Extrinsic sleep disorders

External factors such as sleep schedules, light, noise, drugs, nicotine, alcohol, and activity levels may cause disrupted sleep. In some instances, even chronic insomnia may be due to these factors.

Inadequate Sleep Hygiene

Inadequate sleep hygiene is defined as "a sleep disorder due to the performance of daily living activities that are inconsistent with the maintenance of good quality sleep and full daytime alertness" (p. 73, ASDA, 1997).

The likelihood that sleep disorders will be produced by inadequate sleep hygiene depends on predisposing factors, on the persistence of the behavior, and on the degree to which it deviates from normal practices. For example, while some individuals who maintain highly irregular sleep-wake schedules never experience sleep disturbances, others will report great difficulties in initiating or maintaining sleep even when their schedules are less irregular. The probability that an individual will engage in behavior that is inconsistent with the maintenance of good sleep is increased for individuals with a low tolerance for daytime sleepiness. Such individuals may take naps, increase caffeine consumption, or go to bed too early. Although these behaviors may increase alertness in the short run, they may weaken the underlying biological clock and lead to insomnia when practiced for a long time.

Three aspects of sleep hygiene will be discussed: (a) the regularity of sleep-wake schedules, (b) regular exercise, and (c) the use of caffeine and nicotine.

Sleep-wake schedules. The relation between biological rhythms and the sleep-wake cycle is the basis for the common recommendation to maintain a regular sleep schedule. Those who keep irregular schedules for long periods of time are at risk for developing sleep disturbances. Conversely, those with sleep disturbances frequently benefit from regularizing their sleep schedule. Indeed, when irregular sleepers regularize their nocturnal sleep while at the same time making sure their sleep is of adequate length their sleep and daytime alertness improve and these improvements are greater than those observed when an intervention includes only the provision of

adequate sleep duration without attempts to regularize the sleep schedule (Manber & Bootzin, 1996).

The role of naps within the sleep-wake schedule is complex. In polysomnographic terms, afternoon and evening naps begin the night's sleep and morning naps are a continuation of the previous night's sleep. Thus, afternoon and evening naps have more slow wave sleep and morning naps have more REM sleep. The sleep on a night that follows an afternoon nap resembles sleep in the latter part of a normal night, which is lighter and has more awakenings. Thus, in order to preserve the quality of sleep at night, afternoon and evening naps should be avoided. If they are taken, they should be short and should be made a regular part of the daily schedule, occurring at about the same time each day.

Regular exercise. For exercise to be effective in improving sleep, individuals have to engage in a program of exercise. Recommendations to exercise are not always effective because of variability in compliance. A meta-analysis concluded that exercise must last for at least an hour in order to have reliable effects on sleep (Youngstedt, O'Connor, & Dishman, 1997). Regular exercise programs in older men have been shown to produce increased continuous sleep, increased slow-wave sleep, and decreased nighttime awakenings (Edinger, et al., 1993; Vitiello, et al., 1992).

Use of caffeine and nicotine. Caffeine and nicotine are central nervous system stimulants that lighten sleep and produce arousals (ASDA, 1997). Caffeine is present in chocolate and in coffee (100-200 mgs, depending on the strength of the brew), tea (50-75 mgs), and many carbonated berverages (50-75 mgs). There are individual differences in the degree of sensitivity to caffeine. Some individuals may experience sleep disturbances from the consumption of chocolate and soft drinks alone. Other medications including analgesics (those that contain caffeine), bronchodilators, decongestants, and appetite suppressants are or contain stimulants that produce an increase in sleep latency, decrease in total sleep time, and an increase in spontaneous awakenings (ASDA, 1997; Brown, et al., 1995). Cessation of caffeine or nicotine may be associated with

withdrawal symptoms such as sleepiness, irritability, lassitude, and severe depression (ASDA, 1997).

Complaints of insomnia and/or anxiety may be due to excessive ingestion of caffeine, nicotine, or other stimulants. Since caffeine has a plasma half-life of approximately 6 hours, individuals continue to experience its effects long after it has been ingested. Reducing or eliminating the intake of caffeine, particularly in the afternoon and evening, and quitting smoking can lead to substantial improvement in sleep. However, an increase in disturbed sleep can be expected during the initial period of smoking withdrawal (Wetter, Fiore, Baker, & Young, 1995).

Environmental Sleep Disorders

Among the many environmental factors that affect sleep are noise, temperature, room or bed sharing, institutionalization, and the need for vigilance during sleep. The presenting complaints consist of insomnia symptoms or marked impairment of daytime functioning. This diagnosis is given only when the environmental causes are judged to be the primary cause for the complaints and its prevalence rate in patients seen in sleep disorder centers is estimated as less than 5 percent (ASDA, 1997). Sensitivity to environmental factors increases with age and is subject to great individual variability.

Noise decreases both the amount of deep sleep and the continuity of sleep. There is an increase in body movements and sleep stage shifts. Studies show that a presentation of an auditory stimulus is often followed by very brief arousal (3-15 seconds). The fragmentation of sleep often leads to increased daytime sleepiness (Stepansky, et al., 1984). Unpredictable noise is particularly disturbing (Sanchez & Bootzin, 1985). Thus, continuous white noise, such as from a fan, is often useful for noisy environments.

People can adjust to a wide range of room temperatures but have more difficulty adjusting to cold than to heat. Uncomfortable room temperatures, particularly cold rooms, affect the quality of sleep more than the capacity to fall asleep (Haskell, et al., 1981).

Recommendations for reducing environmental disturbances and strengthening good sleep hygiene have become an integral part of many treatments for insomnia. Frequent

recommendations include maintaining regular sleep-wake schedules, establishing an environment conducive to sleep (quiet, dark, and secure), increasing exposure to light and other sensory stimuli upon awakening, and carefully timing and limiting the use of caffeine and alcohol. The importance of a comprehensive treatment approach, based on a thorough analysis of the problem, should be emphasized.

Drug Dependent Sleep Disorders

The use of drugs to induce or to suppress sleepiness may cause adverse effects with chronic use. Although alcohol and sedative/hypnotics are thought to induce a state of unconsciousness that resembles sleep, in fact the resemblance to natural sleep is weak. Hypnotics produce disruptions in sleep architecture and increases in arousal threshold (Kay & Samiuddin, 1988). The use of stimulants to suppress sleep can produce persistent insomnia.

Hypnotic-dependent sleep disorder. Hypnotic-dependent sleep disorder is characterized by insomnia or excessive sleepiness associated with the tolerance to or withdrawal from hypnotic medications (ASDA, 1997).

Sedative/hypnotics are the most frequently-used method for treating insomnia. With continued use, tolerance rapidly develops and larger doses are required to achieve a soporific effect. As dosage is increased to offset tolerance, daytime carryover effects also increase and may include symptoms such as excessive sleepiness, poor motor coordination, visual-motor problems, and late afternoon restlessness (ASDA, 1997). These symptoms promote psychological dependency as the individual becomes convinced that the daytime effects are symptoms related to poor sleep the night before. This leads to a continued search for pharmacological relief from the insomnia, possibly by consulting several physicians or trying a variety of sleep-inducing medications. Polysomnographic monitoring of chronic sedative/hypnotic users shows decreases in stages 1, 3, 4, and REM sleep, an increase in stage 2 sleep, and fragmented NREM and REM sleep with frequent stage changes (ASDA, 1997).

Once benzodiazepine hypnotics or anxiolytics are begun, it is often difficult for individuals to withdraw. In a study of abrupt discontinuation of individuals who had been taking

benzodiazepines for one year or longer, 56% taking long half-life and 62% taking short half-life benzodiazepines failed to remain free of drugs during a 5-week follow-up (Rickels, et al. 1990). In a companion study of gradual taper, 32% of long half-life and 42% of short half-life benzodiazepine treated patients failed to achieve a drug-free state (Schweizer, et al., 1990).

Cessation of sedative/hypnotics results in withdrawal symptoms that may also promote psychological dependence. Insomnia has been shown to be among the most frequent withdrawal symptoms with the long-term use of benzodiazepines (Busto, et al., 1986; Schneider-Helmert, 1988). Other withdrawal symptoms include a dramatic increase in REM and stage 2 sleep percentages and daytime sleepiness (Schneider-Helmert, 1988). While total sleep time does not decrease with withdrawal of medication, the subjective perception of rebound insomnia is judged to be much worse than before drug therapy, and often leads the patient to resume sedative/hypnotic use.

Alcohol-dependent sleep disorder. About 10 to 15 percent of patients with chronic insomnia have a problem with substance abuse, particularly alcohol and other sedatives (Gillin & Byerley, 1990). Alcohol, like other depressants, is a REM suppressant. Withdrawal from heavy drinking produces a REM rebound effect which is accompanied by restless sleep and nightmares. Alcohol also exacerbates sleep apnea since it affects ventilation during sleep by increasing upper airway resistance (Taasan, et al., 1981; Issa & Sullivan, 1982). The effects of alcohol on sleep can persist as long as 2 to 3 hours after plasma alcohol levels return to zero (Zarcone, 1994).

Alcohol-dependent sleep disorder is characterized by the use of alcohol for initiation of sleep onset (ASDA, 1997). Usually the ingestion of alcohol begins about 3 to 4 hours before bedtime with consumption equivalent to about 6-8 ounces of liquor. This condition is not associated with other alcoholic patterns of drinking (drinking during the day and related socio-economic problems) and is usually considered clinically relevant if the alcohol has been used daily for a minimum of 30 days. Continued use results in tolerance to the alcohol as a sleep-inducing agent and sleep fragmentation becomes more prominent. Often, patients will become desperate and

increase the amount of alcohol or add other sedatives in order to sleep. Patients will often report that they have no sleep disturbance as long as they continue to take the alcohol nightly.

Polysomnographic monitoring of this type of alcohol user shows an increase in stages 3 and 4, fragmentation of REM sleep, and frequent awakenings and sleep stage transitions (ASDA, 1997). Frequent sleep transitions are usually seen later in the sleep period as the alcohol blood level begins to decline. In addition to polysomnogram recordings, diagnosis is confirmed if the patient complains of insomnia, has unsuccessfully attempted to withdraw from bedtime alcohol ingestion, and there is no evidence of medical or psychiatric disorder responsible for the insomnia (ASDA, 1997).

Stimulant-dependent sleep disorder. Stimulant-dependent sleep disorder is characterized by the reduction of sleepiness or the suppression of sleep through the use of central stimulants with resulting alterations in wakefulness following drug abstinence (ASDA, 1997). Many stimulants are used to combat daytime sleepiness. Cocaine, amphetamines, and inhalants have sleep suppressing effects. They increase time to fall asleep and REM sleep latency and decrease total sleep time and REM time (Gillin, 1994). Tolerance may develop with chronic use resulting in increased dosages. During episodes of stimulant abuse, individuals may go for days without sleep followed by periods of hypersomnia and accompanied psychological and mood problems of paranoia, psychosis, social withdrawal, and sleep disturbances. Individuals may use sedatives or alcohol to counteract the sleep suppressing effects of stimulants and reduce anxiety.

Withdrawal from stimulants is associated with hypersomnia and depression which can last two weeks (Gillin, 1994). At the end of the two week period, the individual may experience a few days of insomnia and poor quality sleep. Both sleep disturbances and hypersomnia may be risk factors for reuse of stimulants or use of other drugs.

Parasomnias

Parasomnias are phenomena that intrude into sleep, although they are not primarily part of sleep per se. The more common examples include sleep talking, sleepwalking, sleep terrors, enuresis, and bruxism. These disorders reflect central nervous system activation and are divided

into the following categories: (a) arousal disorders, (b) sleep-wake transition disorders, (c) parasomnias usually associated with REM sleep, and (d) other parasomnias.

Arousal Disorders

Arousal disorders, such as sleep terrors and sleepwalking occur during arousal from slow wave sleep.

Sleep terrors. Sleep, or night, terrors, also called "pavor nocturnus" in children and "incubus" in adults, arise out of slow wave sleep. They are characterized by the subjective experience of panic, rapid autonomic arousal, and behavioral activation. The individual will appear to be in great distress, emit a piercing scream, frequently will sit-up, and may flee from the bedroom.

Despite behavioral activation and the experience of intense emotion, the individual may have either partial or total amnesia for the details of the event. If awakened during the sleep terror, the individual is likely to be confused, may report a sense of terror or dread, but reported sleep mentation will be vague, unlike the detail that occurs during nightmares. Because sleep terrors arise out of slow wave sleep, they tend to occur during the first few hours of sleep. Nightmares, in contrast, are REM phenomena, and are more likely to occur during the latter half of the night. Sleep terrors need also to be distinguished from nocturnal panic episodes though the latter are usually not associated with screaming and do not produce vivid fearful imagery. Another differential diagnosis for sleep terrors are nocturnal seizures. Like sleep terrors, they are associated with difficulty to arouse during the event and with confusion upon arousal. However, whereas nocturnal seizures are often associated with daytime sleepiness, sleep terrors usually are not. Sleep terrors are not associated with daytime EEG abnormalities. Unless the clinical presentation suggests the possibility of a seizure disorder or other sleep disorders, an all night sleep study is not necessary for the diagnosis of sleep terrors.

Recurring episodes of sleep terrors occur in between 1 and 6 percent of prepubertal children and the typical age range for the disorder is between 5 and 7 years old (Thorpy & Glovinsky, 1987). Sleep terrors occur in about 1 percent of the adult population (Thorpy &

Glovinsky, 1987). Although sleep terrors tend to abate after the onset of puberty, sleepwalking sometimes occurs or persists in those who had sleep terrors during childhood.

Pharmacological treatment of sleep terrors is based upon attempts to reduce the amount of slow wave sleep experienced and usually involves the use of benzodiazepines or tricyclic antidepressants (Thorpy & Glovinsky, 1987). More recently, nonpharmacological treatments have also shown promise. Self hypnosis (Hurwitz, 1986; Hurwitz & Mahowald, 1988) and scheduled awakenings with children (Lask, 1988) have been reported to be effective. In the scheduled awakening treatment, parents note how long after the child goes to sleep that a sleep terror is typically experienced. On subsequent nights, the parents are instructed to awaken the child 10 to 15 minutes before the sleep terror would have occurred. This procedure has been reported to be highly successful and may serve to disrupt and lighten slow wave sleep. It is also easier to comfort a child after a planned awakening than from one in which the child is frightened. Because both insufficient sleep and increased stress often exacerbate disorders of partial arousal (Guillaminault, et al. 1995; Moldofsky et al. 1995), including sleep terrors, treatment should include both education about the contribution of these factors and specific suggestions on how to manage stress and insure sufficient sleep.

Sleepwalking. Sleepwalking, or somnambulism, occurs during the first few hours of sleep, out of slow wave sleep, not REM. Thus, sleepwalkers are not acting out their dreams. Their movements are coordinated with their environments, they typically avoid objects, may go to the bathroom, and even return to bed if left alone. If awakened, sleepwalkers are confused and have little memory of dream mentation. Sleepwalking episodes may last from 15 seconds to 30 minutes (Aldrich, 1994). Sleepwalking should be differentiated from REM behavior disorder (see parasomnias associated with REM) in which individuals lack the muscle atonia that accompanies REM so that they do act out their dreams.

Sleepwalking is more common among children than adults. Fifteen to 30 percent of healthy children are estimated to have at least one episode and 2 to 3 percent have more frequent episodes (Thorpy & Glovinsky, 1987). Sleepwalking generally first occurs during pre-

pubescence, reaches a peak prevalence at 12 years of age, and only infrequently occurs in children older than 15 year old. The prevalence in adults is less than 1 percent. While there appears to be no sex related differences, there is evidence for a familial tendency. Sleepwalking occurs 10 times more frequently among first degree relatives than in the general population (Karacan, 1988).

There is no known pathophysiology which corresponds to the occurrence of sleepwalking. Fatigue, stress, sleep deprivation, and drug use (especially sedative/hypnotics) may precipitate episodes. Psychopathology is not usually associated with the occurrence of sleepwalking in children, but it is in adults. Kales, Soldatos, Cadwell, et al. (1980) reported that adult sleepwalkers had elevated MMPI profiles (Pd, Sc, and Ma) and 72 percent of his sample had psychiatric diagnoses.

Parents of sleepwalking children should be cautioned to take safety precautions such as locking doors and windows. It is usually better to direct sleepwalkers back to bed rather than to awaken them. The very process of awakening may frighten and confuse them. When sleepwalking occurs frequently and is associated with potentially injurious or harmful behaviors, the intervention and preventive measures that are used for sleep terrors are also effective with the sleepwalker. These include preventing sleep deprivation, introducing stress coping strategies, and using scheduled awakening (Stores, 1990; Thorpy & Glovinsky, 1987).

Although there is a higher incidence of psychopathology among adults who sleepwalk than among those who do not, a complaint of sleepwalking does not necessarily indicate the presence of psychopathology. Sometimes sleepwalking persists well into the second decade of life, particularly among individuals with a history of parasomnias who report being sound sleepers and difficult to arouse. These individuals are likely to need more sleep, to have higher than average slow wave sleep, and to suffer from sleep deprivation. Nevertheless, a complete psychological evaluation is often recommended for adults who sleepwalk. For sleep-walking adults, a motion-detection alarm is often useful. In addition to the other nonpharmacological interventions described above, pharmacological therapy is indicated under conditions that put the sleepwalker in particular

danger. It includes medications that are known to suppress slow wave sleep and reduce nocturnal arousals, such as benzodiazipines and antidepressants (Karacan, 1988).

Sleep-Wake Transition Disorders

This category includes sleep talking and sleep starts. These disorders usually occur in the transitions between wakefulness and sleep and, more rarely, in the transitions between sleep stages. They occur in otherwise healthy subjects (ASDA, 1997).

Sleep talking. Sleep talking, also called somniloquy, occurs during all stages of sleep. About 80 percent of sleep talking takes place during NREM sleep (Arkin, 1978). Dream mentation is recalled in association with 79 percent of the episodes occurring in REM sleep, 46 percent of the episodes occurring in stage 2, and 21 percent of the episodes occurring during slow wave sleep (ASDA, 1997). Usually sleep utterances occur spontaneously, are brief, and are devoid of signs of emotional distress. Occasionally, sleep talking may be induced or prolonged by conversation with the sleeper. Laughing, singing, and crying occasionally occurs during sleep, as well (Anch, et al., 1988).

Almost everyone has experienced an episode of sleep talking. In a survey of 1508 college undergraduates, 83 percent reported having had at least one episode of sleep talking (Perlis, 1989). Sleep talking may be precipitated by emotional distress or physical illness and may also accompany other parasomnias. Since sleep talking is considered benign, there is no recommended treatment.

Sleep starts. Sleep starts, also called hypnic jerks, are sudden brief contraction of the legs, sometimes also involving the arms and head, that occur during the transitions from wakefulness to sleep. They occur mainly at the beginning of the sleep episode. Sleep starts are sometimes associated with sensory experiences including the subjective impression of falling, a visual hypnagogic dream, fragmentary auditory (e.g., loud bangs, snapping noises) or somesthetic (e.g., pain, floating) hallucination. Sleep starts are considered a normal phenomenon of the sleep onset process and should not be confused with seizures or movement disorders that occur at sleep onset or during sleep. A prevalence of a 60% to 70% in the population has been reported (ASDA, 1997; Mahowald & Schenck, 1998).

Sleep starts may be exacerbated by excessive caffeine, the use of stimulants, strenuous exercise prior to sleep, and stress. Although sleep starts are generally considered to be of little consequence, if they occur frequently causing repeated awakenings of the sleeper or bed partner, they may lead to sleep-onset insomnia.

Parasomnias Usually Associated With REM

This category includes nightmares, sleep paralysis, and REM sleep behavior disorder. These disorders are associated with or occur during REM sleep.

Nightmares. Nightmares are frightening dreams. They are often long and complicated and become increasing frightening, waking the dreamer up. Fear or anxiety are always associated with nightmares, but sometimes seem out of proportion to the content being reported.

Nightmares occur during REM sleep while sleep terrors and sleep panic attacks occur during NREM sleep. Two important differentiating features of nightmares are the time of the night that they occur and the detail of dream content that is reported. Nightmares, as REM sleep phenomena, are more likely to occur during the latter half of the night, and the dream content can be recalled in detail. Sleep terrors, associated with slow wave sleep, and sleep panic attacks, associated with transitions between stages 2 and 3, are more likely to occur during the first half of the night. In both sleep terrors and sleep panic attacks, the sleeper awakens very frightened but is able to recall only fragmentary images, if any, and general feelings of fear and anxiety or of being attacked or suffocated.

Nightmares are a common feature of sleep. Between 10 to 50 percent of children aged 3 to 5 have frequent enough nightmares to concern their parents (ASDA, 1997), and 5 to 8 percent of adults report having problems with nightmares (Bixler, et al., 1979; Klink & Quan, 1987). The frequency of nightmares decreases with age. About 10 percent of adolescents (Muller & Wood, 1991) and college students (Wood & Bootzin, 1990) have at least one nightmare a week as compared to about 1 percent of healthy elderly (Salvio, Wood, & Schwartz, 1991)

The occurrence of frequent nightmares in adults has been considered a primary diagnostic criterion for anxiety and stress disorders. However, no relationship was found between frequent

nightmares and trait anxiety in a study of college students (Wood & Bootzin, 1990). This suggests that other criteria, in addition to nightmares, are necessary to diagnose anxiety disorders.

Sleep paralysis. Sleep paralysis, also called familial sleep paralysis or hypnagogic or hypnapompic paralysis, is a phenomenon during which individuals are unable to perform voluntary movements at either sleep onset (hypnagogic paralysis) or upon awakening from sleep (hypnapomic paralysis). Although temporary, the paralysis immobilizes the head, trunk, limb and, sometimes, speech-related musculature. Episodes usually last less than 5 minutes and can sometimes be terminated by intense efforts to move (Anch, et al., 1988). Sleep paralysis is hypothesized to be due to a dysfunction in REM sleep mechanisms in which the muscle atonia associated with REM sleep occurs during wake-sleep transitions. Sleep paralysis resembles cataplexy, seen in narcolepsy. The two states differ in that sleep paralysis occurs during sleep-wake transitions, whereas cataplexy occurs during the day and is often triggered by an intense emotional stimulus. Both cataplexy and sleep paralysis may be symptoms of narcolepsy.

As an isolated symptom, sleep paralysis occurs in 40 to 50 percent of normal individuals sometime during their lifetime (ASDA, 1997). Recurrent episodes of sleep paralysis, in the absence of a primary diagnosis of narcolepsy, are rare. Between 17 and 40 percent of narcoleptics experience frequent episodes of sleep paralysis. Irregular sleep habits, sleep deprivation, stress, and sleep position have all been reported as predisposing factors for sleep paralysis.

REM sleep behavior disorder (RBD). RBD is characterized by the intermittent loss of muscle atonia during REM sleep. Individuals with RBD may engage in elaborate behavioral sequences during REM sleep. If awakened during an episode, the individual is likely to report dreams which correspond to their behavior. Episodes typically occur once a week, but may occur several times per night on consecutive nights. Polysomnographically, REM behavior disordered individuals exhibit normal, more rapid eye movements than usual, and substantial muscle tonus.

Little is known about the overall prevalence of RBD and the largest series of cases consists of 68 patients (Schenck & Mahowald, 1990). The disorder appears to occur primarily in elderly men and there is some evidence of a familial pattern (ASDA, 1997).

RBD has been considered a human analog of experimental work in animals that found that REM without atonia could be produced by ablating regions in the pons and medulla (Morrison, 1988). Extensive neurological and neuropsychological evaluation of 21 RBD patients revealed that 38 percent evidenced discernible brain damage with the remaining classified as "idiopathic" (Mahowald & Schenck, 1994).

Other Parasomnias

Other parasomnias that are not easily classified include sleep enuresis, sleep bruxism, and sudden infant death syndrome.

Sleep enuresis. Sleep, or nocturnal, enuresis is involuntary urination during the night and occurs primarily in children. Enuretic children are commonly described by their parents as very deep sleepers, a report consistent with laboratory findings of increased duration of slow wave sleep relative to nonenuretic children (Andres & Weinstein, 1972). Enuresis should not be considered an indication of psychopathology for children under age 5, particularly boys, since its prevalence among 4 year old boys is 14 to 38 percent (Feldman, 1983).

There are two types of sleep enuresis, primary and secondary. A child with primary enuresis has always had bedwetting problems and has never had any extended dry periods. Typically, children with primary bedwetting problem are less than 8 years old. A child with secondary enuresis is one who develops enuresis after having had extended dry periods. The onset of secondary enuresis is often at times of emotional distress. Primary enuresis is more likely to be genetic than secondary enuresis (Kales, Soldatos, & Kales, 1980) and is more likely to be influenced by biological factors such as a smaller bladder (Troup & Hodgson, 1971). Enuresis can occur in any stage of sleep, including nocturnal awakenings, and is most common during the first third of the night (ASDA, 1997).

Treatment for sleep enuresis includes bladder training aimed at increasing bladder capacity and control, use of the "bell and pad" system based on classical conditioning, and a combination of information, support, and reinforcement strategies. Whereas the efficacy of bladder training is about 30%, the bell-and-pad treatment has success rates as high as 70%, with the best results in

children older than 7 years (McCain, 1979). A common pharmacological treatment involves desmopressin, an anitidiuretic hormone, which as been shown to reduce the number wet nights in primary enuresis (Hansen & Jorgessen, 1997). The efficacy of desmopressin treatment may be greater for children who exhibit decreased levels of psychosocial distress (Dittmann & Wolter, 1996). Tricyclic antidepressants have been found to have relatively low success rates and high relapse rates upon withdrawal (Kales, Soldatos, & Kales, 1980).

Sleep bruxism. Bruxism involves the grinding or clenching of teeth during sleep. Bruxism can occur during all stages of sleep, but most commonly occurs during stage 2. As much as 90 percent of the population reports that they have ground their teeth some time during their lifetime. In about 5 percent of these, the problem may be severe enough to require treatment (ASDA, 1997) and can lead to dental damage and temporomandibular joint (TMJ) disorders. Severe bruxism is often associated with daytime anxiety.

The most common treatment is a rubber mouthguard worn over the teeth (Hartmann, 1989). In some cases, relaxation training or biofeedback has been effective. In a double-blind clinical trial, the administration of L-dopa was shown to significantly decrease the number of bruxism episodes (Lobbezoo et al., 1997).

Sudden infant death syndrom (SIDS). SIDS is characterized by sudden death during nocturnal sleep in the absence of definable pathology. Epidemiological studies have indicated that the following groups are at increased risk (ASDA, 1997): premature infants, twin or triple births, subsequent siblings of SIDS infants, infants born to substance-abusing mothers, infants who previously experienced apparent life-threatening events (ALTEs) requiring mouth-to-mouth resuscitation or vigorous stimulation, infants of low socio-economic parents, and African-American or Eskimo infants. SIDS is estimated to occur in 1 to 2 per 1,000 live births and is most likely to occur between the 10th and 12th week of life. SIDS occurs slightly more frequently in males then females.

Polysomnographic evaluation in one group of infants who subsequently died of SIDS found 3 variables that differentiated SIDS victims from matched controls (Blum, et al., 1988). All

the variables were related to respiratory function. More specifically, the authors found that the maximal duration of central apneas, the number of sighs followed by a central apnea, and the presence of obstructive or mixed apneas "significantly characterized the future SIDS victims". However, only 7 percent of those infants having ALTEs show evidence of apnea (Ariagno, 1987). Thus, infant sleep apnea may not be the primary cause of SIDS.

Sleep Disorders Associated with Other Disorders

Sleep Disorders Associated With Mental Disorders

Schizophrenia

The majority of schizophrenics undergoing acute psychotic phases experience some sleep disturbance. It has been proposed that different sleep architectures might be of use in diagnosing whether an acute psychotic episode is approaching or dissolving (Kupfer, et al., 1970). Schizophrenics in the waxing phase of an acute psychotic period have been found to have low total sleep times and very disrupted sleep. These patients took an average of 152 minutes to fall asleep. Their quantities of slow wave sleep and REM sleep were also significantly less than the controls. In contrast to these findings, schizophrenics in the waning phase have been shown to display a slight return of their sleep parameters toward those of the controls. During remission, the schizophrenics' sleep resembled the control's sleep values.

A number of studies have found shortened REM latencies in schizophrenics (Hiatt, et al., 1985; Zarcone, et al., 1987) and decreases in slow-wave sleep (Feinberg, et al., 1969; Hiatt, et al., 1985). An exception to these findings come from a studies of young schizophrenics who had never been medicated. They were found to exhibit normal REM latencies (Ganguli, et al., 1987). In contrast to the findings for REM latency, a recent study of medicated and neuroleptic-naive, first-episode schizophrenic patients found decreased amounts of slow-wave sleep in both groups (Keshavan, et al., 1998) which may indicate that slow-wave sleep is more related to the pathophysiology of schizophrenia than is REM sleep.

One puzzling observation about the sleep of schizophrenics concerns their response to experimental REM deprivation. It has been a consistent finding that acutely psychotic

schizophrenics fail to exhibit REM rebound after REM deprivation (Gillin, et al., 1974; Zarcone, et al., 1969). One possible explanation is that hallucinations and other positive symptoms may be an expression of a REM process (Cartwright, 1978). Since acutely psychotic schizophrenics have hallucinations, they may not need as much REM during the night as nonpsychotic individuals. Another REM phenomena, dreaming, seems to be deficient in schizophrenics. Schizophrenics awakened during REM sleep often report sparse dream content. Patients have reported dreams consisting of single objects, alone, without contextual cues (Dement, 1955).

The effect of antipsychotic medications on the sleep of schizophrenics remains somewhat unclear. There have been contradictory reports concerning whether there are increases in slow wave sleep, total sleep, and REM sleep in schizophrenics due to clorpromazine, flenfluradol, and haloperidol (Zarcone, 1988). A longitudinal study of schizophrenic patients, free from medication for at least two weeks, were studied at baseline, 4 weeks, and 1 year after beginning treatment (Keshavan, Reynolds, Miewald, & Montrose, 1996). At 4 weeks, sleep continuity significantly improved and REM latency showed a modest, nonsignificant increase; at the 1 year assessment, REM latency, time, and density significantly increased. It is unclear, however, whether these changes resulted from medication or a change in the illness-state, or both. Slow wave sleep did not change from baseline to the 1 year follow-up. The effects of Clozapine were recently assessed in a population of long-term medication free schizophrenics (Hinze-Selch, Mullington, Orth, Lauer, & Pollmächer, 1997). Clozapine significantly increased sleep continuity, REM density (but not REM time) and NREM sleep, particularly, stage 2 sleep, while stage 4 sleep significantly decreased.

Mood Disorders

Depression. Sleep problems are one of the primary symptoms of a major depressive episode (DSM-IV, 1994). It is estimated that approximately 90 percent of all individuals with depression will have at least a mild degree of sleep architecture abnormality (Reynolds, 1989). Conversely, about 21% of persons reporting serious insomnia have major depression (Mellinger, et al., 1985).

An extensive body of research identifies sleep EEG abnormalities of patients with major depression (see Benca, et al., 1992, for a meta-analytic review of these results). These abnormalities can be grouped into three general categories. The first category, sleep continuity disturbances, includes prolonged sleep latency, increased wakefulness after sleep onset, and, most notably, early morning awakenings, all contributing to decreased sleep efficiencies (see Benca, 1994). Sleep continuity disturbances lead depressed patients to complain about poor sleep and such complaints often precede the relapse of remitted depression (Perlis, et al., 1997). The second category, slow wave sleep (SWS) deficits, includes a reduction of amount and percent SWS primarily during the first nonREM period (Kupfer, et al., 1986; Reynolds, et al., 1985). Depressed men show a greater reduction in slow wave sleep than depressed women (Reynolds, et al., 1990). The third category, REM sleep abnormalities, includes reduced latency to the first REM episode, prolonged duration of the first REM episode, and increased rate of rapid eye movement, also referred to as increase REM density (Vogel, 1981). This category includes the most robust findings regarding sleep of depressed patients. Approximately 60% of depressed outpatients (Rush, et al., 1982a) have reduced REM latencies compared with only about 9% of matched controls. It should be noted, however, that shortened REM latency is not specific to major depression. Short REM latencies have been found in patients with narcolepsy, schizophrenia, alcoholism, and post-traumatic stress disorder (Benca, 1994).

The relationship between sleep abnormalities, particularly REM sleep abnormalities, and depression is also evidenced by the fact that REM sleep deprivation in the laboratory, as well as total sleep deprivation, often produces improvement of depressive symptoms with a time course paralleling the effects of antidepressants (King, 1977; Mendelson, 1987; Vogel, 1975). Some EEG sleep disturbances such as high REM density and low sleep efficiency are often normalized after successful treatment whereas others, such as SWS abnormalities, remain unaffected. Normalization of sleep occurs following successful treatment regardless of modality. This includes electroconvulsive therapy (Lahmeyer, et al., 1989), pharmacotherapy, and psychotherapy. Antidepressants have a suppressant effect on REM sleep (Vogel, et. al., 1990)

and often lengthen the REM latency significantly (Roth, et al., 1982). Successful psychotherapy leads to a reduction in REM density but does not alter REM latencies. It has been suggested that individuals with short latency to REM sleep and with increased eye movements during REM sleep (REM density) have a better response to pharmacotherapy. In contrast, EEG sleep abnormalities do not predict response to psychotherapy regardless of the type therapy, be it individual (Buysse, et al., 1992; Jarret, et al., 1990) or group therapy (Corbishley et al., 1990), interpersonal (Buysse et al., 1992) or cognitive behavioral therapy (Jarret et al., 1990), in a sample of outpatients (Thase, et al., 1994) or inpatients (Thase, Simons, & Reynolds, 1993). However, a recent study that used a composite of EEG sleep parameters in a large sample of outpatients with definite or probable endogenous subtype of depression to predict response to individual CBT (Thase, Simons, & Reynolds, 1996) found that abnormal sleep profiles and higher pretreatment depression severity were independently associated with poorer outcome including lower recovery rates and higher risks of recurrence.

The extensive body of research on sleep and depression has lead to formulation of several theories on the etiology of depression including malfunction of either a REM sleep activation mechanism, a slow-wave activating and REM sleep inhibition system, some component of a circadian timing circuit, or some combination of the three. The fact that some sleep abnormalities persist beyond the active phase of the depression and may be present even before the first episode of depression indicates that sleep abnormalities may reflect a vulnerability for depression (Giles, et al., 1990; Hauri, et al., 1974).

Mania. In comparison to unipolar depression, there is a dearth of research on mania and sleep. Manic patients have extended periods of wakefulness, short total sleep times, long sleep latencies, and deficits in slow wave sleep (ASDA, 1997). Manic patients also have the same shortened REM latencies and more dense REM episodes as do depressed patients (Hudson, et al., 1990).

Seasonal Affective Disorder (SAD). SAD is a disorder involving an annual fluctuation in mood which has been correlatively linked to seasonal fluctuations in melatonin levels and

variations in the period of sunlight. SAD patients present with problems in sleep quantity, sleep quality, and circadian phase position or the timing of sleep and wake. SAD has become one of the most rapidly-expanding areas for research, in sleep research as well as in affective disorders research. Strengthening the circadian oscillation in these individuals through light therapy alleviates the sleep problems and improves mood (Rosenthal, et al., 1984). Light therapy seems to clear up both the nocturnal sleep problems and the daytime sleepiness (Terman, 1994).

Anxiety Disorders

This category includes generalized anxiety disorder, obsessive-compulsive disorder, simple and social phobias, and posttraumatic stress disorder. Sleep disturbances involve sleep onset or sleep maintenance insomnia (ASDA, 1997).

Generalized anxiety disorder (GAD). Patients with GAD have been found to have increased sleep latencies, diminished REM and slow-wave sleep, higher number of transitions to light NREM sleep, increased waking during the sleep period, and decreased efficiency of sleep (Reynolds, Shaw, Newton, Coble, & Kupfer, 1983; Fuller, Waters, Binks, & Anderson, 1997). GAD patients are distinguishable from depressed patients by longer REM latencies and from psychophysiological insomniacs by persistently poor sleep efficiencies.

Obsessive-compulsive disorder (OCD). OCD patients have been found to have lower total sleep times, more awakenings, less NREM stage 4, and significantly shorter REM latencies than controls (Insel, et al., 1982). Short REM latencies for OCD patients, similar to those seen in depressives, have been reported by some (Insel et al., 1982) but not by others (Hohagen et al., 1994).

Posttraumatic stress disorder (PTSD). The sleep of PTSD patients often includes terrifying nightmares. Estimates of the percentage of Vietnam veterans suffering from nightmares have ranged from about 59 percent to 68 percent (DeFazio, et al., 1975; van der Kolk, et al., 1980). In PTSD patients, nightmares are reported to involve the same near-real-life scenes, occurring over and over again, as opposed to the changing nightmare content of the chronic, non-PTSD nightmare sufferers (Hartmann, 1987). As mentioned earlier, nightmares occur frequently in normal

populations (Wood & Bootzin, 1990). Thus, nightmares alone are insufficient for a diagnosis of PTSD.

Polysomnographic evaluations of the sleep of PTSD patients have revealed inconsistent and inconclusive abnormal sleep parameters. Some PTSD patients exhibit short REM latencies and high REM densities (Greenberg, et al., 1972; Kauffman, et al., 1987). Others exhibit increased REM latencies and less total REM time (Lavie, et al., 1979).

Panic Disorder

Panic attacks during sleep are sudden awakenings associated with intense fear. Although very similar to sleep terrors, panic attacks do not begin with a piercing scream and they occur in stage 2 or 3, rather than stage 4 sleep. Sleep panic attacks usually occur in individuals with daytime panic attacks. Out of a group of 45 patients with panic attacks, 31 (69%) reported having had at least one nocturnal panic attack in their lives. Occasional or frequent sleep panic attacks were reported by 15 (33%) of the group (Mellman & Uhde, 1989b).

Several findings emerge consistently in the literature on sleep panic attacks. Panic attack patients have increased sleep latencies, decreased sleep efficiency, and increased duration and number of body movements (Hauri, et al., 1989; Mellman & Uhde, 1989a, 1989b; Uhde, et al., 1985). There is no consistent evidence of any abnormality of REM in panic attack patients.

Roy-Byrne, et al. (1986) examined the effects of a night of total sleep deprivation on patients with panic disorder, along with depressed patients, and normal controls. Unlike the improvement in mood and anxiety ratings evidenced in the depressed patients, the panic attack patients and controls showed no significant effects. However, when analyzing the data from the patients with panic disorder, two subgroups emerged. One group showed no change, while the other actually displayed an increase in next-day panic attacks and anxiety ratings. Although panic disorder and depression have been hypothesized to be related because both respond to antidepressants, the sleep findings indicate independent disorders. Patients with nocturnal panic have been found to respond to cognitive-behavioral treatments for panic disorder (Craske & Rowe,

1997) indicating that the same principles are likely operating in both types of panic disorder patients.

Alcoholism

Insomnia or excessive sleepiness is a common feature of alcoholism (ASDA, 1997). Alcohol often enhances early slow wave sleep in alcoholics while decreasing early REM. There is increased REM and wakefulness during the second half of the night. The acute effects of withdrawal are somewhat inconsistent. Sleep may be more fragmented soon after the cessation of drinking, with associated increases in REM sleep (Johnson, 1972). In two samples of non-depressed alcoholics, shortened REM latency and increased REM percentage upon admission to an alcohol treatment program predicted relapse within a 3-month abstinence period (Gillin et al., 1994). Fragmented and reduced slow-wave sleep has been recorded in recovering alcoholics as long as two years after drinking cessation (Adamson & Burdick, 1973). Acute alcohol use may also precipitate or exacerbate snoring and sleep apnea (ASDA, 1997).

Sleep Disorders Associated With Neurological Disorders

There are a variety of neurological disorders that affect sleep. For more detail see Aldrich (1994), Bliwise (1994b), and Lugaresi, et al. (1988).

Cerebral Degenerative Disorders

Cerebral degenerative disorders encompass a group of heterogeneous diseases (e.g., Huntington's disease, torsion dystonia, supranuclear palsy, and multiple system atrophy) affecting one or more systems and are progressive conditions characterized by abnormal behaviors or involuntary movements, often with evidence of motor system dysfunction. Complaints of insomnia and hypersomnia are common. Sleep disturbance manifestations of these disorders include sleep fragmentation, abnormal motor activities, and disorganization of the circadian sleep-wake cycle. Polysomnography may reveal sleep fragmentation, decreased slow-wave sleep or REM sleep, disordered breathing, fragmentary myoclonus, periodic arm or leg movements, dystonic postures, and prolonged tonic contractions of one or more limb (ASDA, 1997; Chokroverty, 1996).