rechtschaffen 1974). For example, cats tend to sleep at two- or three-hour intervals through the day and night, with a circadian rhythm that is much weaker than that of primates. Second, the adaptive behavior hypothesis does not explain the fact that we have several physiologically distinct types of sleep, including REM and the several NREM stages. The adaptive behavior function of sleep would be adequately served by a single physiological sleep state. Third, it cannot explain the devastating stressful effects of chronic sleep deprivation. For example, rats totally deprived of sleep invariably die within ten to thirty days (Rechtschaffen et al. 1983). Thus, the adaptive behavior hypothesis is not a complete explanation of sleep-wake cycles and the need for sleep.

Possibly, sleep originally evolved as an adaptive behavior. Subsequently, REM and NREM sleep were differentiated out of an original unified sleep state, in order to serve different functions (Cohen 1979a). In a later section I will turn to the question of whether REM sleep has any unique function, distinct from that of NREM sleep. But first, let us consider the question of what happens when humans are deprived of sleep.

## SLEEP DEPRIVATION AND HUMAN PERFORMANCE

In view of the fatigue and relatively high accident rates experienced by night workers, ranging from factory workers to truck drivers and airline pilots, it is important to ask what effects sleep deprivation has on human performance. There have been several studies on this question, but it is impossible to make generalizations that apply to all cases because the effects of sleep deprivation



on task performance depend on several factors (Webb 1975): (1) The nature of the task: harmful effects of sleep deprivation are generally greater for intellectual tasks than for sensory-motor tasks, for complex tasks than for simple tasks, and for tasks requiring flexible thinking and variable responses than for routine, habitual tasks. (2) The individual worker's ability to do the task efficiently under stress; typically, this ability will be greater the more the worker has practiced the task. (3) The worker's motivation or desire to continue trying to perform well when fatigued: by exerting extra effort the worker may be able to compensate for reductions in performance capacity, at least temporarily.

The great paradox of sleep deprivation research is that while everyone "knows" that sleep deprivation hurts performance, in practice it has been difficult to demonstrate dramatic effects in controlled studies with objective measurements. With only a single night of total sleep deprivation, behavioral and performance effects are usually negligible. The only clear effect is persistent daytime sleepiness, which is objectively demonstrated by reduced sleep latency (Coleman 1986).

**Total sleep deprivation.** Total sleep deprivation for four or five days causes modest physiological effects: hand tremor, occasional double vision, droopy eyelids, a lower pain threshold, and a reduction in EEG alpha. Performance on short-term tasks (a few seconds to a minute or so) is usually not affected, as long as the tasks are relatively simple or well-learned and emphasize visual-motor response coordination. Performance on complex reaction-time tasks (involving multiple stimuli and responses) and short-term memory tasks are more likely to be affected. Most affected is performance on tasks that must be sustained over a relatively long period of time, say thirty minutes or so. This is particularly true for boring tasks for which subjects are relatively unmotivated, such as vigilance tasks (signal-detection tasks requiring subjects to react to sensory signals presented at irregular intervals). Behaviorally, there are occasional and transient instances of confusion, disorientation, and irritability, but the personality is generally intact. Surprisingly, both appetite and sexual drive may increase. Truly bizarre behaviors are quite rare, though hallucinations and paranoia may occur with extended sleep deprivation (Webb 1975).

Laboratory studies may reveal the limits of human performance capabilities under sleep deprivation, but they do not necessarily predict what will happen in particular cases outside of the laboratory, such as at work. In the laboratory, when sleepy subjects are being watched, they may be motivated to muster their reserves and perform to the best of their abilities, and so they can perform successfully on short-term tasks. But outside of the laboratory, when people are fatigued and concentration requires great effort, they may choose to postpone a task or avoid it altogether. Thus, the total work output could suffer, even though what is done will usually be done satisfactorily. For example, sleepy power-plant operators may be perfectly capable of checking pressure gauges correctly, but if they are sleepy they may omit one or more of the scheduled checks. On the other hand, the capacity of sleepy people to do intellectual tasks, such as reading or listening to lectures, may be strongly

disrupted, due at least partly to the difficulty of maintaining attention to the task at hand.

It is not necessary to make up for all of the lost sleep after a period of total sleep deprivation. After only a single night of total sleep deprivation, though mean sleep onset latency is reduced from ten minutes to about one minute, there is complete recovery to normal functioning after two successive nights of normal eight-hour sleep periods. Thus, while a night of prolonged sleep leads to faster recovery, it is not strictly necessary for recovery (Coleman 1986). Peter Tripp, a disk jockey, stayed awake for two hundred hours in a booth in Times Square, New York. Though he suffered hallucinations and paranoia, he returned to normal functioning after only thirteen hours of sleep, with no long-term effects. (EEG monitoring showed that Tripp experienced numerous "microsleeps" of two or three seconds during his ordeal. Microsleeps make it impossible to totally deprive someone of sleep for a prolonged period, though it is not clear to what degree microsleeps can substitute for normal sleep.)

Alertness and performance during sleep deprivation are affected by the circadian phase. In subjects sleep-deprived for several days, sleepiness and performance decrements are worst during the hours when the subjects would normally be sleeping. The low point is between 3 and 4 a.m. (Coleman 1986). (I can personally attest to this from two all-night vigils at the hospital maternity ward, keeping my wife company during labor. I felt particularly sleepy between 3 and 4 a.m., but by 6 a.m.—near my normal waking-up time—I felt much more alert.) The time of maximum sleepiness, 3 to 4 a.m., is also the time when body temperature is lowest during a normal 24-hour period with daytime wakefulness and nighttime sleep. Body temperature, sleepiness, and performance cycles are part of the same endogenously controlled circadian rhythm.

**Partial sleep deprivation.** The effects of short-term *partial* sleep deprivation—for example, going with four or five hours of sleep per night for one night, or a few nights—are relatively minor (Coleman 1986; Anch et al. 1988). The main effect is sleepiness. Performance on short-term tasks is unaffected, though people may do worse on boring, routine tasks. People can adapt to partial sleep deprivation. In one study sleep was reduced from 8 hours to 5½ hours per night for 60 days (Webb and Agnew 1974). On a signal-detection task requiring the subjects to maintain vigilance, they responded less frequently, indicating that they were less motivated to maintain attention to the task. Other cognitive tasks were unaffected. Most subjects reported daytime drowsiness during the first week, but by the eighth week they had adapted and no longer suffered from drowsiness. Yet, when the study was over, virtually all of the subjects returned to their customary 8 hours of sleep, because they just "felt better" with more sleep.

**Conclusion.** We can adapt—in task performance and feeling of alertness—to chronic (long-term) *partial* sleep deprivation. As for *total* sleep deprivation, all of the studies have used acute (short-term) deprivation, limited to four or five days (with one volunteer going ten days without sleep). The

effects of acute total deprivation are relatively small, other than the overwhelming sleepiness, and complete recovery occurs quickly, with only one or two nights of extended sleep.

But what about the effects of chronic total sleep deprivation in humans? The question seems too dangerous to ask. In some experiments done in Italy in the 1890s, dogs were kept awake as long as twenty-one days. Some of them died before they got that far. In more recent experiments with rats, the animals died after ten to thirty days of sleep deprivation (Rechtschaffen et al. 1983). Prolonged total sleep deprivation is very stressful, too stressful to test in humans.

## THE FUNCTION OF REM SLEEP

The discovery of different physiological sleep stages led to the question of whether the different stages have special functions. REM sleep is of particular interest, because of its association with dreaming and the paradoxical pattern of physiological responses that are similar to the waking state while the person is behaviorally asleep. Researchers have attempted to discover the function of REM sleep by selectively depriving subjects of REM sleep, while allowing them to sleep undisturbed in NREM stages.

**The dreaming hypothesis.** One hypothesis about the function of REM sleep is that its main purpose is to enable people to dream. This idea followed from the early observation of a strong correlation between REM sleep and dreaming, and from Freud's idea that dreaming serves a valuable psychological function of allowing harmless expression of powerful instinctive drives. Thus, some of the earliest research on REM deprivation was conceived as a study of the effects of dream deprivation (Dement 1960). However, we now know that it is misleading to assume that REM deprivation is equivalent to dream deprivation, for two reasons: (1) Dreaming occurs during NREM sleep, as well as during REM sleep (more on this in the next chapter). Thus, selectively depriving people of REM sleep does not necessarily deprive them of all dreaming. (2) REM sleep is a complex physiological state, and it is unparsimonious to assume that effects of REM sleep deprivation are a result of dream deprivation. Physiological or psychological effects of REM deprivation are more likely to be due to disruption of physiological processes that are unique to REM sleep. With these caveats in mind, let us consider the effects of REM sleep deprivation.

### Effects of REM Sleep Deprivation in Humans

**The REM sleep drive.** Though the function of REM sleep is still a matter of controversy, REM deprivation studies indicate that there is a physiologically based motive, or drive, for REM-state sleep. In Dement's (1960) pioneering study of REM deprivation, the experimental procedure involved three phases: baseline, REM deprivation, and recovery. The baseline phase involved several nights of sleeping in the laboratory undisturbed, but with the usual physiological monitoring hookups. In the REM deprivation phase

the sleepers were awakened each time the polygraph record showed that they had entered the REM sleep state. They were forced to stay awake for a few minutes before returning to sleep. Further, subjects were told not to sleep during the daytime, in order to be sure that they did not catch up on REM sleep during the daytime. Then, after four or five nights of REM deprivation, the subjects were allowed to sleep undisturbed for several recovery nights. Finally, as a control procedure the subjects were awakened during NREM sleep for several nights (with the number of awakenings matching the number that had occurred during REM deprivation), followed by more recovery nights.

Two aspects of Dement's results indicated a motive for REM sleep. (1) *REM pressure*: The number of REM onsets increased, as if the sleeper was trying to get into REM sleep. Whereas REM onsets occur four or five times per night in undisturbed sleep, they occurred an average of eleven times on the first REM deprivation night, and increased to twenty-three times by the last deprivation night. Sometimes the experimenters had to awaken a subject every five or ten minutes to prevent REM sleep. (2) *REM rebound*: On the first recovery night, with undisturbed sleep, the amount of REM sleep was greater than on baseline nights (30 percent versus 19 percent REM, respectively). Other researchers have confirmed Dement's results, and it has also been found that the amount of time from sleep onset to the first REM period decreases on successive nights of REM deprivation.

**Behavioral effects of REM deprivation.** Dement's study supported the Freudian prediction, insofar as his results suggested that short-term REM deprivation caused some short-term psychological disturbances, including "anxiety, irritability, and difficulty in concentrating." One of the eight subjects quit the study "in an apparent panic" in the middle of the REM deprivation procedure, while two other subjects insisted on stopping REM deprivation one night short of the goal of five nights. Also, five subjects developed a marked increase in appetite during the REM deprivation period. However, all of these psychological disturbances disappeared as soon as the REM deprivation phase was over and the subjects were allowed to sleep undisturbed. And none of these disturbances occurred during the control phase, in which awakenings occurred during NREM instead of REM. Although none of the psychological effects of short-term REM deprivation were catastrophic, Dement speculated "It is possible that if the dream suppression were carried on long enough, a serious disruption of the personality would result" (1960, p. 1707).

Psychoanalysts were thrilled by Dement's findings, since they seemed to confirm the Freudian theory that dreaming is critical to maintaining mental health. Dement's findings were discussed in popular magazine articles and introductory psychology textbooks, often with exaggerations. However, subsequent research has usually failed to replicate Dement's findings of psychological disturbances. In most cases, assuming that the subjects do not experience any significant loss of total sleep time, selective REM deprivation does not have any clear-cut negative psychological effects. Some of the studies can be criticized on methodological grounds, such as failure to use NREM control awakenings, failure to use double-blind control procedures, failure

to use an adequate number of subjects, and the use of anecdotal reports rather than systematic psychological measurements (Ellman et al. 1978). However, the failure of REM deprivation to produce consistent psychological disruptions seems to go beyond methodological problems. One wonders whether Dement's original results might have been affected by negative expectancies on the part of the subjects. It has been shown that negative expectancies can produce unpleasant results in sensory deprivation experiments (Orne & Scheibe 1964), and the same problem might occur in REM deprivation experiments if the possibility of unpleasant experiences was somehow to be subtly communicated to the research subjects.

If there is a motivation for dreaming, then one might predict that REM deprivation would produce an overflow of dreamlike thinking into the waking state, producing an excess of vivid, fantastic daydreaming, and perhaps even waking hallucinations. However, this prediction has not generally been supported by research. As Hoyt and Singer (1978) pointed out, waking life is characterized by frequent daydreaming in most people, and in some people daydreams may be quite vivid and fantastic, even without prior REM deprivation. There is an increasing realization that the psychological effects of REM deprivation are likely to depend importantly on the personality characteristics of the individual, though unraveling the relationships is an enormously complex process (Cohen 1979a).

If REM deprivation has any consistent psychological effect at all, one might expect to find it within sleep itself. For example, perhaps the "dreaminess" of NREM mentation would increase during REM deprivation. Or perhaps some qualitative aspects of REM dreaming would be intensified on REM recovery nights. However, neither of these predictions was supported by the results of a careful experiment by Arkin et al. (1978).

In conclusion, researchers have looked for a variety of emotional and cognitive effects of REM deprivation, with inconsistent and largely negative results (see reviews by Arkin et al. 1978; Cohen 1979a; Ellman et al. 1978; Hoyt & Singer 1978; Vogel 1975). Thus, there is no clear evidence that REM dreaming (or REM sleep) is specifically necessary for the mental health of adults. (This is not to deny that some form of sleep is important for physical and mental health.) The idea that the function of REM sleep is to enable people to dream appears to be discredited. Dreaming may be a by-product of REM sleep, rather than a specific adaptive function of REM sleep (Hobson & McCarley 1977).

### **Ideas About the Function of REM Sleep**

Aside from the hypothesis that the function of REM sleep is to enable people to dream, most speculations about the function of REM sleep have had a neurophysiological orientation (Cohen 1979a).

**The CNS development hypothesis.** Observing that both the percent of sleep time spent in REM state and the total daily amount of REM drop progressively and substantially during the first four years of life, Roffwarg et al. (1966) suggested that REM sleep may be critical for the maturation of the central nervous system (CNS). A related, but narrower, idea is that REM sleep

is critical for the maturation of the oculomotor (eye movement) system. It seems impossible to test these ideas experimentally, even in animals, since it would be impossible to separate effects of the stress of repeated REM awakenings from effects of REM deprivation *per se*. Recent research indicates that protein synthesis in the brain increases during REM sleep, a finding that is consistent with the CNS development hypothesis. However, the functional significance of this finding is uncertain, since in the absence of information about the rate of protein breakdown during REM we do not know whether there is any net gain in brain protein during REM sleep (Horne & McGrath 1984). Of course, changes in protein "turnover" might be important, even without any net gain in protein.

**The memory consolidation hypothesis.** Currently the most influential idea about the function of REM sleep is that it promotes the physiological processes involved in consolidating long-term memories. Several experiments with animals have suggested that REM sleep deprivation interferes with memory of recent learning experiences. The research on humans is less clear, though largely negative. Perhaps REM is unnecessary for simple learning in humans, but it may be more important for memory of more complicated material; there is not enough evidence for firm conclusions on this point. Changes in protein synthesis, RNA turnover, and neurotransmitter production during REM are potentially relevant, though their functional significance cannot yet be determined. Two major reviews of the literature have concluded that the evidence offers some support for the consolidation hypothesis, without being sufficient to prove it (Horne & McGrath 1984; McGrath & Cohen 1978). REM sleep may facilitate memory consolidation, but REM is not strictly necessary for all consolidation.

Crick and Mitchison (1983, 1986) proposed that the function of REM sleep is to promote forgetting, not memory. They noted that when computer models of neural networks are overloaded with too much information they can "freeze up" and become dysfunctional. Random inputs can unfreeze the network, making it functional again. By analogy, they suggested that the brain's information processing efficiency may be reduced due to "parasitic modes" of useless memories. Random neurophysiological events during REM sleep may serve to remove the parasitic modes, thus restoring the brain's efficiency. Thus, REM sleep supports a "reverse learning mechanism. . . . We dream in order to forget" (1983, p. 112). In particular, "unconscious dreams"—dreams that we can never recall—are side effects of the reverse learning (information-dumping) process. Also, this process may account for some of the bizarre features of dreams that we do recall, such as condensation, in which dreams seem incoherent because parts of the story have been left out. Crick and Mitchison admit that there is no obvious way to test their hypothesis, aside from computer models of the brain's neural network. However, it should be noted that the reverse learning hypothesis is not entirely incompatible with the memory consolidation hypothesis, since the elimination of trivial memories may allow more important memory traces to be strengthened (Anch et al. 1988).

**Individual differences.** David Cohen (1979a) reviewed these and other ideas about the function of REM and NREM sleep in considerable detail. He argued that the mysteries of REM sleep will be unraveled only when researchers start to pay more attention to the interaction between sleep variables and individual difference variables, such as age, gender, temperament, and cognitive style. The issue of individual differences raises the problem to a level of complexity that is beyond the scope of this book, but it points out one reason why there are no easy answers to the question of the function of REM sleep.

**Differential functions of REM versus NREM sleep.** REM sleep is common in mammals, but rare or absent in reptiles. The evidence from living animal species suggests that NREM sleep evolved earlier than REM sleep (Cohen 1979a). This conclusion is admittedly speculative, since it is not possible to measure brain waves in fossils. In any case, given that we have two major sleep states, what might be their different functions?

Ernest Hartmann (1973) proposed that the primary function of REM sleep is brain restoration and growth, whereas NREM sleep is more concerned with vegetative processes and somatic (body) restoration and growth. A corollary of Hartmann's theory is that a need for REM sleep is experienced as mental fatigue, whereas a need for NREM sleep is experienced as physical fatigue. Mental fatigue is associated with dysphoria, strain, tension, and "regressive" behavior, whereas physical fatigue is a more pleasant condition, without the neurotic-like features of mental fatigue. There is some evidence that is consistent with Hartmann's hypothesis, though not conclusive (see Cohen 1979a).

Studies of selective NREM Stage-4 deprivation indicate a drive for Stage 4, with Stage 4 pressure and rebound effects even greater than those following REM deprivation (Webb 1969). Some evidence tentatively supports the notion of a relationship between NREM and somatic restoration (Cohen 1979a). Circumcision and short-term starvation are associated with increased delta sleep. Also, some studies have suggested that physical exercise is followed by increased Stage-4 sleep, though the evidence is inconsistent. Following total sleep deprivation, Stage-4 rebound takes precedence over REM rebound. However, research on the effects of both selective and total sleep deprivation leads to the conclusion that neither sleep state is absolutely necessary for any important physiological process. Rather, certain physiological processes may be facilitated in certain sleep states. We are left with a great unsolved scientific mystery: although partial sleep deprivation produces discomfort and fatigue in humans, and total sleep deprivation produces death in animals, no specific, vitally necessary function of either REM or Stage-4 sleep has yet been identified.

## SLEEP DISORDERS

In recent years the health and safety implications of sleep disorders have been increasingly recognized, and centers for diagnosing and treating sleep disorders have opened up across the country. A manual of criteria for diag-

nosing sleep disorders has been published by the Association of Sleep Disorders Centers (ASDC 1979).

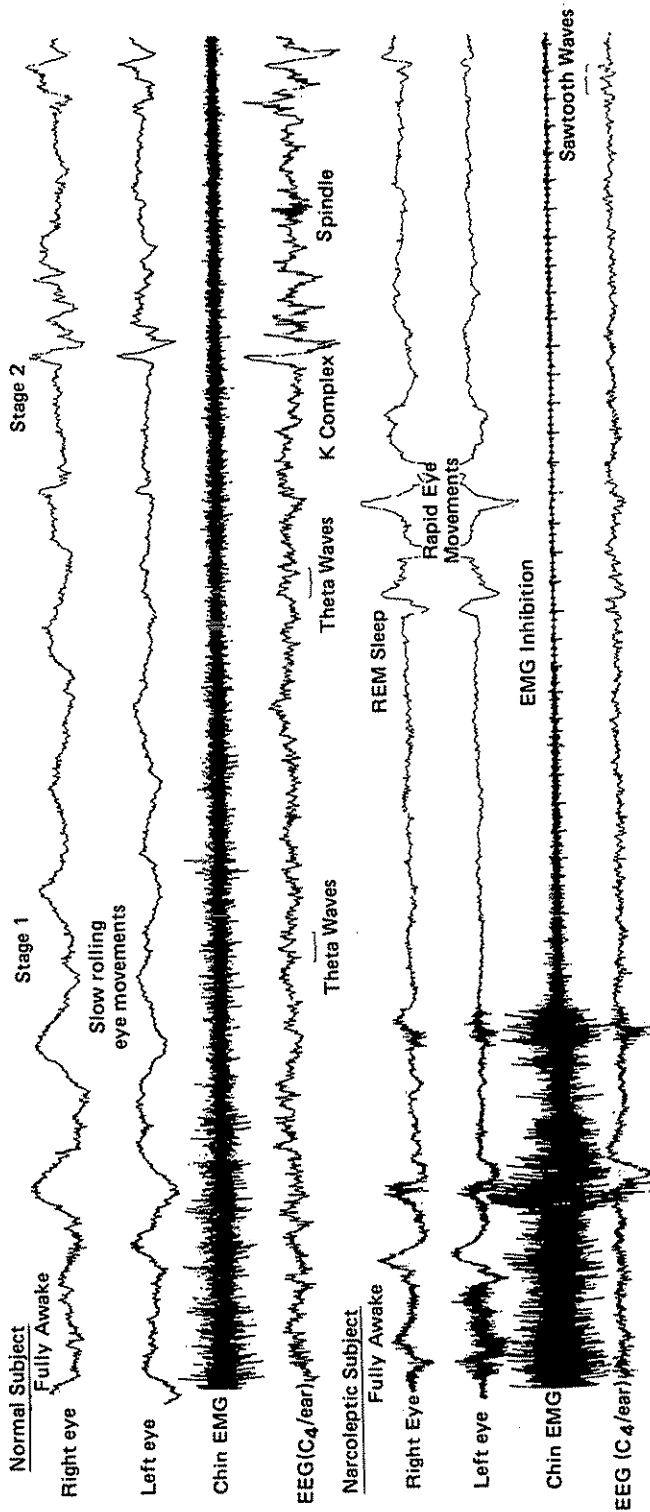
Sleep disorders are classified in three major categories (Webb 1975): (1) *primary sleep disorders*, in which sleep intrudes on the waking state (termed DOES, disorders of excessive somnolence, in the ASDC manual); (2) *parasomnias*, in which waking-like behaviors intrude on sleep (termed DSWS, disorders of the sleep-wake schedule, in the ASDC manual); and (3) *insomnias*, in which the onset or maintenance of sleep is disrupted (termed DIMS, disorders of initiating and maintaining sleep, in the ASDC manual; Anch et al. 1988).

### Primary Sleep Disorders

**Narcolepsy.** Narcolepsy is a chronic, lifelong condition characterized by excessive daytime sleepiness and the occurrence of REM at sleep onset (see Figure 10.6). "Sleep attacks," brief uncontrollable periods of sleep, may occur several times each day. The attacks, which usually last from about two to fifteen minutes, may occur at any time when the narcoleptic is going about his or her usual daily business. Sometimes they are brought on by strong emotional arousal; narcoleptics sometimes fall asleep while making love. Approximately five people in a thousand suffer from narcolepsy, which is known to have a genetic basis. In the large majority of cases the narcoleptic syndrome first appears between the ages of fifteen and twenty-five years.

There are four characteristic clinical symptoms of narcolepsy, called the "narcoleptic tetrad" (Anch et al. 1988; Webb 1975). (1) *Excessive daytime sleepiness*. The narcoleptic is chronically sleepy, even after a normal night of sleep. Narcoleptics tend to have recurrent "microsleep" episodes during the daytime. During these episodes, which last from five to fifteen seconds, the EEG pattern indicates bursts of theta waves or Stage 1 NREM. Without overtly napping, the individual has a glassy-eyed look, and perceptual processing and thinking decrease. The frequency of microsleep episodes tends to increase unless the narcoleptic takes a nap. When allowed to nap during the day the narcoleptic typically falls asleep in less than five minutes, compared to ten to fifteen minutes for the normal adult. However, chronic daytime sleepiness by itself is not an indicator of narcolepsy. Other symptoms must also be present, especially the occurrence of REM state at sleep onset during daytime naps; in normal adults brief daytime naps involve NREM sleep stages. When they go to sleep at night, narcoleptics usually go directly into REM stage, whereas the first REM period is delayed at least sixty minutes in normal adults. (2) *Cataplexy*, which involves muscular weakness, and may range from a feeling of tiredness to complete inability to move. It occurs in about 70 percent of the daytime sleep episodes. Cataplexy often occurs without the person actually falling asleep. It may be brought on by high emotional arousal. For example, a narcoleptic hunter, presented with an exciting opportunity to shoot a deer, may drop his gun and fall to the ground without necessarily losing consciousness. Cataplexy and extreme daytime sleepiness are the most frequent symptoms of narcolepsy. (3) *Sleep paralysis*, which involves a total paralysis during the time that the narcoleptic is falling asleep, and sometimes upon awakening from sleep. The muscular paralysis charac-





**FIGURE 10.6.** Polygraph recordings showing the transition from wakefulness to sleep in a normal subject and a narcoleptic subject. The normal subject moves from the waking state to Stage 1 NREM to Stage 2 NREM. Note the EEG K-complex and spindle in Stage 2. The narcoleptic subject moves from waking directly to REM stage sleep. Note that in REM stage the right and left eye movements are better synchronized (conjugate) with each other than in NREM sleep. Also note the deep muscle relaxation (EMG) in REM stage. [From Hauri, P. (1982). *The Sleep Disorders*. Kalamazoo, MI: The Upjohn Company. Reprinted with permission of the publisher.]

teristic of REM stage can occur before entering full REM sleep and continue after awakening. (4) *Hypnagogic hallucinations*, which involve vivid visual or auditory images—sometimes frightening—that occur at the beginning of the sleep attack, before the narcoleptic is fully asleep. Hypnagogic hallucinations also occur in normal people, but in narcoleptics they seem to be particularly frequent and frightening, perhaps due to their appearance along with sleep paralysis.

Narcolepsy has been characterized as a disorder of REM sleep, in which REM intrudes into the waking state. Sleep attacks can be dangerous, as when a person is driving a car, and they disrupt intellectual and social functioning. There is no cure for narcolepsy. The symptoms can be managed by careful planning of one's social and work schedule to allow for frequent planned daytime naps, and by stimulant drugs (Anch et al. 1988).

**Hypersomnia.** Hypersomnia involves excessive sleep, beyond twelve hours per day. A variety of different conditions may be related to hypersomnia in different patients. Often there is a brain disorder. Some cases are related to insomnia due to *sleep apnea*, which is disrupted sleep caused by a breathing disorder. Cases of "functional" hypersomnia (without a clear organic basis) are relatively rare. Such cases have distinctly different EEG profiles, compared to narcoleptics. Hypersomniacs spend a relatively large amount of time in Stages 3 and 4 NREM, and relatively little in REM. They may spend a lot of time napping, but their naps are not refreshing, and they have difficulty waking up afterward (Webb 1975).

### Parasomnias

Parasomnias involve intrusions of certain wake-like behaviors into the sleeping state. These include sleepwalking, sleeptalking, enuresis (bed-wetting), and night terrors. The parasomnias often occur together in various combinations in the same patient. They all seem to involve episodic disturbances of NREM sleep. The parasomnias occur more often in males than in females, and there appears to be a genetic basis, in that they tend to run in families (Anders et al. 1980).

**Night terrors.** Night terrors (*pavor nocturnus*, or sleep terrors) are panic reactions associated with sudden, spontaneous arousal from sleep, usually during NREM Stage 3 or 4. Night terrors rarely occur in adults. They occur most commonly in children three to five years old (affecting about 2 to 5 percent of this age group; Anders et al. 1980), and they may continue into early adolescence. The sleeping child sits up suddenly and screams. There are signs of intense emotional arousal suggesting panic: exceptionally rapid heartbeat, rapid breathing, perspiration, and a wide-eyed stare. Full waking consciousness may not occur until several minutes after the initial arousal. Parental efforts to comfort the child have no effect. The child usually has no recollection of any dream. When imagery is recalled, it is more likely to be a single frightening image, rather than a story-like dream. The whole episode usually lasts only a minute or two, after which the child goes back to sleep. The episode usually will not be recalled by the child in the morning.

Sleep laboratory studies have shown that night terrors occur during the first third of the night, usually about forty to sixty minutes after falling asleep. The large majority of cases involve sudden arousal from NREM Stage 3 or 4, usually from the first ones of the night. It is not uncommon for the night terror to be accompanied by sleepwalking or sleepwalking or enuresis, though these events can also occur independently of night terror.

Night terrors are more likely to occur during periods of daytime stress or fatigue. For example, the stress of moving to a new city, or starting school, may bring on night terrors in susceptible children. Sleep loss also aggravates night terrors (Anch et al. 1988). They may have an organic basis, such as neurological immaturity (Anders et al. 1980) or illness with a high fever (Kales, Kales et al. 1980).

Night terrors differ from nightmares in several ways (Hartmann 1984). *Nightmares* are emotionally disturbing dreams that typically occur in REM sleep, rather than NREM. People usually recall their nightmares vividly, whereas night terrors usually have no associated dream imagery (at least in children). Nightmares usually occur in the latter part of a typical night of sleep (six to eight hours), when REM sleep is most intense, whereas night terrors usually occur before the first REM period of the night. Nightmares may be accompanied by physiological signs of emotional arousal, though arousal is not as intense, nor is its onset so rapid, as in night terrors. Nightmares are more likely to emerge during ages seven to ten, whereas three to five is the age for most night terrors. Occasional nightmares are not unusual, but recurrent nightmares may reflect stress or anxiety that has a basis in daytime experience. Night terrors, on the other hand, are more distinctly an abnormality of sleep.

Broughton (1966) suggested that night terrors are disorders of arousal. That is, they stem from a rapid transition from Stage 4 NREM to waking. He suggested that in those relatively rare cases (in children) in which dreamlike images are associated with the terror, the images are in response to the terror, rather than the cause of the terror. Fisher et al. (1974), on the other hand, finding rather frequent frightening dream images associated with night terrors in adults, suggested that night terrors may be caused by frightening NREM dream images. The suddenness of the physiological arousal in night terrors (as contrasted with its more gradual buildup in REM nightmares) suggests that a sudden, unexpected and frightening dream image might elicit the terror reaction. (Fisher et al. observed that night terrors could sometimes be elicited also by a sudden, loud buzzer. External stimuli might account for some cases of night terrors in children.)

Fisher et al. (1974) found night terrors in adults to be associated with daytime stress and psychopathology, which is not necessarily the case in children. As present, we know only that frightening dream images are sometimes associated with night terrors in adults, though rarely in children. The direction of causality is uncertain. It may be that the causal factors in night terrors are different for children and adults.

**Sleepwalking.** Sleepwalking (*somnambulism*) occurs in Stage 4 sleep. Episodes may last from a few seconds to a few minutes. Sleepwalking is not the acting out of a dream. If you awaken a sleepwalker he or she will proba-

bly be confused, with no dream recall. Unless fully awakened during the episode, the sleepwalker will be amnesic for the event in the morning. The sleepwalker is relatively nonreactive to the environment. Behaviors during sleepwalking are typically habitual or "automatic" ones. Simple actions are most common, such as briefly standing up, then going back to bed. Somewhat more complex actions such as getting partially dressed, or going to the bathroom (perhaps mistaking the closet for the bathroom) also occur. Rarely, complex episodes occur, such as going downstairs and rearranging objects (Anch et al. 1988).

Sleepwalking is most common in children and adolescents, and is relatively rare in adults. Many people may know of one or two instances in which they have sleepwalked, without it being a common occurrence for them. Sleepwalking is sometimes, but not always, associated with night terrors. Sleepwalking in adults is associated with severe life stress and psychopathology (Kales, Soldatos, et al. 1980). Contrary to a popular myth, sleepwalking is potentially dangerous. To protect sleepwalking children, parents should lock doors and use stair gates. There is no danger in awakening a sleepwalker, though he or she is likely to be confused, and may need reassurance.

**Sleeptalking.** Sleeptalking (*somniloquy*) may not be an every-night occurrence for most people, but it is not rare (Arkin 1978). Most people know of at least one instance when they have talked in their sleep, but the actual frequencies can never be known with much confidence, since people do not recall their own sleeptalking episodes; rather, their knowledge of their own sleeptalking depends on what other people have observed and reported to them.

Sleeptalking is not strongly associated with any particular sleep stage; it can occur in either REM or NREM, and it occurs more often in NREM because NREM takes up about 80 percent of a normal night's sleep. Sleep utterances are often associated with body movements. Some cases of sleeptalking occur at times when the EEG shows alpha waves suggesting wakefulness, though the person is behaviorally asleep in the sense of being nonreactive to the environment. Though sleeptalking sometimes is associated with psychopathology, it occurs so often in normal people that it is not, in itself, considered to be a sign of psychopathology. Sleeptalking sometimes occurs during night terrors (Kahn et al. 1978), but most cases have nothing to do with night terrors.

What is sleeptalking like? Arthur Arkin (1978), who has observed many sleeptalkers in the laboratory, said that it is not possible to give a brief description of sleep utterances because they show almost as much variability as those of wakefulness.

Although the majority of sleep speeches contain at least a few words, some consist of only one, such as "good," "no," "okay," "yes," or "Mm-hm." Others are of paragraph length, occasionally in excess of one hundred words. Most speeches last a few seconds or less, but longer ones may continue for a minute or more. The range of clarity extends from unintelligible mumbles to crystal-clear words. . . . Often, speeches contain silent pauses, in which case the context suggests sleep dialogues with hallucinated partners, sometimes resembling one

side of a telephone conversation. Another frequent occurrence is sudden interruption of a speech in the middle, followed by sustained silence or an apparently meaningless mumbled petering out. The hearer is left with a feeling that a thought has been fragmented or left incomplete (Arkin 1978, p. 521).

What about the structural features of sleep speech? Sleep utterances are somewhat more likely to have correct syntax (word sequencing) and inflection when they occur during REM. When markedly abnormal utterances occur, they typically occur during NREM. Such abnormal speech can range from mildly distorted to sheer gibberish, in which clang (rhyme) associations and recurrent utterances are prominent, with occasional neologisms (novel, nonsense words; see Arkin 1978).

As for the content of sleep speech, Arkin (1978) was struck by the rarity of secrets. Though the telling of secrets during somnolity is a popular device in literature (for example, in Shakespeare's *Othello*), in actuality it hardly ever happens. Nor do obscene utterances occur very commonly in sleep speech. Arkin reported that:

the majority of intelligible, clear utterances sound like fragments of overheard, unremarkable daily conversations. . . . [Also, some cases] resemble words and sounds one utters in solitude while awake. Exclamatory words, phrases, sounds of surprise, curiosity, pleasure, agreement, and so on are common. In addition, one encounters utterances resembling wakeful vocal self-priming or stimulation as if someone were following a recipe or other stepwise task, and wondering aloud what to do next (Arkin 1978, p. 522).

Sleep utterances can suggest a wide range of emotions, from positive to negative. When sleeptalking occurs during night terrors or nightmares, the utterances may show strong emotion, such as anxiety or panic. But in most cases sleep utterances are unemotional.

One of the most interesting questions about sleeptalking concerns the degree of *concordance* between the content of sleep speech and the content of dream or thought reports obtained by waking the subject shortly after the speech episode. Arkin (1978) examined content concordance in 166 sleep speech episodes, obtained from 28 adult chronic sleeptalkers in the laboratory. When sleep utterances occurred, they were more likely to be followed by mentation reports for REM (96%) than for NREM Stage 2 (79%) or Stages 3-4 (62%). In cases in which mentation was recalled, high levels of content concordance were more common for REM periods (54% concordance) than for NREM Stage 2 (26%) or Stages 3-4 (17%). Only 15 percent of all sleep utterances occurred in REM.

Though concordances occurred fairly often, especially in REM, the sleep speech was not a detailed description of the dream events. Early hopes by researchers that sleeptalking would be a way to obtain "play-by-play" commentary on dream action have met with disappointment. In commenting on his sleeptalking data, Arkin (1978) suggested that failures of concordance may indicate "multiple dissociated concurrent streams of mentation." That is, the speech control mechanism may sometimes be under the control of a stream of thought that is dissociated (disconnected) from dream consciousness. Dream consciousness and another stream of consciousness may some-

times occur independently, in parallel, with only the latter controlling sleep utterances, and only the former controlling dream reports upon awakening.

### **Insomnia**

People may complain of insomnia when they believe that they are having problems with any of four different aspects of sleep: (1) trouble falling asleep, or technically, long sleep onset latency; (2) frequent awakenings during the night, after initially falling asleep; (3) early awakening from sleep, without being able to get back to sleep; and (4) light sleep. Surveys have shown that approximately 15 percent of adults are often troubled by insomnia, and another 30 percent say they sometimes have insomnia (Webb 1975).

Insomnia presents an important practical problem. Sleep difficulties may be a symptom of other psychological problems, such as depression. And sleep difficulties may themselves be a source of anxiety. Insomnia produces daytime sleepiness. People often feel that their work or study performance, or their interpersonal relationships, would be better if only they could sleep better. Even though occasional or mild insomnia may not cause any serious decrement in performance, people believe that it does, and the belief increases anxiety over insomnia (and the anxiety may, in turn, exacerbate the insomnia problem).

Diagnosing insomnia is not as clear-cut as you might think. There are wide individual variations in sleep parameters (such as sleep onset latency and frequency of nighttime awakenings) within the normal range. So clinicians have to use some arbitrary criteria in classifying patients' insomnia problems. Wisle B. Webb (1975) used the following criteria, based on EEG recordings during sleep: (1) sleep onset latency: over 30 minutes; (2) nighttime awakenings: five or more, or totaling over 30 minutes; (3) early termination: spontaneous termination before six hours have elapsed; (4) light sleep: an EEG record showing over 12 percent of Stage 1 NREM, or less than 3 to 5 percent Stage 4 NREM. About 95 percent of healthy adults from twenty to forty years of age would be defined as normal according to these criteria, whereas about 5 percent would be classified as having at least transient (short-term, temporary) insomnia.

**Transient insomnia.** Transient insomnia, lasting one or a few nights, is common. Virtually everyone experiences it at one time or another. Transient insomnia is a reaction to a temporary situation, such as anxiety over an upcoming exam or job interview, or sleeping in a strange place. (Transient insomnia is fairly common the first night subjects sleep in a sleep laboratory or clinic.) Transient insomnia does not require treatment—it clears up spontaneously when the source of anxiety is resolved, or when you adapt to the new situation.

**Chronic insomnia.** Chronic insomnia—insomnia that persists for several months—can have a variety of causes, and it requires different treatments depending on the cause. Most cases of chronic insomnia fall into one of five categories (Coleman 1986).

(1) *Psychiatric insomnia* is the most common category (35 percent). In

these patients insomnia is a symptom of some more comprehensive psychological disorder, the most common being depression and anxiety. Thus, the main concern is treating the psychological disorder.

(2) In *stress-conditioned insomnia* (about 15 percent of cases) the patient's main problem is anxiety over whether he or she will sleep well and be able to function well the next day. Such a condition begins with what would ordinarily have been a transient insomnia in reaction to a stressful life event, but the insomnia becomes chronic because the patient worries that poor sleep will lead to poor performance the next day. Treatment requires a program of counterconditioning to establish better knowledge, attitudes, and habits regarding sleep.

(3) *Physiological insomnia* (30 percent) is a broad category that encompasses several types of physiological causes of insomnia. The most dramatic is *sleep apnea*, in which people stop breathing when they fall asleep. Loud snoring is a major preliminary diagnostic symptom of apnea. Snoring is caused by a progressive narrowing of the airway as throat muscles relax during sleep, or by an anatomical abnormality. When the airway is completely closed breathing stops. Subsequent buildup of carbon dioxide in the bloodstream triggers a brief awakening, when breathing starts again, until sleep resumes. Thus, such patients go through repeated cycles of brief sleep periods (about 30–60 seconds) and brief waking periods (5–10 seconds). In the morning they typically do not recall the brief nighttime awakenings. Since apneics sleep poorly, they often suffer from severe daytime sleepiness. Some clinicians believe that apnea is a cause of Sudden Infant Death Syndrome (SIDS), and death during sleep in the elderly. The standard treatment is tracheotomy, a surgical procedure that involves making a small hole in the trachea. At night the patient puts a tube in the hole, through which he or she can breathe while asleep.

Another group of patients who experience nighttime awakenings are those who make periodic reflexive leg jerks during sleep, which may occur as often as every 30 seconds in some patients. Various medical disorders, such as arthritis and biological rhythm disorders, also cause frequent nighttime awakenings.

(4) *Poor sleep habits* are characteristic of 10 to 15 percent of chronic insomniacs. They can be a part of other disorders, and also a problem in their own right. Excessive or inappropriate use of drugs, such as alcohol, caffeine, and sleeping pills, can seriously disrupt sleep cycles. For example, coffee after 6 p.m. may cause delayed sleep onset at 11 p.m. Alcohol does not interfere with sleep onset, but it may cause frequent awakenings or early termination of sleep. Irregular sleep schedules are another poor sleep habit associated with insomnia; a regular bedtime and awakening time are the solution. Insomnia is really a twenty-four-hour problem. Your habits during the day will affect how you sleep at night.

(5) *Pseudo-insomnia*. In this category (about 10 percent of sleep patients) are those puzzling cases in which the patient's complaint of chronic insomnia is not supported by the objective EEG records obtained in the laboratory or sleep clinic. When confronted with the objective evidence, some patients are relieved to find that they do not really have insomnia. Others are incredulous and insist that they had little or no sleep and that the EEG records are

in error; some patients have a paranoid reaction. Coleman (1986) suggested that "In some cases, our measuring techniques may not be sufficiently sensitive to detect small physiological arousals or active thinking, either or both of which may cause the perception of no sleep at all." Perhaps in some cases the subjects dreamed that they were awake. These patients had presleep anxiety about being able to sleep successfully, and these presleep thoughts may have affected their subsequent sleep mentation.

### Drugs and Sleep

When people suffer from insomnia, doctors sometimes prescribe sleep-inducing drugs, termed *sedatives* or *hypnotics* ("sleeping pills," such as barbiturates). Hypnotics have disadvantages in that they may be addictive, they tend to reduce subsequent daytime alertness, and they suppress REM sleep. Withdrawal from hypnotics leads to REM rebound, sometimes with nightmares, and irregular sleep patterns characterized by frequent awakenings (Anch et al. 1988; Webb 1975). Drugs taken for other reasons may reduce alertness as a side effect (for example, antihistamines [used in allergy and decongestant medicines], tranquilizers, alcohol).

*Stimulants* (such as caffeine and amphetamine) are intended to counteract sleep and maintain alertness. Caffeine is the most widely used stimulant in the world (Schlaadt & Shannon 1986). It is found in coffee, tea, cola soft drinks (and also some noncola soft drinks—read the label), chocolate, and in some nonprescription drugs (such as *No-Doz*). As a method of maintaining alertness, caffeine is certainly safer than amphetamines. But caffeine has its drawbacks. People develop a tolerance to caffeine. As a general rule, the more often you drink coffee on a habitual, daily basis, the less effective a single cup will be in keeping you awake and alert. Thus, in order to produce the same stimulating effect, the dosage must be steadily increased. High doses cause "jitters" and a need for frequent urination. And caffeine is physiologically addicting. Coffee and cola drink addicts crave the drug, and when they try to quit they suffer withdrawal symptoms, such as headaches, nervousness, and irritability. The optimal way to use caffeine is to use it occasionally, when you really need it. Do not use it on a daily basis. That way you will get the maximum benefit from a single dose, but you will not suffer the disadvantages of addiction.

It is best to avoid using drugs to regulate the sleep-wake cycle. Some people get into a vicious circle when they use sleeping pills to help them sleep at night, then they feel sleepy the next day so they use stimulants to stay awake, and consequently they have trouble falling asleep that night so they take more sleeping pills, and so on and on until they develop serious drug dependencies.

### SUMMARY

Sleep is characterized by reduced awareness of the external environment, reduced muscle activity and voluntary action, and reduced self awareness, as well as changes in brain waves and other physiological measures. Human



sleep is not a uniform state. Rather, it involves several physiologically distinctive stages that recur in a cyclic manner. The waking state, REM (rapid eye movement) sleep state, and non-REM (NREM) sleep state are distinguished by their unique patterns of three physiological indicators: (1) brain waves (faster and lower amplitude in waking and REM than in NREM); (2) eye movements (more frequent in waking and REM than NREM); and (3) chin muscle tension (lower in REM than in waking and NREM). NREM may be subdivided into Stages 1, 2, 3, and 4, characterized by increasingly slower and higher amplitude brain waves. REM periods of several minutes' duration recur on a cycle of about 90 minutes; successive REM periods are increasingly longer in duration. Vivid dreaming occurs during REM sleep.

The function of sleep is generally assumed to be physiological restoration. Animals totally deprived of sleep for several days or weeks have died. Yet specific, vitally necessary physiological processes that occur only in sleep have not yet been identified. In humans, sleep deprivation, either partial or total, is characterized by feelings of fatigue and irritability, and by decreased performance on tasks that require sustained attention. However, there is little or no effect on performance of brief tasks when their timing is under the subject's control and performance motivation is high. There have been several hypotheses about the functions of REM and slow-wave sleep, but none has been clearly confirmed. Selective REM deprivation in humans results in REM persistence (increased frequency of REM onset) and REM rebound (increased total REM duration on recovery nights), which suggest that there is some need or motive for REM sleep. However, no changes in personality or performance have been identified that can be attributed to REM deprivation *per se*.

Sleep disorders can be classified in three categories: (1) primary sleep disorders, in which sleep intrudes on the waking state; (2) parasomnias, in which waking-like behaviors intrude on sleep; and (3) insomnias, in which the onset or maintenance of sleep is disrupted. The major primary sleep disorder is narcolepsy, characterized by excessive daytime sleepiness and microsleeps, REM at sleep onset during daytime naps, cataplexy (muscular weakness), sleep paralysis, and hypnagogic hallucinations. Parasomnias include sleepwalking, night terrors, and enuresis, all of which tend to occur during Stages 3 and 4. Sleepwalking can occur in any sleep stage; its content is sometimes related to dream images, though it does not provide a detailed description of ongoing dream action. Insomnia may be associated with psychological stress, psychopathology (especially depression and anxiety), physiological disorders (such as sleep apnea, in which breathing stops during sleep), poor sleep habits (irregular sleep-wake patterns), and misuse of drugs that affect the sleep cycle.

## ENDNOTES

<sup>1</sup>See Hobson (1988), *The Dreaming Brain*, for an interesting description of the history of research on sleep, dreaming, and the brain processes involved in them.

<sup>2</sup>A note on terminology: Alternate names for REM sleep, used by some writers, are: "Stage 1 REM", "ascending Stage 1" (Stage 1 NREM is "descending Stage 1"), and "paradoxical" sleep

(PS). REM is paradoxical because the brain waves, eye movements, and autonomic arousal response-measures give the appearance that the subject is awake, whereas in fact he or she is sound asleep. REM has also been called "D-state," referring to both desynchronized brain waves and dreaming, in contrast to "S-state" sleep for NREM sleep, referring to the more synchronized brain waves (Hobson & McCarley 1977). Stages 3 and 4 are sometimes called "slow wave" sleep (SWS). The transition period from waking to sleeping is called the "hypnagogic state" (to be discussed in Chapter 11).

<sup>3</sup>Some studies have found body movements to be more frequent in Stage 4 than in REM. The discrepancies in results between different studies probably have to do with different methods of measuring body movements (Anch et al. 1988).