In H.E. Adams & P.B. Sutker (Eds.) (in press), Comprehensive Handbook of Psychopathology, 3rd Ed. New York: Plenum Press.

SLEEP DISORDERS

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During the past 15 years there has been an explosion of research on sleep and sleep disorders. Much has been learned about the causes and treatment of sleep disorders including insomnia, sleep apnea, and narcolepsy. In addition, alterations in sleep patterns are so commonly associated with some disorders, such as affective disorders, posttraumatic stress disorder, and fibromyalgia, that they are used as diagnostic criteria for those disorders. More generally, we are all affected by the amount and quality of the sleep we obtain. Sleep has broad, systemic effects on mood, performance, and physical functioning. Thus, an understanding of sleep is essential to a complete understanding of normal and abnormal behavior.

The current chapter is a revision of Bootzin, Manber, Perlis, Salvio, and Wyatt (1993). References to research that appeared since the last chapter was written have been added; some sections have been substantially rewritten, new sections have been added, and some sections have been deleted. This chapter must, by necessity, be selective. Sources for additional information are the International Classification of Sleep Disorders, Revised: Diagnostic and Coding Manual (American Sleep Disorders Association, ASDA, 1997) and Principles and Practice of Sleep Medicine, 2nd ed. (Kryger, Roth, & Dement, 1994; the third edition is in press and will be available in 2000).

BASIC SLEEP INFORMATION

Sleep must be understood within the context of the 24-hour day. There is now substantial evidence that sleep-wake behavior has an endogenous rhythm; that is, a rhythm that persists in the absence of environmental cues. Core body temperature is used as a means of measuring the

underlying sleep-wake circadian rhythm. Within the normal range, higher core body temperatures are associated with alertness and lower temperatures are associated with sleep and sleepiness.

The presence of an endogenous sleep-wake rhythm of 24.2 to 25.1, rather than 24, hours has been observed under free-running conditions in which participants were kept in isolation from cues regarding the time of day (Czeisler, et al., 1999). Under normal circumstances, environmental influences, such as sunlight, meals, and social activity, entrain the endogenous rhythm to a 24-hour cycle. The combination of bright light during wake and total darkness during sleep is a particularly strong entraining influence and can facilitate adjustment to changes in sleep schedules (Czeisler, et al., 1989).

The fact that the endogenous sleep circadian rhythm is longer than 24 hours may help explain some findings regarding jet lag. It has been observed that it takes twice as long to adjust to time changes produced by eastward than westward travel (Moore-Ede, Sulzman, & Fuller, 1982). When traveling east, one is shortening the day, thereby increasing the difference between the endogenous sleep-wake rhythm and the new time zone.

Electrophysiology of Sleep

It is possible to distinguish sleep from wakefulness based solely upon behavioral criteria. There are and stereotypical postures associated with sleep periods, reductions in responsiveness to the environment, and reductions in activity,. These distinctions between sleep and wake can be seen throughout the phylogenetic scale from insects through mammals (Amlaner & Ball, 1994; Hartse, 1994; Zepelin, 1994). In birds and mammals, there are also detectable changes in brain activity associated with sleep.

Sleep recording in humans, called polysomnography, is scored using information from the electroencephalogram (EEG), eye-movements (EOG), and muscle activity from the chin (EMG) following the Rechtshaffen and Kales (1968) scoring manual. When an individual is awake, the EEG is characterized by a low voltage, fast frequency signal. This signal is dominated by beta (14-20 Hz) and alpha (8-12 Hz) EEG frequencies. As one becomes drowsy, the EEG pattern slows down to a slightly higher amplitude theta rhythm (3-7.5 Hz). If more than 50 percent of an

EEG epoch of 20 or 30 seconds consists of theta, the individual is considered to be in stage 1 sleep. Stage 1, a transitional stage, may also contain slow, rolling, eye-movements. Although the first epoch of stage 1 sleep is often used to operationally define sleep onset, individuals aroused from stage 1 usually report that they were about to fall asleep. A more conservative criterion for sleep onset is the appearance of stage 2 sleep.

Stage 2 sleep occurs when certain phasic events, sleep spindles and K-complexes, appear in the EEG. Sleep spindles, named for their sewing-spindle appearance, are short bursts of 12-14 Hz activity and are thought to be due to inhibitory neuronal activity during sleep. K-complexes are slow, high amplitude voltage bursts that consist of a large negative deflection followed by a large positive deflection and are thought to be brain responses to external or internal stimuli. The appearance of high amplitude, slow delta waves (.5-2.5 Hz) marks the presence of stages 3 and 4, also called slow wave sleep. Stage 3 contains 20 to 50 percent delta waves in an epoch and stage 4 has more than 50%. The progression from stage 1 to stage 4 sleep is characterized by decreases in frequency and increases in amplitude of the EEG. Arousal thresholds to external stimuli increase with increases in sleep stage.

Rapid-eye-movement (REM) sleep is an activated stage of sleep that occurs cyclically, about every 90 minutes throughout the night, and is associated with dreaming. Reports of cognition obtained from REM sleep are longer, more visual, more story-like, and more bizarre than are reports from stages 1 through 4 (NREM sleep) which are similar to reports of waking cognition. REM sleep is also quite different physiologically from NREM sleep. In addition to rapid-eye-movements, there is postural muscle atonia, irregular respiration, increased heartrate, disrupted temperature regulation, and penile erections during REM sleep.

As seen in Figure 1, most slow-wave sleep occurs during the first half of the night. As the

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night progresses, REM sleep periods are longer. Thus, more REM sleep occurs during the last half, than the first half, of the sleep period.

Sleep Deprivation

The short term effects of one or two days of sleep deprivation are increased irritability, deficits in sustained attention, and sleepiness, particularly at night . With longer periods of sleep deprivation, there is an increase in sleepiness, a loss in fine motor control, and the appearance of mild flu-like symptoms. Extended periods of partial sleep deprivation can also lead to decrements in vigilance performance, and objective, as well as subjective, measures of sleepiness (Dinges, et al., 1997). Recovery sleep following three or four nights of sleep deprivation consists mostly of slow wave and REM sleep. Most of the lost slow wave sleep and about half of the lost REM sleep are made-up during recovery nights (Horne, 1988).

Circadian sleep-wake and temperature cycles persist even during sleep deprivation. Thus, sleep-deprived individuals are more alert during the hours they would normally be awake and more sleepy when they would normally be asleep. Many industrial accidents such as Three-Mile-Island and Chernobyl occurred during the night shift at times when individuals would be sleepiest.

Developmental Changes in Sleep

Sleep, its duration, architecture, and diurnal distribution, changes across the human lifespan. These developmental changes in sleep parameters are accompanied by corresponding changes in the types and prevalence rates of sleep disorders. Childhood sleep problems are usually transient, but when they do persist they may influence the child's daytime functioning and impact upon other family members.

A neonate's sleep is comprised of active and quiet phases, similar but not identical to REM and NREM sleep seen in older children and adults. During the first six months of life, slow waves become more continuous, sleep spindles and K-complexes emerge, and the four stages characterizing adult NREM sleep become distinguishable (Williams, et al., 1974). During the same time period, there are changes in the distribution and duration of sleep stages. The percent of

time spent in REM sleep decreases from 50 to 30 percent, and the latency to REM sleep, which is zero minutes at birth, gradually increases and starts to exhibit circadian rhythmicity (Schulz, et al., 1983). The percent of REM sleep gradually decreases during childhood and reaches the adult level of 20 to 25 percent at adolescence. The number of sleep cycles decreases from a childhood level of 7 cycles to 4 or 5 cycles during adolescence (Karacan, et al., 1975), a fact reflecting both a lengthening of the sleep cycle and a shortening of total sleep duration.

The amount of slow wave sleep increases in the first 3 years of life, stays constant until the teens, and then gradually declines. Beginning in the teens, the amplitude of slow waves declines with age (Smith, et al., 1977). These changes in slow wave sleep, as individuals age, are associated with a lightening in depth of sleep, as measured by arousal thresholds and the frequency of brief arousals from sleep.

Infants and toddlers. The total duration of sleep declines sharply during the first year of life from about 17 hours at birth to about 13 hours by the end of the first year and continues to decline gradually to about 11 hours at age 2. By the fourth month of life, the circadian sleep-wake cycle becomes more prominent and more synchronized with the light-dark cycle (Harper, et al., 1981). The changes in sleep duration are accompanied by changes in the distribution of sleep throughout the day. By six months, most infants have one extended period of sleep at night and two naps.

The most common parental concerns about their children's sleep during infancy and toddlerhood are difficulties in settling to sleep and waking up at night. Treatments are commonly based on stimulus control principles. Parents are informed about the importance of a consistent and relaxing bedtime routine and on the importance of teaching their children to fall asleep on their own so that the parent's presence will not become a distinguishing stimulus for sleep onset. Often, treatment involves the extinction of old habits and the establishment of a new and consistent bedtime routine. Extinction of parental presence in the room at bedtime can be accomplished abruptly or by successive approximations in which parents gradually decrease the interval of contact time with their children at bedtime (Ferber, 1995). Estimates of the efficacy of this

approach are very high (Adair et al, 1992; Durand & Mindell, 1990; Richman, et al., 1985; Sadeh, 1994) and its benefits often generalize beyond the sleep problem (Minde, Faucon, & Johnson, 1994). When the infant's or toddler's sleep difficulties are a manifestation of separation anxiety, an alternative and effective approach is for a parent to sleep in the child's bedroom for about a week avoiding interactions with the child during the night (Sadeh, 1994). Some form of co-sleeping with infants (sharing the same room or sharing the same bed) is customary in the majority of the world's cultures. The impact on the mother's sleep indicates a small reduction in slow-wave sleep and an increase in arousal frequency (Mosko, Richard, & McKenna, 1997) allowing for increased monitoring of the infant.

Preschool and early childhood. Sleep problems common in this age group are those associated with initiating and maintaining sleep and the parasomnias. Results of prospective investigations suggest that childhood complaints about sleep onset and sleep maintenance difficulties are transient in nature (Strauch & Meier, 1988). However, Hauri and Olmstead (1980) found that childhood insomnia does in some cases persist into adulthood. They coined the term childhood onset insomnia to describe adult insomniacs whose problems started before age 10.

Sleep onset difficulties during early childhood may be manifested as bedtime struggle stemming from oppositional struggle with parents, fear of going to sleep, or difficulty in settling down. Distress at bedtime may be related to developmental changes affecting the prevalence of fears in children (Ollendick, et al., 1985). One treatment approach for cases in which fear and anxiety are central to the child's difficulty in initiating sleep consists of cognitive rehearsal of appropriate bedtime behaviors during the day and positive reinforcement in the morning (Cashman & McCann, 1988). Children's anxiety at bedtime may also benefit from a relaxation routine designed to match their cognitive and attention level. Relaxation routines designed for use with children typically include imagery such as a melting snowmen as well as tense-release instructions (Oaklander, 1988; Ollendick & Cerny, 1981; Schumann, 1981). Older children may be helped by a comprehensive intervention based on self-management strategies which include relaxation, self-statements, self-assessment, and self-reinforcement (Graziano & Mooney, 1982). Sleep onset

problems that are judged to stem primarily from oppositional factors are commonly treated by positive reinforcement or by paradoxical intention. When oppositional struggles mark daytime behavior, treatment focuses on the most urgent oppositional problem which often results in improvement at bedtime as well (Cashman & McCann, 1988). Stimulus control instructions (see below) can be tailored to school aged children when poor association between the bed and sleep are apparent.

The prevalence rate of sleep maintenance problems decreases sharply from 30 to 35 percent at ages 4 to 5 to about 15 percent at age 6 and continues to decline to 8 percent at age 10 (Klackenberg, 1982). Sleep maintenance problems during the preschool years are often found in conjunction with sleep onset problems and tend to disappear as the latter problems are solved (Mindell, 1990).

Parasomnias, including sleep walking, sleep talking, night terrors, and enuresis, are most prevalent in children aged 5 to 12. The presence of parasomnias beyond the teens is commonly associated with psychopathology. Parasomnias are discussed in detail later in the chapter.

The second decade of life. Physical and endocrinological maturation associated with puberty may result in an increase in sleep need (Carskadon & Dement, 1987). Nevertheless, there is a decline in total sleep time from 10 to 7 hours per night from pre- to late-puberty. The reduction of total sleep time reflects the increasing demands on adolescents' time through school, work, and social interactions.

Epidemiological studies examining sleep-wake problems in adolescence found that the most common complaint is daytime sleepiness (Carskadon & Dement, 1987; Strauch & Meier, 1988). More than half of the adolescents surveyed complained of daytime sleepiness. The prevalence rate of daytime sleepiness, which is very low at preadolescence (Palm, et al., 1989), increases with the onset of puberty and peaks at mid-puberty.

Another common, though less prevalent, complaint is frequent or severe insomnia reported by about 12 to 14 percent of adolescents (Price, et al., 1978; White, et al., 1980). The possibility that adolescent sleep onset difficulties may reflect a mild form of circadian rhythm disturbance was

suggested by Henschel and Lack (1987). Life-style patterns typical in adolescence are likely to contribute to irregular sleep-wake schedules and to sleep deprivation on school nights. Teenagers who work after school and who have a heavy load of extracurricular activities have been found to be particularly sleepy during the day (Carskadon, Mancuso, & Rosekind, 1989). Nevertheless, in addition to psychosocial factors, the tendency to delay sleep does in some cases appear to be related to the biological pubertal process itself (Carskadon, Viera, & Acebo, 1993).

Sleep deprivation during adolescence may result in irritability, vulnerability to minor illness, inability to sustain attention, sleepiness, and fewer personal resources being available to deal with emotional stresses. In addition, emotional stresses and alcohol and drug abuse are likely to amplify the effects of sleep deprivation.

To help meet the demands of adolescence and reduce sleepiness, teenagers should be encouraged to get more regular and longer durations of sleep (Ferber, 1990). The treatment of severe daytime sleepiness due to intrinsic sleep disorders such as narcolepsy and sleep apnea are discussed later. The treatment of disturbed sleep during adolescence is similar its treatment during adulthood and is outlined later in this chapter.

Adults and the elderly. From early adulthood to old age sleep continues to change due to normal aging, psychosocial/lifestyle factors, and concomitant medical and/or psychiatric conditions (Vitiello, 1997). While for most individuals their sleep need remains constant throughout adulthood, their ability to maintain uninterrupted sleep, unfortunately, diminishes with age (Ancoli-Isreal, 1997). The increased sleep disturbance at night often leads to increased daytime sleepiness and fatigue. People differ in the amount of sleep changes associated with aging; some show little change in their sleep pattern, while others experience dramatic deterioration (Webb, 1992).

The nightly amounts of total sleep, stage 4 sleep, and REM sleep level off at puberty, remain stable throughout adulthood, and decrease with advancing age (Smallwood, Vitiello, Giblin, & Prinz, 1983). Gross sleep architecture also remains relatively stable, comprised of approximately 70% to 75% of NREM sleep and 20-25% of REM sleep relative to total sleep

throughout adulthood (Carskadon & Dement, 1994). There are some differences in monthly sleep patterns between men and women that are likely related to hormonal changes associated with the menstrual cycle. As many as 15% of women experience a significant increase in sleep disturbance in the late luteal or premenstrual part of their cycles (Manber & Armitage, 1999).

It is during the middle age years that most people first notice a shallowing of sleep along with a growing sense of fatiguing more easily in the daytime. Sleep becomes less continuous beginning in middle age and becomes progressively more fragmented with advancing age and declining health. Of all the age-related changes in sleep, the reduction of SWS is the most prominent. With the reduction in SWS sleep, there is a corresponding increase in the lighter stages of non-REM sleep, especially Stage 1, and increased time spent awake during the nocturnal sleep period. A pattern of lighter, more fragmented sleep with frequent and prolonged awakenings is common in the elderly (Webb & Campbell, 1980; Bliwise, 1994). Thus, the elderly get less total sleep at night than their younger counterparts, although they may spend more time in bed (Dement, Miles, & Carskadon, 1982). When sleep across the entire 24-hour day is accounted for (including naps), the elderly get about the same amount of total sleep as middle-aged adults.

The effect of aging differs in men and women. This gender difference emerges between ages 30 and 40. While men and women in their twenties have similar percentages of slow-wave sleep (SWS), a significant reduction in the percentage of SWS occurs in men during their thirties but not in women (Ehlers & Kupfer, 1997). Objective measures of sleep reveal that the sleep of women tends to be better preserved with aging than that of men. In contrast, subjective rating of sleep is worse in elderly women than that of elderly men as the former report more sleep complaints than the latter (Rediehs, Reis & Creason, 1990).

There are many biological and psychological consequences of aging that may affect sleep, either directly or indirectly. Biological processes include decreased bladder control, the presence of degenerative diseases, dementing disorders, poor health, and the presence of sleep disorders such as sleep apnea and periodic leg movements. Psychological processes include bereavement, anxiety about health and financial security, and enforced changes from daily routine (Morgan,

1987). While a common assumption has been that depression, anxiety, and poor health account for many instances of poor sleep in the elderly, research on the healthy elderly has found that frequent awakenings and decreased amounts of stage 4 and REM sleep occur even in the absence of depression, anxiety, or overt disease processes (Feinberg, Koresko, & Heller, 1967; Prinz, et al., 1984).

The deterioration of sleep that parallels aging can also be a direct result of sleep disorders such as sleep apnea (see later section) and periodic limb movement disorder (PLMs; see later section. Sleep apnea and PLMs, which can be primary or secondary to other medical conditions, are increasingly prevalent with age and affect a high percentage of the geriatric population (Carskadon, van den Hoed, & Dement, 1980), with PLMs occurring in up to 44% of residents of nursing home (Ancoli-Israel, Kripke, Mason, & Massin, 1981). The rate of PLMs in the aged population is due in part to the high prevalence of renal disease, arthritis, diabetic neuropathy, iron deficiency anemia, and venous insufficiency in this population, as PLMs are often associated with theses disorders (Bliwise, 1994).

Some researchers believe that age-dependent changes in circadian rhythm also contribute to the gradual deterioration of sleep at night and the increased propensity to nap during the day seen in the elderly (Bliwise, 1993). The sleep-wake pattern of many elderly, especially those in nursing homes resembles the polyphasic sleep wake cycle of infancy and may be secondary to the weak entrainment of the sleep-wake cycle into the circadian rhythm that occurs in these institutions (Dement, Miles, & Carskadon, 1982; Schnelle, Cruise, Alessi et al. 1998). The most commonly described circadian rhythm disturbances in the elderly is a gradual phase advance of the sleep period relative to the desired sleep time (see section on advanced sleep phase syndrome). This phase advanced rhythm results in early-evening drowsiness and sleep, with concurrent early-morning awakening and difficulty returning back sleep. (Miles & Dement, 1980; Czeisler et al., 1986; Weitzman et al., 1982). Although the elderly show a clear phase advance of their circadian rhythms, this may not be due to differences between elderly and younger adults in the length of the endogenous daily rhythm. A recent study found no differences in age in the length of the

endogenous rhythm when no light cues were available (Czeisler, et al., 1999). Age differences in sleep are more likely due to changes in sleep/wake schedules and changes in sleep architecture such as increased frequency of awakenings.

While naps are seldom reported in the 20-year-old working population, nearly all 60-year-olds report some napping, averaging approximately 2 naps per week (Webb, 1992). Two factors that may lead to daytime napping in the elderly are (a) attempts to compensate for lost sleep, and (b) understimulation and weakening of social constraints (Morgan, 1987). In addition, napping may not interfere with nighttime sleep in the elderly to the same extent that it does in young adults (Feinberg et al., 1985; Morin & Gramling, 1989; see section on sleep-wake schedules). Although napping can be adaptive as it can supplement nocturnal sleep, excessive napping can disrupt the circadian entrainment of the sleep-wake cycle exacerbating nocturnal sleep disturbance.

Changes in sleep patterns in the elderly do not necessarily lead to the subjective experience of insomnia. Some complain about sleep problems, while others notice but do not complain, and still others do not notice. Morin and Gramling (1989) compared self-identified poor and good older sleepers on measures of sleep, mood, life-style, health, and sleep requirement expectations and found that the poor sleepers showed greater discrepancies between their current sleep patterns and expectations and acknowledged more depression and anxiety than did the good sleepers.

There are many biological and psychological consequences of aging that may affect sleep, either directly or indirectly. Practice of good sleep hygiene, such as keeping a regular sleep-wake schedule, avoiding alcohol, tobacco, and caffeine, reducing excessive time awake in bed, and treating underlying sleep disorders will facilitate the preservation of good sleep.

Assessment of Sleep and Sleepiness

To identify sleep disorders, it is necessary to have reliable and valid measures of sleep and sleepiness. Although substantial information about sleep can be obtained from a detailed history, more direct assessment is usually desirable. There are many methods to choose from, and each method has advantages and disadvantages.

Assessment of Sleep Variables

Polysomnography. As mentioned earlier, the progression of sleep stages throughout the night as well as transitions from wakefulness to sleep are typically assessed by all-night recording of brain wave activities at one or more sites (EEG), and by monitoring eye-movements (EOG) and muscle activity from the chin (EMG). Additional physiological measures may be used for special purposes such as the evaluation of sleep apnea and periodic leg movements. The evaluation of sleep apnea requires measurement of respiratory effort, air flow, and blood oxygen saturation. Periodic leg movements are measured by recording EMG from the anterior tibialis muscles of the legs.

Several factors indicate the need for more than one night of recording in insomnia. Among these factors are the presence of night-to-night variability in sleep parameters and the fact that staying in an unfamiliar sleep environment, such as a sleep laboratory, may result in disrupted sleep. This is often called a "first-night effect". In addition, rare events, such as sleep terrors, may not be observed in a single night of recording.

Polysomnographic recording can be done in the sleep laboratory or at home. The effects of a unfamiliar setting on sleep can be reduced by the use of home recording (Palm, et al., 1989). Home recording can be accomplished either by telephone transmission of the recording signals to the laboratory or by recording the signals on magnetic tape to be analyzed later.

Auditory Event-Related Potentials. Neurophysiological assessment of sleep and waking states may also be conducted using auditory event-related potential (ERP) measurements. ERPs are micro-electrical EEG events that are elicited by and time-locked to the presentation of a stimulus. The ERP is comprised of a series of positive and negative-going waves (components) labeled either sequentially or according to their peak amplitude latency (i.e., a positive wave at 300 ms is labeled the 'P300'). The waking auditory ERP is comprised of a series of components which, labeled sequentially, are the: P1, N1, P2, N2, and P3. The onset of stage 1 sleep is associated with increased amplitudes for the P1, P2, and N2 components, whereas the N1 and P3 amplitudes are diminished (Campbell, Bastien, & Bell, 1992). During sleep stages 2, 3, and 4, the amplitude and latency of the N2 increase dramatically and a large amplitude, non-REM-sleep-

specific wave complex emerges called the 'evoked' K-Complex (N550-P900). ERP component structure in REM sleep resembles waking, however, amplitudes are reduced.

Auditory ERPs show reliable variations with states of consciousness and levels of cognitive processing. ERPs have shown enhanced cognitive processing in insomniacs, relative to good sleepers, during evening wakefulness and initial stage 2 sleep (Loewy & Bootzin, 1998). Cognitive impairment in healthy adults, due to prolonged sleep deprivation, is positively related to reductions in P300 amplitude (Morris, So, Lee, Lash, & Becker, 1992).

Activity monitoring. When the focus of the evaluation is the distribution of sleep and wakefulness across the night, including sleep parameters such as sleep latency, time awake after sleep onset, and total sleep time, it is possible to use activity monitoring devices that are typically worn on the wrist. A wrist actigraph is a small, solid-state, computerized movement-detector that continuously records wrist movement throughout the night. Several studies support the reliability and validity of the assessment of different sleep parameters based on actigraphs and its validity as an assessment tool of sleep disorders (ASDA, 1995b; Sadeh, et al., 1989; Sadeh, et al., 1991). A wrist actigraph provides a relatively unobtrusive, objective all-night measure of several sleep parameters that allows monitoring of the patient's sleep for extended periods of time in the patient's own environment.. To evaluate circadian rhythm disturbances, some versions of wrist actigraphs can be equipped with light meters that allow for the continuous measurement of light exposure. When worn continuously, day and night, actigraphs can be used to evaluate rest-activity behaviors and versions that are equipped with a behavioral response capacity can assess reaction-time upon a prompt and assist in the evaluation of alertness.

Sleep questionnaires. Sleep questionnaires provide valuable information about the perceived severity of a presenting sleep complaint as well as the perceived efficacy of treatment. In addition, they allow for collection of data on a wide variety of behavioral variables. However, the retrospective nature of the questionnaires may introduce intentional or unintentional biases (Bootzin & Engle-Friedman, 1981). Therefore, while sleep questionnaires are of value in providing

adjunctive data, they should not be used alone in assessing sleep. The use of multiple measures (e.g., collateral reports, mechanical devices, or PSG) will improve reliability and validity.

Daily sleep diaries. Daily sleep-wake diaries are a valuable assessment tool that can provide continuous daily information on sleep parameters as well as on daytime functioning. Unlike polysomnography that usually records only one or two nights of sleep, sleep diaries can provide information about sleep behavior for weeks and even months. Although insomniacs often overestimate sleep onset latency and underestimate total sleep when compared to polysomnography, the correlation between diaries and polysomnography is substantial (e.g., Carskadon, et al., 1976). The validity of diaries with other measures increases over time. Individuals become more exact at assessing their sleep as they continue to monitor it (Franklin, 1981).

To be maximally useful, care must be taken to ensure that daily sleep diaries are in fact completed each morning. If diaries are collected infrequently, patients may not fill them out until immediately before they are collected. Under these circumstances, the diaries would be no more useful than retrospective questionnaires (Bootzin & Engle-Friedman, 1981). One way to eliminate this problem is to have patients call in their data each morning to a telephone answering machine (Spielman, Saskin, & Thorpy, 1987). In this way, problems with compliance can be detected immediately and corrective actions taken.

Observers. Observers, such as nurses, spouses, or parents can be used to assess sleep onset, movement, respiration, and reaction to noise. In addition to general problems common to observational data, some specific problems arise in the use of observers in assessment of sleep-related variables. For example, spouses may fall asleep before their to-be-observed partners and be unaware of their sleep later at night. Sleep reports by parents and children are similarly often in poor agreement (Clarkson, et al., 1986). The relatively low validity of observer data suggests that observers be used primarily as supplemental, rather than as primary, sources of information.

Assessment of Daytime Sleepiness

The measurement of sleepiness is complex because it is an inferred construct with multiple indicators. There are cognitive, physiological, and behavioral indicators of sleepiness that are reflected in self-report, physiological, and performance measures. Further, there are a number of related constructs that, although they overlap with sleepiness, are not identical, such as fatigue and lowered arousal. In addition, daytime sleepiness is not solely a function of the length and quality of the previous night's sleep. Sleepiness and alertness are strongly influenced by the sleep-wake circadian rhythm and by factors such as threat, motivation, and interest in the activities being undertaken.

Stanford Sleepiness Scale (SSS). The subjective evaluation of an individual's current level of sleepiness is most commonly measured by the SSS, which consists of seven descriptively anchored points (Hoddes, et al., 1973). The statements reflect the severity of sleepiness from "Feeling active and vital; alert and wide awake" to "Almost in reverie; sleep onset soon; lost struggle to remain awake". Hoddes, et al. (1973) found that the scale was sensitive to sleep loss and validated it with performance measures when mental tasks were long (at least an hour), monotonous, and boring. Polysomnographically measured sleep latency has also been shown to correlate highly with subjective feeling of sleepiness as measured by the SSS.

Epworth Sleepiness Scale (ESS). In a clinical setting, it is often useful to have a measure of the extent to which individuals exhibit excessive sleepiness in different situations. The ESS (Johns, 1991) asks respondents how likely they are to doze off or fall asleep in different situations such as while a passenger in a car for an hour or while sitting or talking to someone. The test - retest reliability over five months is .81 and the correlation between spouses and respondents is .74 (Johns, 1994). Self-report measures, such as the ESS, are useful as quick screen for sleepiness and as a supplement to objective measures.

Multiple Sleep Latency Test (MSLT). The MSLT is arguably the most important assessment device for quantifying daytime sleepiness in human subjects. Since its development at Stanford University in the mid 1970's, it has been used to measure sleepiness in a wide range of experiments examining normal and abnormal functioning (Carskadon, 1994).

The MSLT is a measure of how long it takes to fall asleep on multiple opportunities throughout the day. It is intended to be a standardized set of procedures for obtaining an objective measurement of an individual's "physiological sleepiness" at different times of the day. Physiological sleepiness should be distinguished from "manifest sleepiness," an individual's behavioral or subjective level of arousal. Manifest sleepiness is influenced by other physiological needs, motivation, and demands from the immediate environment.

The MSLT consists of a series of four to seven polysomnographically-monitored naps, administered every two hours (see Carskadon, et al., 1986, for detailed procedures). Subjects should not be given caffeine or alcohol during the day of MSLTs to avoid confounding effects on sleep latency. Similarly, investigators should be aware of any medications being taken by the subject that might influence the latency to sleep onset.

The nap is considered to have begun as soon as the lights are turned off and is terminated after 20 minutes, three consecutive 30-second periods of stage 1 NREM sleep, or one 30-second period of any other sleep stage. The latency to sleep is defined as the amount of time between "lights out" and the first 30-second period of any sleep stage.

Different average MSLT scores indicate different levels of sleepiness, with shorter scores indicating a higher degree of physiological sleepiness. MSLT averages below 5 minutes are considered "pathological" and indicate a severe degree of sleepiness. Scores in this range are often seen in individuals with narcolepsy, sleep apnea, and those with either acute or chronic sleep deprivation (Carskadon & Dement, 1981; Dement, Carskadon, & Richardson, 1978; Richardson, et al., 1978). Pathologic levels of sleepiness have been found to correlate with performance deficits (Carskadon, Littell, & Dement, 1985). Nocturnal values, such as sleep duration the previous night, respiratory disturbance index, and lowest oxygen desaturation, do not correlate well with MSLT measures and account for less than 25% of the variance in MSLT scores (Pollak, 1997). A greater percentage of the variance can be explained (over 40%) if multivariate [rather than bivariate correlations] analyses are used. In a community sample, 6 variables (chronic daytime tiredness, body-mass index, psychological distress, nocturnal motor activity, serum

thyrotropin level, and age) were found to independently and significantly predict MSLT scores (Kronholm, Hyyppä, Alanen, Halonen, & Partinen, 1995).

Although the MSLT has been helpful in identifying excessive sleepiness due to narcolepsy and sleep apnea, it is not a pure measure of physiological sleepiness in all individuals. There are determinants, in addition to sleepiness, that affect the capacity to fall asleep. Insomniacs tend to take a long time to fall asleep on the MSLT, even following a poor night's sleep. The same variables that produce insomnia at night appear to result in elevated MSLT scores. On the other hand, many individuals who fall asleep quickly at night are able to fall asleep quickly on the MSLT even when they are not sleep deprived (Dorsey & Bootzin, 1987). Thus, the MSLT appears to measure both sleepiness and individual differences in sleep tendency.

Maintenance of Wakefulness Test (MWT). The MWT is a variant of the MSLT in that the ability to remain awake, rather than the ability to fall asleep, is measured (Mitler, Gujavarty, & Browman, 1982). This test has been used clinically to assess daytime sleepiness and treatment efficacy. The procedure involves sitting in a comfortable chair or sitting up in bed in a darkened room for either a 20- or 40-minute period or until the beginning of sleep onset. The criteria used for sleep onset includes either the first consecutive 10 seconds of sleep or the same criteria as in the MSLT: three consecutive 30-second epochs of NREM stage 1 or a single epoch of any other stage of sleep. The first trial occurs two hours following the final morning awakening and is repeated three more times every two hours.

A normative study found that healthy sleepers had an average sleep latency of 18.1 minutes for a 20 minute MWT and 32.6 minutes for a 40 minute MWT (Doghramji, et al., 1997). Sleep latency values in clinical populations (e.g., narcolepsy and obstructive sleep apnea syndrome) are significantly lower. Using a statistically-based criterion of two standard deviations below the mean, the lower limit of the normal range is 10.9 and 12.9 minutes for a 20 and 40 minute MWT using the first 30 second epoch of stage 1 as the criterion for sleep onset. Using the more stringent criterion, the lower limit of the normal range is 13.5 and 19.4 minutes. Based on their normative data, Doghramji and colleagues (1997) recommend the 20 minute protocol over the 40 minute one

as it is not as susceptible to age effects (older people are able to maintain wakefulness longer than younger people) and is more cost effective.

The MWT appears to be more sensitive than the MSLT to the effects of treatment. Many patients' MSLT scores remain pathological after treatment. In people with disordered sleep (i.e., obstructive sleep apnea, narcolepsy, idiopathic hypersomnia, depression, periodic limb movement, and head trauma), MSLT scores showed a slight, nonsignificant increase after treatment while the MWT scores showed large increases 1 to 6 months after treatment (Sangal, Thomas, & Mitler, 1992).

Pupillometry. The pupil is fully dilated in a darkened room but constricts as the individual begins to fall asleep. This has led to measures of sleepiness based on the degree of constriction and the degree of stability of pupil size. Untreated patients with excessive sleepiness have smaller diameter pupils than normals and their pupils show frequent constrictions and dilation, while normals show only minor changes in pupil diameter (Lin, et al., 1990; Yoss, et al., 1969). Research on the pupillary light reflex, which measures the time taken for a pupil's diameter to dilate to baseline levels after light flashes, indicates that sleep apnea patients show increased latencies to recovery with increased sleepiness as measured by MSLT (Pressman & Fry, 1989). Although there have been encouraging results with pupillometry for those who are severely sleepy during the day, such as those with narcolepsy and severe sleep apnea, it has been less consistently useful for use with other sleep disorders.

Performance measures. Performance measures provide objective data in assessing the effect of sleepiness and can be use to evaluate the efficacy of treatment. The most commonly used performance measures are reaction time, attention and vigilance, mental arithmetic, tracking and memory tasks (see Bootzin & Engle-Friedman, 1981). Slow reaction time, lack of response, or errors are indicators of sleepiness. A common finding in sleep deprivation studies is that sleep loss impairs performance on most cognitive and sustained attention tasks. Such impaired performance is most consistently documented when the task is long (30-60 min.), repetitive, and boring. In general, when severely sleep deprived subjects are motivated, they can sustain attention for short

periods of time and perform no differently than non-sleep deprived subjects. Even on simple, short duration tasks (3-10 min.), however, researchers have found performance decrements in sleepy subjects (Dinges, 1992). Lapsing, which refers to a period of marked response delay or response omission, is the foremost effect of sleep loss. Characteristic performance patterns in sleep deprived individuals include a slowing on self-paced cognitive tasks and the speed of the fastest response time, progressive and accelerated performance degradation, increased variability in response time, and erratic or unevenness in performance across trails (Kribb & Dinges, 1994; Dinges, 1992).

SLEEP DISORDERS

In providing a review of sleep disorders, we will use the diagnostic categories of the recently published, International Classification of Sleep Disorders, Revised: Diagnostic and Coding Manual (ASDA, 1997). The major categories are dyssomnias, parasomnias, and sleep disorders associated with medical or psychiatric disorders.

Dyssomnias

The dyssomnias include both disorders of initiating and maintaining sleep and disorders of excessive sleepiness. There are three major categories, circadian rhythm disorders, intrinsic sleep disorders, and extrinsic sleep disorders.

Circadian Rhythm Disorders

Circadian rhythm disorders occur when individuals attempt to sleep at times that are inconsistent with their underlying biological clocks. Intrinsic circadian rhythm disorders refer to desynchronies between attempts to sleep and the sleep-wake circadian rhythm that are due presumably to internal rather than external causes. These disorders might result from a weak circadian rhythm or from an inability to entrain the rhythm to the environment. Extrinsic circadian rhythm sleep disorders occur when environmental conditions, such as working night shifts or traveling across time zones, result in disrupted sleep-wake circadian rhythms.

Intrinsic circadian rhythm disorders