77 Sleep Disturbances

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Definition

Sleep disturbances encompass disorders of initiating and maintaining sleep (DIMS, insomnias), disorders of excessive somnolence (DOES), disorders of sleep–wake schedule, and dysfunctions associated with sleep, sleep stages, or partial arousals (parasomnias). Table 77.1 presents a useful classification.

Technique

To clarify the complaint of sleep disturbance, the clinician should become familiar with the broad categories of sleep disorders within which the Association of Sleep Disorders Centers (1979) has detailed a diagnostic classification of sleep and arousal disorders. Of clinical relevance is that the diagnoses addressed in the first two divisions of the classification were ordered according to the patient's complaint of insomnia or excessive daytime sleepiness. Historically exploring the patient's presenting sleep-related complaints provides the highest likelihood of obtaining significant data about the underlying condition.

Disorders of Initiating and Maintaining Sleep

The sleep and arousal disorders most frequently encountered by the clinician are disorders of initiating and maintaining sleep, the insomnias. When the patient complains of "trouble sleeping," is the patient having difficulty falling asleep? Once asleep, is the patient able to remain asleep for the desired length of time? How long does the patient sleep? What is the patient's perception of the quality of the sleep?

Since the sleeping state and the waking state are both integral components of a 24-hour continuum, the quality of the patient's waking state is equally relevant. How does the patient feel upon awakening in the morning? How does he or she function during the day? Does the patient feel tired and/or involuntarily fall asleep during the day?

A few patients awaken refreshed and function perfectly well, despite sleeping much less than expected. Except for brevity, their sleeping appears to be normal. These so-called short sleepers regularly have a daily sleep total that is less than 75% of that usually expected for their age.

In other apparently psychologically healthy patients who complain of insomnia, objective studies fail to reveal any sleep pathology. Despite falling asleep in less than 15 to 20 minutes and sleeping more than 6.5 hours, these patients are convinced that their sleeping is inadequate (insomnia without objective findings). The combination of these "pseudoinsomniacs" with the "short sleepers" constitutes about 30% of patients complaining of insomnia.

In others, disturbed sleep may involve some very fundamental aspects of sleep hygiene. Does the patient have a regular time for awakening? Are there disturbing environmental factors? Does a change in lighting occur during sleep? Is the room's temperature suitable for the patient's particular sleeping needs? Is the patient's bed partner bothersome?

Tolerance to environmental factors varies from individual to individual. In attempting to establish a relationship between an environmental factor and disturbed sleep, the clinician needs to consider whether the disturbance correlates temporally with a stimulus that is not only disturbing but that can be physically measured. Second, the disturbing aspects of the stimulus should arise from its physical properties rather than its emotional meaning to the patient. Third,

Table 77.1
Classification of Sleep Disorders

I. Insomnias: Disorders of initiating and maintaining sleep
  A. Psychophysiologic—situational or persistent
  B. Associated with psychiatric disorders, particularly affective disorders
  C. Associated with drugs and alcohol
    1. Tolerance to or withdrawal from CNS depressants
    2. Sustained use of CNS stimulants
    3. Sustained use of or withdrawal from other drugs
    4. Chronic alcoholism
  D. Associated with sleep-induced respiratory impairment
    1. Sleep apnea syndrome
    2. Alveolar hypoventilation syndrome
  E. Associated with sleep-related (nocturnal) myoclonus and "restless legs"
  F. Miscellaneous—other medical, toxic, or environmental conditions

II. Hypersomnias: Disorders of excessive somnolence
  A. Psychophysiologic—situational or persistent
  B. Associated with psychiatric disorders, particularly affective disorders
  C. Associated with drugs and alcohol
  D. Associated with sleep-induced respiratory impairment (as in D above)
  E. Narcolepsy–cataplexy
  F. Miscellaneous—other medical, toxic, environmental, or idiopathic conditions

III. Disorders of the sleep–wake schedule
  A. Transient—jet lag, work shift
  B. Persistent
    1. Delayed sleep phase syndrome
    2. Advanced sleep phase syndrome
    3. Non-24-hour sleep–wake syndrome

IV. Parasomnias: Dysfunctions associated with sleep, sleep stages, or partial arousal
  A. Sleepwalking
  B. Sleep terrors and dream anxiety attacks
  C. Enuresis
  D. Nocturnal seizures
  E. Other sleep-related dysfunctions

From Schwartz WJ, Stakes JW, Martin JB. The sleep-wake cycle and disorders of sleep. In Braunwald et al., eds. Harrison's principles of internal medicine. New York: McGraw-Hill, 1987, p. 111. Reproduced by permission.

there should be at least a gradual return to satisfactory sleep once the stimulus is removed.

Does the patient nap during the day? Naps may improve nighttime sleeping, but often they interfere with it. This is particularly true of elderly patients.

What is the duration of any sleep disturbance? This information is relevant to the differential diagnosis of the complaint and to the management of the problem. A disturbance of recent origin will prompt the clinician to seek precipitating causes. With regard to chronic insomnia, the most common cause is *psychiatric disturbance.*

In the proper context, a sleep disturbance may constitute strong evidence for the diagnosis of unipolar depression. How is your sleeping? A frequent response is, "No good." In what way is it not good? Commonly, the patient awakens and has difficulty getting back to sleep. At what time do you awaken? A frequent reply names 2:00 or 3:00 A.M. Can you fall asleep again? The answer often is no.

Other psychiatric illnesses may be associated with different sleep symptoms. Bipolar depression is more often associated with sleeping that is excessive yet unrefreshing. Patients with manic phases often seem to require little sleep. The generally anxious patient may have difficulty getting to sleep, restless sleep, or both. Sleep is often disturbed in psychotic conditions. There seems to be direct correlation between the degree of sleep disturbance and the severity of the psychiatric illness.

Even in the absence of major psychiatric illness, insomnia may begin with an emotionally arousing event. This is termed *situational psychophysiological insomnia.* The sleep can be expected to normalize when the emotional reaction to the event subsides. A disturbance of less than 3 weeks is regarded as transient.

In persistent psychophysiological insomnia, there is usually a history of prestress sleeping that was fair at best. The added burden of excessive stress leads to a further deterioration of sleep with an accompanying deleterious effect on daytime functioning. Increased efforts to obtain sleep may themselves stimulate arousal. Eventually, even the bedtime routine may continue to stimulate arousal long after the original stressful situation has abated. Such patients usually sleep better when on vacation or even on weekends.

In any case of insomnia, a *drug history* is essential. Some patients use alcohol as a sleep inducer. Since the effect is short-lived, the patient may awaken in a few hours and have difficulty returning to sleep. In contrast, the chronic alcoholic may have a low total sleep time because of persistent interruptions of sleep. Acute withdrawal may be accompanied by a marked delay in sleep onset, which may progress to delerium tremens.

Some patients are exquisitely sensitive to caffeine. Inquiry should address not only coffee but also tea, cola, and chocolate. Cigarette smoking can interfere with sleep, most likely by stimulating catecholaminergic systems. In addition, the heavy smoker who stops smoking may transiently experience sleep disturbance resulting from nicotine withdrawal.

The patient must also be asked about more traditionally recognized sleep medications. How long have any such medications been used? Has the dose progressively increased? Hypnotic medications typically lose their efficacy within a few weeks, often prompting an increase in dosage. Any added sleep response also becomes merely transient. When hypnotic agents are used chronically, frequent nighttime awakenings of more than 5 minutes each may occur. A history of disturbed sleep maintenance during the latter half of the night may suggest shortened drug efficacy due to the development of tolerance. Conversely, precipitous withdrawal can produce a marked worsening of sleep disturbance, often associated with nightmares.

Is the patient taking central nervous system (CNS) stimulants (e.g., amphetamines)? Insomnia can also result from steroid medications and from centrally acting adrenergic blocking agents. The adrenergic agonists and the xanthines used as bronchodilators can interfere with sleep. The latter substances are chemically related to caffeine.

What is the patient's *general medical condition?* Pain is particularly disturbing to sleep. Does the patient have frequent headaches, angina pectoris, reflux esophagitis, peptic ulcer disease, or arthritis? Is there nocturia, fever, or pruritus? Are there paresthesias? Does the patient have nocturnal dyspnea or coughing? Is there hyperthyroidism or renal failure or an organic brain syndrome?

Some patients with disturbed sleep complain more of the associated impaired daytime wakefulness. Such is usually the case with *sleep apnea,* a syndrome characterized by multiple periods of absent airflow, lasting 10 seconds or longer and occurring at least 30 times during a full 6-hour sleep study. Sleep apnea may be purely secondary to obstruction of the upper airway, in which case there is no airflow through the nose or mouth despite continued respiratory efforts of the thorax and abdomen, or it may be central, in which the cessation of airflow is concomitant with interruption of thoracic and diaphragmatic movements. The latter type likely reflects a disturbance in the CNS regulation of breathing. Most cases are mixed—initially central, later obstructive. Since sleep apneic patients who complain of insomnia are more likely to have the central form, the history from a bed partner may not necessarily reveal loud snoring or gasping respirations. Nevertheless, an observant bed partner may note periods of apnea.

Patients with *nocturnal myoclonus* may report that they are restless sleepers, but they are usually unaware of their periods of repetitive twitching of the legs, interspersed with periods of apparently normal sleep. The bed partner may complain of being kicked by the patient. The patient may reveal a history of leg cramps, disheveled bedding, and even a history of falling out of bed.

A patient who complains of difficulty in falling asleep or even in relaxing because of unusual crawling sensations in the thighs and calves may be experiencing the *"restless legs" syndrome.* The dysesthesias often temporarily subside with exercise. Virtually all the patients also experience sleep-related myoclonus, although most of the patients with the latter condition do not also have the "restless legs" syndrome.

Disorders of Excessive Somnolence

As noted with sleep apnea, the patient's primary complaint may center on the expected period of wakefulness, when he or she experiences excessive sleepiness. The clinician must distinguish daytime sleepiness from the more generalized symptom of fatigue. Sleepiness refers to a difficulty in remaining awake during the hours when the patient would normally expect to be awake. There may be impairment of the patient's cognitive and motor performance, and there may be incomplete arousal on awakening. It is not clinically appropriate to apply the word sleepiness to the state of weariness that follows exertion or to those feelings of las-

situde, irritability, or impaired concentrating ability associated with inadequate duration of sleep.

The clinician may determine that the patient does indeed have excessive daytime sleepiness, which occurs even after the usual full night of sleeping. An organic basis underlies most such complaints. While a brain tumor or a CNS infection must be considered, most of these patients will have either *narcolepsy* or a *sleep apnea syndrome*. Historical data can help make the distinction.

What was the age of onset of the symptoms? Onset during the teens or 20s suggests narcolepsy. The patient may report a long history of social difficulties caused by excessive sleepiness. When the onset occurs beyond age 30, the probability of narcolepsy decreases. Hypersomnolence after age 40 would make the sleep apnea syndrome more likely. Yet some forms of sleep apnea can occur at any age.

Are the sleep attacks absolutely irresistible? Narcoleptic patients usually are unable to postpone an attack, whereas sleep apneic patients may be able to delay a sleep episode if absolutely necessary. Is the patient at least temporarily refreshed once he or she awakens from the episode? The narcoleptic patient usually can proceed alertly with various tasks such as driving and may have even learned to self-manage the problem by including several brief naps during the course of the day. In contrast, the sleep apneic patient awakens from longer naps still unrefreshed.

What is the quality of the patient's nighttime sleeping? Although many narcoleptic patients experience frequent nocturnal awakenings and/or nightmares, some may have relatively restful nighttime sleeping. Usually sleep apneic patients report restless nocturnal sleeping along with fatigue upon awakening in the morning. When a collateral history confirms the restlessness and describes marked nocturnal snoring, periods of apnea, or both, the diagnosis of sleep apnea syndrome becomes strengthened.

Does the patient experience any unusual phenomena associated with emotions? Many narcoleptics experience some degree of muscular helplessness in response to a variety of emotional experiences. This involves partial or complete paralysis of various antigravity muscles. When this phenomenon, cataplexy, is present, it strengthens the diagnosis of narcolepsy. In some patients, cataplexy may be present in the absence of excessive daytime sleepiness.

The clinician should seek two other phenomena of narcolepsy. While most normal people have experienced a mild, transient, generalized paralysis on awakening, this phenomenon is much more marked in the narcoleptic patient and usually occurs at sleep onset. In addition, dream images (hallucinations) may occur at sleep onset (hypnagogic) or on awakening (hypnopompic). Although most narcoleptic patients do not experience all the above features, the presence of the complete tetrad clinches the diagnosis on historical grounds alone. Automatic behavior is also experienced by about half of narcoleptic patients.

Recurrent daytime sleepiness in the absence of sleep attacks raises the remote possibility of another condition: *idiopathic CNS hypersomnolence*. Commonly associated symptoms include headaches, syncope, and the complaints of Raynaud's disease. Other family members often have similar symptoms. Some of these patients may manifest sleep drunkenness in which the process of awakening is prolonged and often associated with disturbed thinking and irrational behavior.

In any case of hypersomnia, it is important to elicit a thorough personality profile. Grief-associated situations may be associated with increased sleepiness, regarded as *psychophysiological*. This disorder is usually transient, but rarely it may persist as a disposition to weariness. Bipolar depression is characteristically associated with excessive daytime sleepiness. In these cases, the depressed patient extends increased nocturnal sleep into the daytime hours.

Of particular importance is a *drug and alcohol history*. Is the patient repeatedly using CNS depressants? An individual may be addicted to opiates, barbiturates, or other sleep-inducing drugs and may use these agents primarily for their somnolent effects. Other patients may unintentionally experience such effects when using moderate to high doses of medications intended for other therapeutic purposes (e.g., antihistamines, certain antihypertensive agents, beta-adrenergic blocking agents, and many psychotropic drugs). Elderly patients are particularly susceptible to the somnolent effects of these medications, just as they are to hypnotic medications intentionally used to counter insomnia. Furthermore, very large bedtime doses of CNS depressants may induce a sleep-associated impairment of ventilation known as the *alveolar hypoventilation syndrome*, which is not associated with significant apneic pauses. This condition can also occur in the absence of CNS depressants.

CNS stimulants may induce excessive somnolence. Although they are often taken to counter daytime fatigue, sleepiness often results when the effect of the drug wears off. Furthermore, as tolerance develops, the duration of action of the drug shortens, and "miniwithdrawals" may appear with associated sleepiness. Excessive sleepiness may also be noted when CNS stimulants are purposely withdrawn.

Patients who excessively use caffeine-containing substances may similarly develop tolerance and become progressively dependent on caffeine. The need for several cups of coffee to wake up in the morning may reflect caffeine withdrawal. Alcohol should not be overlooked as a possible etiologic agent in excessive somnolence.

The *general medical status* may contribute to hypersomnolence. Conditions to be considered include endocrinologic disturbances of the thyroid gland and pancreas, uremia, hepatic encephalopathy, and morbid obesity. Cheyne-Stokes respirations may be associated with multiple nocturnal miniarousals, resulting in excessive daytime sleepiness. The same may occur with nocturnal myoclonus or the "restless legs" syndrome.

Finally, some patients who require longer than the conventional amount of sleep for a given age may simply be at the high end of the continuum for requirements of normal sleep. These "long sleepers" do not complain of poor quality of sleep or of daytime sleepiness or of difficulties with daytime attitude or performance, provided their sleep is not abbreviated.

Disorders of Sleep–Wake Schedule

A sleep-related complaint may represent another cluster of disturbances: disorders of the sleep–wake schedule. To conceptualize these disorders, one must view sleep and wake as behaviors that are intimately related to physiological and societal processes. Furthermore, this relationship is superimposed on and influenced by the 24-hour day–night cycle inherent in the earth's rotation. Disorders of the sleep–wake schedule constitute a collection of syndromes that share in common the element of dyssynchrony between the behav-

iors of sleep and of wake, on the one hand, and the processes of human physiology and/or society's expectations, on the other, all within the context of the 24-hour day.

This class of disorders may be transient or persistent. With regard to *transient* disorders, it is important to inquire about recent long-distance travel by air across different time zones. This "jet lag" syndrome occurs as a result of the individual's usual desire immediately to set his or her sleep–wake schedule according to the local time cues of the destination. Since physiological functions other than sleep and wake may take a week or so to adapt, a transient misalignment occurs between the internal sleep–wake phases and the clock times for sleeping and waking. The result is a syndrome characterized by insomnia during the desired sleep period and sleepiness and impaired physical and mental functioning during the desired wake period. The rapid time zone change syndrome is usually prolonged after eastward flights, is often less severe after the return trip to the time zone of origin, and does not occur after north–south jet travel if there is no more than a 1- or 2-hour change in time zone.

An equivalent syndrome is termed the "work shift" change in conventional sleep–wake schedule. Symptoms in this case, instead of resulting from an attempt to adapt to local time cues, arise from adopting a sleep–wake pattern that runs counter to the local schedule. The result is impairment of the wake period and the sleep period.

A *persistent* disorder may result when the sleep–wake schedule is changed frequently. The ensuing clinical picture combines elements of both the insomnias and the disorders of excessive somnolence. Sleep periods are usually shortened and disrupted, performance during the desired waking state is impaired, and temporary opportunities to revert to a regular sleep schedule are unsuccessful.

Several questions may help correlate the frequently changing sleep–wake schedule with the severity of the symptoms: With each change, how many hours of shifting are involved? How frequently do the shifts occur? What is the interval between changes in schedule? Changing of shifts at intervals of less than 3 to 4 weeks superimposes new adjustment demands on physiological processes that have still only incompletely adapted to the previous shift.

Another key historical piece of information is the hour at which the patient usually goes to bed. Especially in the elderly, a very early bedtime hour may account for early morning awakening, which the patient interprets as disturbed sleep. In contrast, young adults often delay sleep until after midnight and then sleep until around the noon hour. In either case, it may be difficult to adjust the sleeping pattern when circumstances demand more conventional sleeping hours.

When the clinician encounters a patient in whom paired sleep onset and wake times are not only either earlier or later than desired, but intractably so, the respective diagnoses of *advanced sleep phase syndrome* or *delayed sleep phase syndrome* should be entertained. These patients may complain about the social or occupational conflicts that result from an apparently uncontrollable lack of correlation between the patient's physiologic clock and the usual time clock. In these disorders the patient has no difficulty in falling asleep whenever the natural urge to do so is followed; when circumstances allow, the sleep is refreshing and of sufficient duration.

A patient may present with the complaint of insomnia or of excessive daytime sleepiness that is periodic. In such a case, a log of the sleep and awakening times over several weeks should display the pattern of the phenomenon and in some cases reveal that the patient is retiring for the night at a progressively later hour. This may indicate that the periodicity of the patient's circadian rhythm is appreciably longer than the usual 24 or 25 hours, constituting the *non-24-hour sleep–wake syndrome*. The periodicity of the patient's complaint reflects the fact that as the patient's internal physiological sleep–wake clock progressively moves through the 24-hour day, it will temporarily coincide with the conventional clock. However, the prolonged periodicity of the patient's physiological clock will again get out of synchrony with society's, and the patient will again complain of difficulty in getting to sleep at the desired time and of excessive daytime somnolence.

Disorders Associated with Sleep, Sleep Stages, or Partial Arousals (Parasomnias)

Clinicians occasionally encounter patients whose complaints deal with disturbing events associated with sleep, sleep stages, or partial arousals. Although these parasomnias constitute perhaps the most common group of sleep disorders found in children, occasionally adults present with these disorders. The fact that the various complaints usually relate to skeletal muscle phenomena or to events of the autonomic nervous system reflects sleep-associated activation of the CNS.

Disturbing dreams may indicate either *nightmares* or *sleep terrors*. In what part of the night does the disturbance occur? Occurrence within the first 2 hours after sleep onset makes the diagnosis of sleep terror more likely. These episodes usually begin with a scream, followed by signs of distress such as sweating, rapid breathing and heart rate, and seemingly random body movements. Household members usually report difficulty awakening the patient during these episodes. However, normal sleep seems to return spontaneously within a few minutes.

When the disturbing dreams occur in the latter part of the night, nightmares (dream anxiety attacks) are more likely. An exception to this timing is a more recently recognized type of nightmare in which frightening dream imagery appears as the patient is passing from wakefulness to sleep. These phenomena may be part of a posttraumatic stress syndrome, or they may be an early manifestation of a psychotic disorder.

Can the patient recall the dream content? The patient who can give a relatively detailed account of the disturbing dreams is more likely to be experiencing nightmares. The patient with sleep terrors will usually have no recollection of those episodes.

The complaint of nightmares should prompt questions about the patient's use of medications (e.g., reserpine). Nightmares may be precipitated by abrupt withdrawal from hypnotics or CNS stimulants or alcohol. Unrelated to drugs, sleep deprivation itself can at times induce nightmares.

Another sleep disturbance that usually occurs during the first part of sleep is *sleepwalking* (somnambulism). Here again, there is a difficulty in arousing a patient during the episodes and a subsequent lack of recollection of the episodes by the patient; hence, the importance of a collateral history.

Frequent sleepwalking in an adult often suggests an emotional disturbance. A drug history is important, since hypnotics or alcohol can either induce this disorder or make it

more severe. Prolonged periods of voluntary wakefulness can also exacerbate the disorder.

The clinician should be familiar with other parasomnias. *Bedwetting* (sleep-related enuresis), perhaps the best known of these, is only rarely a presenting complaint of adults. In such cases, the clinician needs to consider the possibility of underlying conditions such as a seizure disorder, neurogenic bladder, stress urinary incontinence, or a lower urinary tract infection. Usually the patient's primary complaint deals with these underlying conditions rather than the bedwetting itself.

Other sleep-associated dysfunctions may be manifested by a variety of other complaints. Headaches may be secondary to teeth grinding (bruxism). The history of repeated abrupt awakenings in the middle of the night with severe unilateral frontal headaches, often associated with ipsilateral lacrimation and rhinorrhea, is very typical of cluster headaches. Asthmatics may have attack-related dreams. The worsening of cardiovascular symptoms and the appearance of arrhythmias can occur during sleep. Some patients may be awakened from sleep because of the heartburn that results from gastroesophageal reflux. In some patients, even in the absence of heartburn, there may be multiple miniarousals of which they are unaware but which may occur as a result of delayed clearing of acid from the esophagus during sleep. These are just some of the many examples that underscore the impact of sleep on the central nervous system in general and the autonomic nervous system in particular.

Basic Science

Normal Sleep

In recent years sleep has been defined largely in terms of electrical phenomena recorded during clinical sleep—brain wave activity by the electroencephalogram (EEG), eye movements by the electrooculogram (EOG), and facial muscular activity by the transcutaneous electromyogram (EMG). These three study techniques constitute the essentials of polysomnography and allow for the somewhat arbitrary but useful staging of the sleep–wake cycle. Although the stages are defined primarily by the characteristics of the EEG, the features of the EOG and EMG are often more useful in defining transitions between stages. On the basis of polysomnographic studies, the basic sleep–wake cycle has been categorized into three different states: wakefulness, rapid eye movement (REM) sleep, and non-rapid eye movement (NREM) sleep.

In the waking state, the EEG typically demonstrates low voltage and frequencies in the range of 4 to 25 Hz, or cycles per second. Clusters with a frequency range of 4 to 7 Hz are known as *theta waves*. Sinusoidal waves in the range of 8 to 13 Hz constitute *alpha rhythm*, which usually occurs when the patient is relaxed with eyes closed but still awake. Clusters with a frequency greater than 13 Hz are termed *beta rhythm*. The waking EMG reveals a certain baseline tone of the facial muscles. The waking EOG, even with the patient resting, reveals quick, conjugate eye movements.

NREM sleep, which usually follows wakefulness, is separated into four stages. *Stage I* is marked by an EEG loss of alpha rhythm, a gradual generalized diminishing of EEG voltage, and the subsequent appearance of theta activity. Eye movements, the most reliable sign of stage I, become slow, random, circular, and at times disconjugate. The facial muscles become slightly relaxed. It is in stage I (drowsiness) that the sleeping patient is most easily aroused. Daydreams may occur.

Stage II (light sleep or spindle sleep) is characterized by the EEG findings of rhythmical, spindle-shaped clusters in the frequency range of 10 to 16 Hz. These may be accompanied by slow, blunted, high voltage, biphasic waves (K-complexes), frequently associated with hypnagogic myoclonus. In stage II sleep, vertex (or V) waves may indicate nonspecific arousal. During this stage, eye movements are usually quiet, and the electrical activity of the facial muscles diminishes further.

Stages III and IV are known as slow wave sleep. Stage III is usually said to be present once delta waves (1 to 3 Hz) constitute at least 20% of an EEG epoch and have an amplitude of 75 μV or greater. Hence, this stage of sleep (as well as stage IV) is often also referred to as delta sleep. Once delta waves constitute 50% or more of an epoch, stage IV is said to be present. This distinction is somewhat arbitrary and should be regarded as biologically insignificant. During slow wave sleep, eye movements are usually absent and muscle tone is similar to that in stage II sleep. Marked resistance to arousal is a clinical feature of slow wave sleep; hence, the synonym "deep sleep."

The EEG findings of *REM sleep* include a relatively low voltage and a random, mixed-frequency spectrum in the range of 4 to 25 Hz. There are usually clusters of rapid, conjugate eye movements. The synonym "paradoxical sleep" derives from the contrast of these bursts of eye movements and phasic body movements with a loss of postural tone and resistance to arousal. Since it is during REM sleep that the most vivid dreams occur, the term "dream sleep" also applies.

Normal sleep is initiated by a sequential progression of NREM sleep from stages I through IV, followed typically by a return to stage II sleep, which usually, even if only briefly, precedes the initial and subsequent periods of REM sleep. REM cycles, the time from the start of one REM period to the start of the next, last about 90 minutes. Since most REM periods lengthen as the night progresses, most of the REM sleep occurs during the second half of the night, whereas the predominant sleep of the first half is slow wave. It is in mostly the latter form of sleep that prolonged periods of body immobility occur, which presumably give rise to the feelings of relaxation often associated with sleep.

At all ages, REM sleep accounts for 20 to 25% of the total sleep time, whereas the remainder of the sleep time is divided among the various stages of NREM sleep. However, the proportion of sleep in the various NREM stages differs with age.

The duration of nocturnal sleep is fairly well maintained between ages 20 and 60. Older people spend less of their night in the sleeping state. Despite this, persons over age 60 are similar to younger adults in the total average amount of time spent sleeping during a typical 24-hour period. That average of 7.5 hours is reached by the older person's supplementing the lessened amount of nocturnal sleep with daytime naps and/or "microsleeps," which last from 1 to 10 seconds and are associated with a lowering of the eyelids.

Abnormal Sleep

The microsleeps so typical of the elderly also appear in younger individuals when they are progressively deprived of sleep. Even before the appearance of microsleeps, shorter daytime sleep onset latencies are noted as a lack of nocturnal

sleep becomes progressive. Furthermore, studies in which deprivation of a particular stage of sleep is produced have demonstrated that once sleep is subsequently freely allowed, an excess of that deprived stage will occur. This is particularly true of REM sleep in which not only will there be a subsequent rebound in the amount of REM sleep but it will also become increasingly difficult to suppress that sleep state.

The above phenomenon, known as *REM pressure,* also can become operative whenever pharmacologic suppressants of REM sleep are used. Such substances (e.g., tricyclic antidepressants, amphetamines, and certain hypnotics) initially suppress the onset and duration of REM sleep. Because of REM pressure, the normal amount of REM sleep will subsequently return despite continued use of the substance. However, the discontinuance of the substance does not immediately shut off REM pressure. The result is REM rebound—an increase in the frequency, duration, and intensity of REM periods, often experienced as nightmares and as generally disturbed sleep.

This same phenomenon occurs with the abrupt cessation of alcohol following its chronic use. REM deprivation during alcohol consumption leads to exaggerated levels of REM sleep once alcohol is stopped. This REM rebound is antecedent to delerium tremens.

Apart from any consideration of drug use, familiarity with the various stages of sleep fosters an enhanced understanding of sleep-related enuresis, sleepwalking, sleep terrors, and nightmares. The first three disorders, which probably represent partial arousals, occur in slow wave sleep. The association with this sleep stage is consistent with the usual occurrence of these phenomena during the first third of the night, when slow wave sleep is most predominant. Consistent with the "deep sleep" synonym for this stage is that the individual resists arousal during each of these first three disorders and usually does not recall the episodes.

On the other hand, nightmares typically involve an awakening from REM sleep. Their more frequent occurrence in the mid to late segment of nocturnal sleep is in keeping with the increased proportion of REM sleep during the second half of the night. Also, the ability to recall nightmares is consistent with the vivid dream imagery of REM sleep. The integration of emotionally important information may be one of the functions of dreaming and REM sleep. Conceivably, a nightmare may represent failure of this integrative process.

There are a few other disorders in which the basic polysomnographic findings are of particular clinical interest. For non-bipolar depression, short REM sleep latency (the time from initial stage II to the onset of REM sleep) is now accepted as a biologic marker. REM sleep latency is even shorter in narcolepsy, which may represent an imbalance between the wake, NREM, and REM states. The REM system continually intrudes upon the wake state. Accordingly, polysomnographic documentation of narcolepsy typically involves the demonstration of abnormalities of daytime sleep rather than primarily nighttime sleep. Characteristic of narcolepsy is the appearance of REM sleep within 10 minutes of sleep onset. In contrast, idiopathic CNS hypersomnolence does not involve sleep-onset REM periods.

With regard to the auxiliary symptoms of narcolepsy, hypnagogic hallucinations likely represent REM dreaming prior to the loss of consciousness. Sleep-onset paralysis reflects the inhibitory processes of REM sleep. In cataplexy, the muscular paralysis aspect of REM sleep occurs in response to emotional arousal while the patient is fully conscious. On occasion, cataplexy can evolve into a complete REM sleep episode. The automatic behavior of many narcoleptics may amount to daytime sleepwalking occurring during repetitive microsleep episodes.

Clinical Significance

Sleep disturbances—as distinguished from sleep and arousal disorders per se—should be regarded as symptoms. They consist of a broad range of clinical presentations that involve a variety of physiologic, emotional, and behavioral abnormalities. Consistent with the 24-hour day–night cycle, these abnormalities often do not confine themselves to the sleep period and frequently manifest themselves primarily during the waking hours. Consequently, the clinician must also maintain a vigilance for the possibility of sleep and arousal disorders in considering many of the patient's daytime complaints. To explore such considerations, competence in history taking is essential. Polysomnography will be appropriate for only a small minority of patients, and even in these cases, such studies usually will serve only to confirm what the clinician already suspects on the basis of history.

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