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SLEEP DISORDERS

Evaluation and Management in the Office Setting

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Introduction

The evaluation and treatment of sleep disorders constitute an important area of psychiatric practice. Psychological factors are prominent in the etiology of insomnia, certain cases of hypersomnia, secondary enuresis, and in cases of adults who suffer from sleepwalking, night terrors, and nightmares. In childhood the development of sleepwalking, night terrors, and nightmares is usually related to maturational factors; psychiatric disturbances are occasionally primary. In other sleep disorders such as narcolepsy, primary enuresis, and sleep apnea, psychological factors are rarely causative. However, since these disorders often have extensive psychosocial consequences, they are frequently the cause of psychological disturbances.

Disturbance of sleep is a common symptom of such psychiatric conditions as depression, mania, schizophrenia, or anxiety. Even when disturbed sleep is associated with a medical illness, psychological factors such as anxiety, depression, insecurity, lowered self-esteem, and fear of a more permanent invalidism or death are often causative.

The prevalence of sleep disorders in the general population is quite striking. In a survey of representative households in the Los Angeles metropolitan area, the overall prevalence of current or past sleep disorders in adults was 52.1 percent. The prevalence of specific sleep problems was as follows: insomnia, 42.5 percent; nightmares, 11.2 percent; excessive sleep, 7.1 percent; and sleepwalking, 2.5 percent. In many cases, these conditions were chronic, having started early in life. The prevalence of insomnia, nightmares, and hypersomnia was higher among individuals with a lower socioeconomic status or educational attainment. In addition, the presence of these disorders was correlated with more frequent complaints of general physical and mental health problems. Previous studies have reported that mental disturbances occur more frequently in groups with limited education and lower income. Consequently, it is not surprising that the development and persistence of sleep disorders in adults is more prevalent among individuals of lower socioeconomic and educational status.

Sleep disorders are frequently encountered in general medical practice. A survey of nearly 5,000 physicians determined the prevalence of sleep disorders seen within each major medical specialty. Physicians reported that an average of 17 percent of their patients had insomnia; the highest prevalence of insomnia, 32.4 percent, was reported by psychiatrists. The estimated prevalence of the other sleep disorders was as follows: nightmares, 4.3 percent; hypersomnia, 2.9 percent; enuresis, 2.2 percent; night terrors, 1.2

percent; narcolepsy, 0.6 percent; and somnambulism, 0.6 percent. Insomnia, nightmares, and hypersomnia were reported most frequently by psychiatrists and child psychiatrists; narcolepsy was encountered most often by neurologists; and enuresis, somnambulism, and night terrors were most often seen by child psychiatrists and pediatricians. Areas of high-population density had a greater prevalence of insomnia, hypersomnia, night terrors, and nightmares. These data show that physicians, particularly psychiatrists, need to keep current with the considerable clinical advances that are being made in the etiology, assessment, and management of sleep disorders.

At the present time there is no official diagnostic classification for sleep disorders. The proposed classification of sleep disorders, which is included as an appendix in the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III), has a number of major shortcomings that seriously limit its usefulness. There are about seventy diagnoses, many of which are either unsubstantiated or based on electrophysiologic criteria that have relatively little clinical significance. Also, this classification does not follow the DSM-III format. While the DSM-III encourages the use of multiple, separate diagnoses within a multiaxial system, the sleep classification consistently combines diagnoses—an approach that is confusing and misleading. It is probably for these reasons that this "sleep appendix" has limited applicability from a clinical standpoint and is therefore uncoded.

The International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM), on the other hand, provides succinct and practical diagnoses for sleep disorders, particularly in the section dealing with sleep disorders of nonorganic origin. This chapter basically follows the format and most of the individual diagnoses included in the ICD-9-CM. Included are discussions of insomnia, disorders of excessive sleep (narcolepsy, the hypersomnias, and sleep apnea), and episodic sleep disturbances (sleepwalking, night terrors, nightmares, and enuresis).

Insomnia

Insomnia is the most frequently encountered sleep disorder. In several national or regional surveys, the prevalence of insomnia has been estimated at 30 to 35 percent.- Although the term "insomnia" literally denotes a complete lack of sleep, it is used to indicate a relative inability to sleep, that is, difficulty in falling asleep, difficulty staying asleep, early final awakening, or combinations of these complaints.

Sleep laboratory studies demonstrate a positive correlation between subjective complaints of insomnia and the objective measurements of sleep disturbance. Compared with age-matched controls, insomniac subjects show increased objective values for sleep latency, wake time after sleep onset, and total wake time. However, the subjective estimates of their sleep difficulty are

usually exaggerated. Thus, the clinical complaint of insomnia, although usually valid, is often overestimated. When compared with controls in terms of sleep stages, insomniacs have less stage 4 sleep, and the amount of rapid-eye-movement (REM) sleep they obtain is either similar or slightly less. In addition, insomniacs vary considerably from night to night in terms of sleep latency, wake time after sleep onset, stage REM, and stage 4 sleep.

The sleep of insomniacs is different qualitatively as well as quantitatively from the sleep of normal subjects. Poor sleepers have higher levels of physiological arousal both prior to and during sleep; heart rate, peripheral vasoconstriction, and rectal temperature are elevated both before and during sleep, and they have more body movements during sleep. Also, insomniacs show a degree of physiological instability in the production of sleep spindles from night to night.

Causative Factors

In most cases, chronic insomnia is secondary to psychological disturbances. When medical conditions and aging are excluded, about 80 percent of patients with chronic insomnia have one or more elevated Minnesota Multiphasic Personality Inventory (MMPI) scales. These patients usually fall within the diagnostic categories of the neuroses or personality disorders, and have a history of chronic anxiety or depression, or both. The

most frequently elevated MMPI scales are depression, conversion hysteria, psychopathic deviate, and psychasthenia.

Analysis of the MMPI patterns of insomnia patients strongly suggests that many insomniacs characteristically internalize their emotions rather than express them outwardly. The internalization of psychological conflicts may lead to chronic emotional arousal, which in turn leads to physiological arousal and, consequently, insomnia. The fact that poor sleepers have higher levels of physiological arousal during sleep supports this hypothesis. During the day, the insomniac typically tries to deny and repress conflicts. At night, however, as external stimulation and distractions wane, attention is focused internally, the individual relaxes, and regression occurs. Feelings of anger, aggression, and sadness threaten to break through into consciousness and, as the insomniac fights to ward off preconscious thoughts, sleeplessness worsens. Thus, in addition to the underlying psychological disturbances, a fear of sleeplessness, which is independent of the primary psychological causes, soon develops in insomnia patients. Also, a chronic pattern of disturbed sleep eventually conditions the patient to expect insomnia. The ultimate result is a vicious circle of continued sleep disturbance, with an escalation of psychological conflict, physiological arousal, sleeplessness, fear of sleeplessness, further psychological arousal and still further sleeplessness.

Sleep disturbance is an extremely common complaint of patients who

have significant psychiatric problems; 70 to 75 percent of any given outpatient or inpatient psychiatric population have some type of sleep disturbance. For example, sleep disturbance is one of the most consistent symptoms of depressive illness. During the acute manic phase of manic-depressive illness, the typical patient has a marked reduction in total sleep, though he may not complain of a lack of sleep. In schizophrenic patients, the degree of sleep disturbance varies considerably, depending upon whether the process is acute or chronic. Severe sleep disturbances are frequent in acute schizophrenic episodes and often reach the point of complete sleeplessness.

Insomnia can also be caused by situational circumstances, medical conditions, or pharmacological agents. Situational insomnia is most often transient and related to major life events, such as difficulties with work or family, or personal loss. It is a universal but highly individualized phenomenon; almost everyone has experienced situational insomnia at one time or another. Insomnia is frequently a symptom in medical conditions that are accompanied by pain, physical discomfort, anxiety, or depression. Sleep difficulties can also result from the use of various pharmacological agents, such as amphetamines or other stimulants, steroids, central adrenergic blockers, bronchodilators, or the caffeine contained in colas and coffee. Also, an intense degree of insomnia, which is termed “rebound insomnia,” may follow the withdrawal of certain short- or intermediate-acting benzodiazepine hypnotics, even when they are given in single nightly doses for short periods.

"Drug-withdrawal insomnia" is produced by the abrupt withdrawal of large nightly doses of non-benzodiazepine hypnotics after prolonged administration.

The elderly, especially women, frequently complain of insomnia. Older people not only sleep less than younger persons and have many nocturnal awakenings, but the amount of stages 3 and 4 sleep they obtain is markedly reduced and may even be absent. The aging process itself may contribute to sleep difficulty; the elderly often have medical illnesses, in which pain and discomfort cause frequent sleep disturbance. Also, as the older person faces the realities of declining function and inevitable death, he may become anxious, fearful, and depressed. Fear of death and uncertainty about the "after death" situation are more prominent during the night, when sleep is perceived as a transient deathlike state. Consequently, the process of falling asleep involves varying degrees of emotional regression.

Diagnostic Considerations and Procedures

Failure to take an adequate history is one of the most common shortcomings in the management of insomnia. A complete history includes a general sleep history, a psychological history, and a drug history.- It is essential to thoroughly describe the sleep problem, including the type(s) of insomnia, the nature of the onset of the problem and its duration (that is,

acute or chronic), the severity and frequency of the sleep difficulty, and the circumstances that precipitate or accentuate the insomnia. Critical information is obtained from the bed partner to rule out the presence of sleep apnea or myoclonus nocturnus. Assessment of the sleep problem should not only focus on an eight-hour, nightly period, but also cover the twenty-four-hour sleep-wakefulness pattern.

A complete psychiatric history is essential and can usually point out a relationship between the onset of sleep difficulties and psychological conflicts or major life-stress events. In taking a psychiatric history, the physician should be aware that insomniac patients tend to focus on symptoms that are ego-syntonic and socially acceptable, such as the complaint of insomnia itself, as opposed to such ego-dystonic symptoms as depression and impotence. The psychiatric history should include a thorough sex history and should assess for symptoms suggestive of endogenous depression or a psychoneurotic disorder.

The drug history should evaluate the patient's consumption of coffee, colas, or alcohol. Excessive caffeine may result in difficulty in falling asleep, whereas alcohol causes difficulty in staying asleep. The drug history also assesses previous pharmacological treatment for insomnia or other disorders. Therapy with a tricyclic antidepressant is often unsuccessful because the daily dosage has been insufficient or excessive daytime sedation has been caused

by daytime administration of the medication. Overtreatment is frequently a problem with hypnotic drugs and is in large part caused by the relative ineffectiveness of these drugs over intermediate- or long-term use. The loss of efficacy may cause the patient to increase the dose. Under such conditions, drug-withdrawal insomnia may occur when abrupt withdrawal is attempted.

Nonpharmacologic Management and Psychotherapy

Since transient insomnia is most often a reaction to physical or psychological stress, it usually subsides when the individual adapts through his own coping mechanisms. If elimination of the stress-generating situation is impossible or impractical, the main therapeutic role of the physician in these cases is to help the patient strengthen these mechanisms. In treating chronic insomnia, however, the physician must be aware that the disorder is multidimensional: Any approach that is directed to only one of the factors involved will usually be inadequate or unsuccessful. In general, the most effective treatment for the patient with insomnia combines the following elements: (1) general therapeutic measures; (2) supportive, insight-oriented and behavioral psychotherapeutic techniques; and (3) appropriate adjunctive use of antidepressant or hypnotic medication.

General therapeutic measures may be applied in most cases of insomnia. The patient should be encouraged to increase his physical activity and

exercise during the day, but not close to bedtime since exercise at that time may heighten physiologic arousal. Complex mental activity, such as studying, especially late in the evening, may aggravate insomnia; thus, patients are instructed to avoid mentally stimulating situations and engage in relaxing mental activities prior to bedtime. In addition, patients should regulate their daily schedules and establish a regular bedtime hour, although the patient's sleep schedule should remain flexible so that he does not become unduly obsessive about the schedule itself. The insomniac should go to bed only when he is sleepy. If he is unable to sleep, he should get up, leave the bedroom, and engage in some kind of relaxing activity. In this way, the patient learns to associate the bedroom with sleep rather than with obsessive thoughts and concerns. Naps during the day should be discouraged. Finally, cigarette smoking and drinking beverages containing caffeine should also be discouraged, particularly close to bedtime.

Support, education, and reassurance often help to minimize the patient's fear of sleeplessness and the consequences of insufficient sleep. Patients are often obsessed with the fear that their insomnia will severely affect their physical health and even cause their death. It can be very helpful to explain to the patient how his own anxiety can become part of the vicious circle that exacerbates and maintains insomnia.

Behavioral treatment approaches that minimize rumination and help

the patient to focus his attention can be beneficial. In this regard, relaxation training combined with suggested pleasant imagery is therapeutic, since it focuses the thoughts of the insomniac patient on a positive or neutral theme, helping him to avoid rumination by shifting his attention from the internal to the external. Accordingly, attention and patterned thought are substituted for the ruminative concerns that maintain the insomniac's high levels of cognitive arousal. One advantage of behavioral therapy is that the individual's active participation reduces his feelings of passivity and helplessness. The effectiveness of behavioral therapies in treating insomnia is generally limited, however, to patients who have difficulty falling asleep. In addition, behavioral approaches require a much greater degree of cooperation and compliance by the patient.

In treating the psychological aspects of insomnia, the psychiatrist needs to be active and direct in exploring conflict areas rather than using a gradual, uncovering approach. Insomniacs deny the psychologic conflicts that underlie their sleep disorder. Instead, they focus on the somatic aspects of their problem and are mainly interested in symptomatic relief. If areas of psychological conflict are delineated early in treatment, these patients are more likely to become actively involved in therapy.

Psychodynamic techniques allow for delineating and resolving the original conflict that underlies the insomnia and its development. A chronic

insomniac patient may fear going to bed because of suppressed memories of traumatic events and experiences in childhood or adolescence that were associated with sleep or bedtime (for example, parental drunkenness, domestic violence, or incest). Insomniac patients often have difficulty expressing and controlling their aggressive feelings. Going to sleep represents a loss of control, and insomnia is a defense against this fear.

Insight-oriented psychotherapy thus provides a means for dealing with current, unexpressed, psychological conflicts and emotions that predispose the patient to emotional and physiological arousal at night. For the insomniac, getting in touch with anger, overcoming binding inhibitions and apprehensions, and restoring the balance of outwardly expressed versus self-restrained aggression are important benefits from psychotherapy. This, of course, minimizes the potential for emotional release and arousal at bedtime.

Sexual difficulties, such as avoidance of sexual relations and unsatisfactory sexual relations, are frequent in patients with insomnia. These problems may be related to general interpersonal difficulties with the spouse, to fear of aggression, or attempts to control or manipulate the spouse through the sexual aspects of their relationship.

Drug Treatment

When patients with transient insomnia are severely anxious,

prescribing anxiolytic medication as an adjunctive treatment for a short time can be helpful. In treating chronic insomnia, the use of hypnotic medication should be only an adjunct to the main therapeutic procedures. It is particularly helpful in breaking the vicious circle of insomnia, fear of sleeplessness, emotional and physiologic arousal, and further insomnia. The issues involved in the pharmacological treatment of transient and chronic insomnia are similar, except that the lack of continued effectiveness of the medication is an important additional consideration when treating chronic insomnia.

Barbiturates should generally be excluded from the adjunctive pharmacotherapy of insomnia. They have many shortcomings, including interaction with anticoagulants, and a high potential for drug-withdrawal insomnia and drug dependence. Most important, the potential for a lethal overdose is very high with barbiturates. The benzodiazepine hypnotics are preferred over barbiturates because they have a very wide therapeutic window and can be used with relative safety. Most of them are effective for a period of one to two weeks, that is, within the usual treatment period of transient insomnia. Nevertheless, all benzodiazepines are not alike. Those with shorter half-lives have been shown to produce rebound insomnia even after brief periods of nightly administration of single, therapeutic doses. On the other hand, long-acting benzodiazepine hypnotics may reduce performance during the day following their bedtime administration. Certain

benzodiazepines may also cause anterograde amnesia.

The clinician needs to be aware of the side effects of the benzodiazepines he chooses to prescribe, and he should instruct his patient to use the medication cautiously. With shorter-acting benzodiazepines, tapering of the dose before complete withdrawal is necessary to minimize difficulty with rebound insomnia. The potential for impairment of memory or daytime performance should be dealt with by educating the patient about the possibility of their occurrence with certain benzodiazepines and cautioning him to avoid tasks that necessitate heightened memory or performance capabilities.

Although L-tryptophan has been proposed as a sleep-inducing agent, the hypnotic efficacy of this compound has not been proven. This failure to demonstrate effectiveness may be due to methodological problems, since most of the existing studies to date involve non-insomniacs. Similarly, the effectiveness of over-the-counter "hypnotics" has not been established, and these compounds should not be used. Sleep-laboratory assessment of these drugs suggests that they are ineffective. Moreover, over-the-counter drugs containing scopolamine can be hazardous since even in the recommended dose range they may precipitate acute glaucoma, especially in elderly patients who have a narrow corneal-iris angle. Also, a dose of two to three times the recommended dosage may produce transient disorientation and

hallucinations.

To date, only two hypnotics, nitrazepam and flurazepam, have been found to be effective for more than a two-week period. However, only flurazepam is available in this country. With most patients, treatment should be initiated with a dosage of 15 milligrams (mg) of flurazepam at bedtime. If after one to two weeks the 15 mg dose does not sufficiently improve sleep, the dosage should be increased to 30 mg. When administering flurazepam, the physician should alert the patient to possible decrements in his daytime performance. Also, the physician should avoid increasing the dose in the elderly and in individuals with impaired metabolic systems, since active metabolites of the drug are more likely to accumulate in these individuals.

Withdrawal of flurazepam is facilitated by its carry-over effectiveness. On the first and second nights following withdrawal of flurazepam, sleep is still significantly improved, whereas upon withdrawal of most hypnotic drugs, sleep difficulty returns immediately to predrug levels and, in some cases, rebound insomnia occurs.

When anxiolytics or antidepressants are properly used in the pharmacotherapy of insomnia, they reduce the anxiety or depression underlying the insomnia, and their sedative side effects help to ameliorate the sleeplessness itself. When insomnia is secondary to anxiety states, the use of

anxiolytic benzodiazepine drugs (for example, clorazepate, chlordiazepoxide, diazepam, or lorazepam) is indicated for both the daytime anxiety and, in an increased bedtime dose, the sleep difficulty. Insomnia associated with agitated depression should be treated with an antidepressant (for example, amitriptyline or doxepin). Administration of most or even all of the daily dose of the sedative antidepressant at bedtime not only alleviates sleeplessness, but also reduces daytime sleepiness.

Insomnia associated with retarded depression may be treated with an energizing tricyclic antidepressant, such as imipramine, which may be used in divided doses during the day together with a benzodiazepine hypnotic, such as flurazepam, at bedtime. When the therapeutic effects of the tricyclic have been established, which takes about two to three weeks, flurazepam can be discontinued, since relief of insomnia usually accompanies improvement of the underlying depression.

A sufficient dose level is critical to the effectiveness of antidepressant medications. A major problem with the tricyclic antidepressants that have sedating side effects is that many patients are given too much of their medication during the day rather than at bedtime. The sedative side effects of the tricyclic antidepressants are immediate, while the antidepressant effect is more likely to occur after about ten days. Confusing the side effects of these drugs with their basic action may account for much of the difficulty in

attaining a sufficient total daily dosage, and also in appropriately adjusting the daytime-to-bedtime dose ratio.

On the other hand, the administration of a large bedtime dose of a sedative tricyclic antidepressant should be implemented gradually. In this manner, the psychiatrist can avoid severe decrements in performance the following day, as is often the case when a sedative tricyclic is initiated in a bedtime dose of 75 to 100 milligrams.

For the treatment of insomnia associated with schizophrenic psychosis or the manic episodes of manic-depressive psychosis, neuroleptics with sedative properties, such as chlorpromazine and haloperidol, are indicated. Since a large bedtime dose is usually effective in controlling the patient's sleeplessness, administration of hypnotics can be avoided.

Narcolepsy

The disorders of excessive sleepiness are narcolepsy and the various hypersomnias, including those associated with sleep apnea. About 7 percent of the general adult population has had a complaint of excessive sleep at some time in their lives.

Recognition of the narcolepsy syndrome has important psychiatric implications since a lack of a definite diagnosis and therapy may have serious

psychosocial consequences for the patient. In addition, problems arising from prolonged administration of stimulant drugs when treating narcolepsy are of special interest to psychiatrists. Daytime sleep attacks, the primary component of the narcoleptic tetrad, may be associated with one or more of three auxiliary symptoms: cataplexy, sleep paralysis, and hypnagogic hallucinations. Narcoleptic sleep attacks may be as short as a few seconds in duration or as long as thirty minutes. In milder forms of narcolepsy, sleep attacks are precipitated by boring, monotonous, and sedentary situations; whereas in severe narcolepsy, sleep attacks may occur under circumstances which are quite stimulating or exciting, for example, work or sports activities and even sexual intercourse. Cataplexy, a sudden loss of muscle tone without loss of consciousness, may be complete, causing the patient to fall as if fainting. In less pronounced situations, the cataplexy may be partial, with simply a buckling of the knees or sagging of the facial musculature. Cataplectic attacks are generally quite brief, lasting from seconds to one or two minutes. The attacks are characteristically precipitated by strong emotions such as laughter, anger, or surprise. The remaining auxiliary symptoms of sleep paralysis and hypnagogic hallucinations occur during the transition between wakefulness and sleep. Episodes of sleep paralysis last from a few seconds to one or two minutes and may occur in normal individuals, leaving the individual fully conscious but unable to move. The hypnagogic hallucinations of narcolepsy are visual, auditory, or tactile perceptions that, in general, are

more vivid, emotionally charged, and unpleasant than those that sometimes occur in normal individuals.

Narcolepsy is not a rare disorder. The prevalence of narcolepsy is estimated to be from 0.02 to 0.07 percent of the general population. The actual prevalence is most likely higher than reported, however, since the disorder is frequently misdiagnosed. The incidence in men and women is about equal. Narcolepsy usually appears between the ages of ten and thirty, and in 90 percent of the cases, onset is prior to age twenty-five.

The narcoleptic tetrad, especially the auxiliary symptoms, corresponds to manifestations of REM sleep. Narcoleptic patients who also have cataplexy have REM periods at or close to sleep onset, rather than after the normal seventy to ninety minutes of non-REM (NREM) sleep. Also, sleep attacks are about as long as REM periods, that is, fifteen to thirty minutes. Furthermore, the loss of muscle tone in cataplexy is a reflection of the motor inhibition of REM sleep, and hypnagogic hallucinations correspond to the dream activity associated with REM sleep. In those narcoleptic patients who do not have associated auxiliary symptoms (independent narcolepsy), sleep-onset REM periods do not usually occur.

Disturbance of nocturnal sleep is common in narcolepsy. The sleep disturbances may consist of generally restless sleep, frequent nocturnal

awakenings, or less total sleep time. Since sleep attacks are reported to be more frequent after a night of poor sleep, the physician needs to be concerned about the patient's nocturnal sleep and especially how it may be adversely affected by stimulant medication taken late in the day or in the evening. Excessive nocturnal sleep is not characteristic of narcolepsy.

Causative Factors

Genetic factors are important in the development of narcolepsy; narcoleptic patients frequently have positive family histories either for narcolepsy or hypersomnia. In a study of narcoleptic probands, it was found that the prevalence of narcolepsy among relatives was 2.5 percent, a rate about sixty times greater than the estimated prevalence in the general population.

The exact pathophysiology of narcolepsy/cataplexy has not been determined; however, it is clear from the absence of epileptic discharges in the electroencephalogram (EEG) that narcolepsy is not related to epilepsy. Narcoleptic patients show a high degree of psychological conflicts, but these generally appear to be consequences of the disorder rather than causative factors.

Diagnostic Considerations and Procedures

A thorough history is important in establishing the diagnosis of narcolepsy and initiating treatment. The physician must determine the age of onset for narcolepsy, the frequency and duration of episodes, the time of their occurrence, the circumstances precipitating the symptoms, and the general daytime behavior and adjustment. If cataplexy and other auxiliary symptoms are present, the diagnosis is easily made on a clinical basis. If sleep attacks are the only symptom, an all-night hypno-polygram and a daytime-nap EEG recording may confirm the diagnosis by detecting a sleep-onset REM period. If the hypno-polygram and the nap EEG are negative, the physician again proceeds on the basis of the clinical symptomatology.

A thorough history is usually sufficient to differentiate narcolepsy from other disorders. Narcolepsy is frequently misdiagnosed as hypersomnia, hypothyroidism, hypoglycemia, epilepsy, myasthenia gravis, or multiple sclerosis. The characteristics that differentiate narcolepsy from hypersomnia are the presence of auxiliary symptoms (cataplexy, sleep paralysis, or hypnagogic hallucinations), usually shorter daytime sleep episodes, an absence of post-sleep confusion, and an absence of prolonged nocturnal sleep. Narcolepsy is differentiated from epilepsy by the presence of auxiliary symptoms, the absence of bladder or bowel incontinence and tongue biting, and the lack of clinical EEG abnormalities characteristic of epilepsy.

Therapy

Nonpharmacologic Management and Psychotherapy

Narcoleptics often are considered by their family, friends, fellow employees, and employers as being lazy, malingering, or psychologically disturbed.- The physician should inform the patient and the people who comprise his family, social, and occupational lives that the sleep attacks and other symptoms are beyond the patient's control. Since it is not uncommon for narcoleptic patients to be terminated from employment, it is sometimes helpful to suggest to the employer that he allow the patient one or two short naps every day. Such naps often significantly improve work performance.

Narcoleptic patients should be advised of the potential dangers of long-distance driving or other activities that would expose them to danger if a sleep attack or cataplectic episode were to occur. The physician has to be judicious whenever advising restriction of activities to the narcoleptic patient. Warnings should not exceed those warranted by the clinical course of the illness before, and especially after, treatment.

Psychotherapy alone, of course, is not effective in treating narcolepsy itself, since accompanying psychological difficulties are secondary rather than primary. Supportive psychotherapy is helpful, however, as an adjunct to the pharmacological treatment of narcolepsy, since alleviation of the serious psychosocial consequences of the disorder facilitates the overall treatment.

Drug Treatment

The sleep attacks of narcolepsy can be effectively treated with stimulant medication such as the amphetamines and methylphenidate.'' Methylphenidate has been recommended as the drug of choice for the management of narcoleptic sleep attacks because of its prompt action and the relatively low incidence of side effects. Since this drug has a short duration of action and is effectively absorbed only from an empty stomach, patients should take the medication at least forty-five minutes before, or more than one hour after, eating. Initially, patients take 5 mg upon awakening in the morning, at midday, and again at 4 P.M. Depending upon the patient's initial response and the time of day when sleepiness is worst, one or all of the three doses may be increased as high as 30 to 40 mg.

Methamphetamine is the most tolerated of the amphetamines since it causes fewer sympathomimetic side effects. Initially, patients may be given 10 mg upon arising in the morning. They are then assessed so that the effectiveness and approximate duration of action of a single dose can be determined. Subsequently, this dose can be gradually increased until the appropriate dose level is reached, at which point a regimen is developed to ensure alertness throughout the day. Many patients require only a single dose, others may need two doses, and a few may require three doses a day. The total daily dose of methamphetamine may be as low as 20 mg for mild

narcolepsy, or higher than 100 mg for severe narcolepsy.

Treatment of narcolepsy with amphetamines, and to a lesser degree with methylphenidate, is complicated by the many problems that are associated with these drugs. For example, psychiatric syndromes may complicate amphetamine treatment; a syndrome of irritability, paranoid tendencies, and even psychosis may develop with prolonged amphetamine use. The incidence of these side effects, however, even with high doses of amphetamines, is lower in narcoleptic patients than it is in other individuals taking these drugs. Stimulant drugs may also disturb nocturnal sleep, particularly if they are taken in the late afternoon or early evening. This may cause the patient to take more drug in the daytime in order to ward off sleep attacks, which occur more frequently when disturbed nocturnal sleep is present.

Recent data suggest that propranolol in a dose range of 240 to 480 mg may be effective in treating the sleepiness and sleep attacks of narcolepsy. In most cases, however, the beneficial effects do not extend beyond several months.

When the auxiliary symptoms of narcolepsy are present, the drugs of choice are antidepressant medications such as the tricyclic drugs. The most frequently used drug for treating the auxiliary symptoms has been

imipramine. In managing cataplexy (the most frequent and disturbing auxiliary symptom), imipramine has a rapid onset of action (within hours in some cases) and is effective at lower doses than those used to treat depression. While it is extremely effective in relieving auxiliary symptoms, imipramine is only minimally effective in decreasing sleep attacks.

When the patient complains of both sleep attacks and auxiliary symptoms, treatment with the analeptics and imipramine may be combined. Since this combination may produce serious side effects such as hypertension, dose increments must be carefully titrated and monitored. As suggested by the manufacturer, methylphenidate may inhibit the metabolism of tricyclic antidepressants so that downward dosage adjustment of imipramine may be necessary.

Hypersomnia

Hypersomnia is generally characterized by periods of excessive daytime sleepiness and sleep attacks that usually are longer than narcoleptic sleep attacks; episodes of hypersomnia may last from one to several hours, f Nocturnal sleep is often prolonged in hypersomnia; otherwise it is not usually disturbed. Hypersomnia usually begins later in life, often between thirty to forty years of age. Approximately one-third of patients with hypersomnia experience sleep drunkenness, which consists primarily of difficulty in fully

awakening in the morning and lasts from fifteen minutes to an hour.

Hypersomnia may be idiopathic, psychogenic, periodic, associated with sleep apnea, or secondary to other organic conditions.* In the idiopathic type there is often a positive family history. About 40 percent of the primary probands among hypersomniac patients have a family history of hypersomnia or narcolepsy, suggesting that there is an autosomal dominant mode of genetic transmission for this disorder.

In the sleep laboratory, hypersomniacs average close to nine hours of total sleep time. The sleep-stage distribution is similar to normal, and although a decrease in autonomic functioning would be expected in these patients, their heart rate is actually higher than that of controls.

Psychological factors are quite important in the hypersomnias, specifically in the psychogenic type.- Psychogenic hypersomnia is usually secondary to a depressive disorder and is characterized by nocturnal sleep that is not only prolonged but also restless and generally disturbed. These patients also feel worse upon arising in the morning, but their symptomatology is distinct from that of sleep drunkenness. In psychogenic hypersomnia, the underlying depression may be “atypical” and therefore difficult to diagnose. More often, it may represent the depressive phase of a bipolar affective disorder.

The Kleine-Levin syndrome, which is the most frequent type of periodic hypersomnia, is nevertheless an extremely rare condition. The excessive sleepiness and sleep usually last two or three days, occur once or several times a year for several years, and then terminate. This condition is usually associated with excessive appetite and is most common in adolescent males.

Sleep apnea is frequently associated with excessive daytime sleepiness or hypersomnolence. f In patients with sleep apnea, there may be sleep attacks, which are often indistinguishable from narcoleptic sleep attacks.

There are three types of sleep apnea: central, peripheral, and mixed. Central apnea is a cessation of air flow through the nares and mouth accompanied by a cessation of thoracic and abdominal respiratory efforts. In peripheral or obstructive sleep apnea, no air flow is recorded at the entrance to the upper airway in spite of persistent thoracic and abdominal respiratory movements. In mixed apnea, a brief period of central apnea is quickly followed by a longer period of obstructive apnea.

When hypersomnia is related to sleep apnea, the polygraphic sleep recording typically shows repetitive respiratory pauses in both REM and NREM sleep. The apneic episodes are usually of the peripheral type, indicating obstructive rather than central sleep apnea. The exact pathophysiologic mechanism of the upper airway obstruction associated with sleep apnea is

unclear.

Organic hypersomnia is usually secondary to tumors, vascular lesions, or trauma involving the mesodiencephalic area of the brain stem, resulting in a disturbance of the reticular activating system. Other causes of organic hypersomnia include infectious and toxic encephalopathies as well as several endocrine and metabolic disorders.

Diagnostic Considerations and Procedures

The history from the hypersomnia patient should include the age of onset, the frequency and duration of episodes, their time of occurrence, precipitating circumstances, the ability to resist sleepiness, the presence or absence of auxiliary symptoms and sleep drunkenness, the duration and quality of nocturnal sleep, the presence or absence of snoring or interrupted breathing during the night, and the patient's general daytime behavior and adjustment.

Questioning the spouse, parent, or other family members for the presence of snoring with interrupted nocturnal breathing is important in diagnosing sleep apnea. The physician determines if there are frequent periods of interrupted nocturnal breathing associated with snoring, gasping, gurgling, choking, periodic loud snorting, or morning headache. The snoring associated with sleep apnea is unique: There are ten- to forty-second periods

of suspended respiration followed by very loud and abrupt snorting sounds that are two to four seconds in duration. The common type of snoring in non-apneic individuals is not as loud, fluctuates in intensity, and is continuous without any gaps of appreciable duration.

It is important to differentiate hypersomnia from narcolepsy. In hypersomnia, there are no auxiliary symptoms, the daytime sleepiness usually begins later in life, there are generally fewer but longer episodes per day, and nocturnal sleep is often prolonged but not disturbed. A detailed psychiatric history is necessary in order to rule out depression as a causative factor. Also, whenever hypersomnia is suspected, it is important to rule out thyroid disease, hypoglycemia, and diabetes mellitus.

Therapy

Nonpharmacologic Management and Psychotherapy

The physician should educate the hypersomniac patient and his family regarding the symptomatology of hypersomnia and clarify any misconceptions they may have about the disorder. Supportive, and less frequently, insight-oriented psychotherapy may be therapeutic for certain patients with hypersomnia. For example, when the patient sleeps excessively in an attempt to deny and escape from life stresses, the physician might advise him to adopt a more assertive life style. If psychological difficulties

arise from the patient's family relations, family therapy may be more appropriate. In hypersomnia secondary to depressive illness, psychotherapy may be applied as an adjunct to the use of antidepressant medication.

In overweight patients with sleep apnea, weight reduction is first indicated and, at times, results in improvement. However, many patients will not maintain their diet. Patients with obstructive apnea may require tracheostomy, which is almost invariably effective in alleviating both the sleep apnea and the excessive daytime sleepiness.-' At present, an effective treatment of central sleep apnea has not been well established.

Drug Treatment

Stimulant drugs are indicated for treating the excessive daytime sleepiness, the prolonged nocturnal sleep, and sleep drunkenness of idiopathic hypersomnia.- Methylphenidate is the drug of choice, primarily because of its prompt action and fewer side effects. For sleep drunkenness, the drug is either given at bedtime, or the hypersomniac patient is awakened and given the medication thirty minutes before the desired time of awakening. For excessive daytime sleepiness, drug administration is scheduled according to the patient's needs for full awareness.

In psychogenic hypersomnia, when endogenous or characterological depression is the primary difficulty, non-sedating tricyclic antidepressants

such as protriptyline are indicated. Lithium can be used to treat the hypersomniac patient who is in a depressive phase of a bipolar disorder. The patient with an "atypical" depression, however, may respond more favorably to a mono-amine oxidase inhibitor. The sedating tricyclics are contraindicated in all cases.

Sleepwalking

Sleepwalking, or somnambulism, is not uncommon in childhood, but it is rather infrequent in adults. It is a state of dissociated consciousness in which phenomena of the sleeping and waking states are combined. The episodes occur out of stages 3 and 4 sleep, when dream recall is less frequent, rather than out of REM sleep, when dreaming is most likely. Sleepwalking is therefore not the acting out of a dream, as was once commonly believed.

Somnambulism commonly begins in childhood and less often in early adolescence. When sleepwalking is outgrown, its onset was almost invariably before age ten, whereas individuals who continue to sleepwalk as adults have a significantly later age of onset. In addition, those who do not outgrow sleepwalking have more frequent events, experience episodes earlier in the night, and have more intense clinical manifestations of their events.

The typical somnambulistic episode occurs within the first three hours of sleep; the patient sits up, rises out of bed, and moves about slowly in a

poorly coordinated, automatic manner. Sometimes there is more complex activity, which is clearly out of context and reflects the patient's lack of awareness. Effort is usually required to awaken the patient. Unless fully aroused the patient is usually amnesic toward the episode.

Causative Factors

Sleepwalking is a disorder of impaired arousal. In addition to the somnambulist's general confusion and lack of responsiveness during an episode, reactions to even strong stimuli are impaired.

Etiological factors in sleepwalking appear to include both physical and psychological components. An increased familial occurrence of sleepwalking has been identified. Families of sleepwalking probands also show a higher prevalence of night terrors. These data appear to fit a two-threshold, multifactorial model of inheritance in which sleepwalking is the more prevalent condition.

Maturational/developmental delay is considered a significant etiological factor in sleepwalking because most sleepwalkers outgrow the disorder before adulthood. Furthermore, it has been found that an "immaturity factor" in the sleep EEG (that is, sudden, rhythmical, high voltage, slow activity) persists to a later age in child somnambulists than in normal children. Child somnambulists, however, do not show appreciably more psychopathology

compared with normal children.

Adult sleepwalkers may have been affected by a delay in central nervous system (CNS) maturation, but psychological factors also appear to play an important role in the condition's development and, particularly, in its persistence. Sleepwalkers who do not outgrow their disorder show much more psychopathology compared with both past sleepwalkers and normal controls. Their MMPI profiles are consistent with active, outwardly directed, aggressive behavioral patterns. Individuals with this type of profile commonly struggle to deal with life frustrations and become extremely angry when frustrated. Excessive anger in response to frustration, failure, or loss of self-esteem is outwardly directed rather than being self-directed, as is the case in depression.

Diagnostic Considerations and Procedures

Evaluation of the child or adult who presents with a complaint of sleepwalking should begin with the taking of a thorough history. This includes determining age of onset, frequency of episodes, time of occurrence, behavior during the episode, length of episode, degree of recall of mental activity, general daytime behavior and adjustment, major life-stress events associated with onset, and the effects of stressful situations on frequency of episodes.

Much of this information has to be obtained from the parents or, in the

case of adult somnambulists, from the spouse or roommate. A detailed history will often help to determine whether the disorder is due to underlying organic factors or psychopathology. Underlying psychopathology is suggested when sleepwalking begins after about age ten; when it occurs frequently over a period of six months to a year; when there is a negative family history of sleepwalking; when daytime symptoms suggest a functional disorder; and when the onset of sleepwalking appears to be related to major life events or when mental stress increases the frequency of episodes.

On occasion, it may be necessary to differentiate sleepwalking from other conditions, such as dissociative states or nocturnal wanderings. In contrast to sleepwalkers, individuals in dissociative states are much more alert and are capable of performing more complex activities. Their episodes may last for periods of hours to days. A complaint of sleepwalking in the elderly may actually represent a nocturnal wandering episode, which can be easily differentiated from sleepwalking by the history. Nocturnal wandering episodes most frequently occur in patients who have organic brain syndromes with associated intellectual impairment, and they may occur at any time during the night.

Drug-induced, somnambulist-like episodes have been observed to be relatively frequent in psychiatric patients who have taken lithium together with neuroleptics. These patients did not have a prior history of

somnambulism. Also, febrile illness has been found to induce somnambulistic episodes. When the episodes persist long after the febrile illness, there is usually a positive family history for sleepwalking or night terrors.

Therapy

Nonpharmacologic Management and Psychotherapy

An important aspect of the management of a sleepwalker is to recommend appropriate safety measures. These may include sleeping on the ground floor and providing special bolts or locks for windows and doors. The sleepwalker generally returns to his room on his own, but if he does not, he usually can be gently led back. The sleepwalker's episodes should not be interrupted, however, if it is known that such intervention may lead to more confusion and fright.

Any groups or agencies, such as summer camps, that are responsible for children's' safety on overnight outings away from home should be made aware of a patient's sleepwalking and the need for safety measures.

Sleepwalking poses special problems in the armed forces; thus a number of studies have been conducted in this setting.'' In general, the nocturnal behavior of the sleepwalker is inconsistent with military standards of discipline. The sleep of fellow soldiers may be disturbed by the

sleepwalker's activity, there may be an uneasy feeling that there is an "odd" comrade in the barracks, and there is a risk that the somnambulist may be shot by failing to halt at a guard's command. In the navy, a sleepwalker may fall overboard during an episode.

Most children grow out of the sleepwalking condition, particularly if it has an early age of onset. Thus, the parents of children who sleepwalk need to be reassured that, in most cases, sleepwalking does not reflect a serious psychological disorder. On the other hand, adults who sleepwalk often have significant psychiatric disturbances that require psychotherapeutic intervention.

Since adult sleepwalkers typically cope with frustration in an externally directed manner rather than struggling with internalized distress, insight-oriented psychotherapy involves developing the patient's ability to constructively react to stressful life events. Thus, a major therapeutic goal is to help the patient identify and then discharge his feelings of frustration, anger, and aggression, and thereby relieve the need for aggressive responses. The sleepwalker can be taught to think of a sleepwalking episode as a cue or indication of an important, current life frustration with which he is not dealing appropriately. He might say, "I sleepwalked last night. What is it that is so frustrating as to provoke an episode, but is also so troublesome and distressing that I do not want to cope with it?"

Drug Treatment

The physician needs to be very cautious in prescribing psychotropic medication for children who sleepwalk. It should be used only when absolutely necessary and for a very limited period, since the long-term effects of psychotropic drugs on children's' development are not known. The use of stage 4 suppressant drugs in treating adult somnambulism is being investigated. Certain drugs, primarily benzodiazepines such as diazepam and flurazepam, markedly suppress stages 3 and 4 sleep. However, these drugs have not yet been shown to result in a clear-cut decrease in the frequency of sleepwalking episodes.

The effectiveness of imipramine in reducing the frequency of sleepwalking episodes has been suggested but has not been demonstrated in controlled studies. The mechanism for this effect of the drug is not related to its psychotropic action in alleviating depression, since depression is not a primary problem in patients with sleepwalking. In addition, this effect of the drug is not mediated through any changes in stage 4 sleep, since imipramine does not significantly alter this sleep phase. It may be that imipramine's effectiveness in reducing sleepwalking or night-terror events is related to the fact that the drug has increased the arousal levels of patients with sleepwalking and night terrors. In this way, the drug would minimize the arousal disorder that would ordinarily occur out of stages 3 and 4 sleep.

Night Terrors

Night terrors are nocturnal episodes of extreme terror and panic that are associated with intense vocalization and motility and high levels of autonomic discharge. The episodes are of relatively short duration, lasting from one to several minutes. During a typical episode, the patient is confused and disoriented and has little or no recall of the event either immediately following it or the next morning.

While night terrors are not as frequent as nightmares, they are not rare. In one survey of approximately 1,000 children, the incidence of night terrors was reported to be 2.9 percent. In another survey of 1,000 children seen consecutively in a child psychiatry clinic, the incidence was 1.5 percent.

In most cases, night terrors can be easily differentiated from nightmares by their clinical characteristics. The nightmare is accompanied by much less anxiety, vocalization, motility, and autonomic discharge than the night terror. In addition, the sleeper is more easily aroused, and there is usually vivid and detailed dream recall. Most night terrors occur in the first few hours of sleep and arise out of stages 3 and 4 sleep. In contrast, nightmares are related to REM sleep, which is more predominant during the latter part of the night.

Causative Factors

Night terror events are often accompanied by sleepwalking activity. A patient may actually present with both conditions, or he may initially have somnambulism and later develop or switch to night terrors. The two disorders share many of the same clinical and psychological characteristics. In addition, patients with either disorder frequently have a family history for either sleepwalking or night terrors. Pedigrees studied appear to fit a two-threshold multifactorial model of inheritance, in which night terrors are the least prevalent condition. Other similarities are that both sleepwalking and night terrors start in childhood and are usually outgrown before adulthood, and in both cases, episodes occur out of slow-wave sleep and are typically associated with impaired arousal. Further, both disorders are usually due to a maturational/ developmental lag. However, when they persist or start in adulthood, psychological factors are more prominent. Thus, the two conditions have been considered as two different manifestations of the same pathophysiological substrate.

In addition to the etiological role that genetic and developmental factors may play, psychological factors also contribute considerably to the development and/or persistence of night terrors in adulthood. Night terrors are more likely to persist into adulthood when their age of onset is after age twelve, when the frequency of the episodes is high, and when there is a major life-stress event at the time of onset. The primary role of psychological factors is underscored by the high levels of psychopathology in adult night-terror

sufferers. Their MMPI profiles show an inhibition of outward expression of aggression, a predominance of anxiety, depression, and phobicness, and a secondary "schizoid" self-negativity in the absence of overt psychoticism. The inability to express aggression in night-terror patients may lead to the aggression being directed inward, thereby precipitating night terror events marked by extreme defensiveness and fighting behavior, which further frighten the patient.

Diagnostic Considerations and Procedures

Complete evaluation of the child or adult who appears to have night terrors begins with a thorough history. In addition to a carefully taken family history, important data to obtain include the age of onset, frequency of episodes, time of occurrence, behavior during the actual episode, length of the episode, degree of recall of mental activity, general daytime behavior and adjustment, and the relation of the onset of night terrors to any major life stress event. Since the patient is usually amnesic for the episodes, much of this information is obtained from the parents, or in the case of adults, from the spouse or roommate.

A thorough history is essential in evaluating the contribution of organic factors, psychopathology, or both, in the development and persistence of the condition. It is quite unusual for night terrors to begin during or after middle

age; if they do, underlying organic pathology such as a brain tumor is suggested. The possibility of organic pathology should therefore be carefully evaluated.

It may be necessary to differentiate night terrors from other disorders. They are easily differentiated from hysterical dissociative states, such as amnesia, fugue states, and multiple personality, since the individual in a dissociative state is more alert, more capable of complex behavior, and has longer episodes with much less violent or agitated activity. It may be necessary occasionally to distinguish the bizarre and explosive behavior of night-terror episodes from that associated with schizophrenia, which is characterized by disturbed daytime behavior.

Contrary to previous reports, there does not appear to be a relationship between night terrors and epilepsy. In differentiating night terrors from epileptic disorders, the latter are characterized by more repetitive and stereotyped behavior. Also, the duration of epileptic events is briefer, often lasting only a period of seconds. Night terrors may be confused with temporal lobe epilepsy, but it is rare for this condition to occur only at night. In a patient who is thought to have night terrors, a clinical sleep EEG should be obtained if there are similar daytime attacks or other episodic behavior.

Therapy

Nonpharmacologic Management and Psychotherapy

In the management of the patient with night terrors, safety measures are often necessary. Such measures include sleeping on the ground floor and providing special bolts for windows and doors. The spouse, bed partner, or roommate should understand that forcibly interrupting a night-terror episode may aggravate the patient's violent behavior. As with sleepwalking, those groups and agencies that take responsibility for the safety of children with night terrors when they are away from home should give special consideration to the safety of these patients.

Since adult patients with night terrors are often psychologically disturbed, psychotherapy is usually indicated. Night-terror patients commonly inhibit expression of their feelings of aggression and anger and may as a result direct these feelings inward. Such internalization may precipitate night-terror events that consist of extreme defensiveness and fighting behavior that further frightens the patient. Thus, while the sleepwalker is conditioned to respond to stress outwardly both while awake and during sleep, the night-terror sufferer is conditioned to react with fear and apprehension. As a consequence, insight-oriented psychotherapy, by actively exploring fears of failure and hostility as well as of the night-terror event itself, can be beneficial. It is also helpful to strengthen the patient's self-assertiveness. This helps to counteract fears of hostility as well as anxieties

over how others would react if the patient were to experience failures in life.

Assuming that night terrors are a type of discharge of accumulated anxiety and fear, the patient should appreciate the importance of dealing with the anxiety, however distressing, in psychotherapy rather than discharging it during sleep. If the fear involves a specific phobia, then behavior therapy using desensitization or implosive techniques may be useful.

Drug Treatment

Benzodiazepine drugs (diazepam) that suppress slow-wave sleep are effective in reducing the frequency of night-terror events, but drug withdrawal usually leads to a relapse. It is not clear whether diazepam's effectiveness in reducing night-terror events is related to its suppression of stage 4 sleep or to its general anxiolytic properties.

As previously discussed, effectiveness of imipramine has been suggested for reducing the frequency of sleepwalking and night terrors, but this effectiveness has not been demonstrated in controlled studies.

Psychotropic drugs should be used only when a reduction in the frequency of night terrors is absolutely necessary; that is, when special consideration is needed for the safety of the night-terror patient. The use of such drugs in children, however, should be discouraged.

Nightmares

Nightmares occur frequently in childhood, particularly in children who are between the ages of three and eight. Nightmares are also fairly common in adults. In the general adult population of Los Angeles, 11.2 percent of those interviewed indicated that they had difficulty with nightmares at some time in their lives; 5.3 percent reported a current complaint of nightmares and 5.9 percent had only a past complaint. In other studies, frequent nightmares (at least one per week) were reported as 5 percent in college undergraduates and 7 percent in a psychiatric population.

Nightmares are nocturnal episodes of intense anxiety and fear associated with a vivid and emotionally charged dream experience. Nightmares are often accompanied by some vocalization and increased autonomic activity. They can be a serious cause of sleep disruption, since the events are most often followed by lengthy periods of arousal. Typically, the individual has detailed recall of the nightmare. The patient usually awakens easily, often spontaneously, from a nightmare, is lucid following arousal, and usually becomes quickly oriented to the environment, although it may take some time for him to separate nightmare dream content from reality.

In most cases, nightmares, which occur in REM sleep, can be easily differentiated from night terrors, which occur in slow-wave sleep, because of their general clinical characteristics. The nightmare is accompanied by much

less anxiety, vocalization, motility, and autonomic discharge, and there is much greater recall of the event than in the night terror. Also, night terrors usually occur during the first two to three hours of sleep, whereas nightmares may occur at any time of the night.

Causative Factors

Childhood nightmares are most often related to specific phases of growth, being most common during the preschool and early school years; they are related both to ego development and psychological conflicts. Because the child has an active fantasy life during this period of development, fears of imaginary figures and misperceptions of shadows and objects frequently trouble him while he is preparing to go to sleep. Consequently, the child often develops a number of vague, imaginary fears or phobias that may be frightening during the night, such as a fear of death, of the dark, of being alone, or of monsters and ghosts.

Nightmares are also frequently reported during febrile illness, especially in children. Since febrile conditions appear to suppress REM sleep, nightmares may be more likely to occur after the fever subsides, when there is a REM rebound consisting of longer REM periods. The confused and semi-delirious state that often accompanies febrile illness could contribute to the child's inability to distinguish between dream material, fantasy, and reality,

and thus compound his anxiety and fear.

The onset of nightmares after adolescence and their persistence into adulthood appear to be correlated with the presence of significant psychopathology. MMPI data show that adults with chronic nightmares have considerable psychopathology and specific personality characteristics. They are typically distrustful, alienated, estranged, oversensitive, overreacting, and egocentric. Their distrustfulness is not paranoid, however, since it is diffuse and generalized rather than directed to a specific object. Chronic sufferers may present with an underlying chronic schizoid pattern of adjustment, although in general they are not overtly psychotic.

Chronic nightmares can be secondary to long-term difficulties in dealing with interpersonal hostility and resentment. When the excessive hostility resulting from intensely neurotic object relations is not entirely discharged in everyday life, it may be carried over and released in the nightmare. The nightmare serves not only as a vehicle for discharging unreleased hostility, but also for the extinction of unfinished anger and generally negative emotions. When these emotions are finally expressed in the nightmare, they become anxiety-provoking.

The physiological basis for the occurrence of nightmares is not entirely known. It is well documented, however, that nightmares occur out of stage

REM sleep and that an excess of REM sleep, as in REM rebound following drug withdrawal, is associated with a temporarily increased incidence of intense dreaming and nightmares.

Diagnostic Considerations and Procedures

In evaluating the patient complaining of chronic nightmares, the physician needs to determine the age at onset, frequency of episodes, time of occurrence, behavior associated with the episode, length of the episode, degree of recall of mental activity, general daytime behavior and adjustment, and relation of the onset or recurrence of nightmares to major life-stress events. The age of the patient is important in deciding whether the nightmares are related to a phase of development or whether they are due to psychological conflicts. Underlying psychopathology is suggested if the nightmares begin after about ten years of age, if they persist for several months without decreasing in frequency, if daytime symptoms suggest a functional disorder, or if there is a correlation between the onset or recurrence of nightmares with major life-stress events.

It is also helpful to obtain a general sleep history from the patient, since individuals with frequent nightmares tend to have a higher incidence of other sleep disturbances, particularly insomnia.'' Similarly, patients with a major complaint of insomnia frequently report difficulty with nightmares dating

back to childhood.

Since many drugs, including alcohol, induce marked changes in both the frequency and intensity of dreaming, a complete drug history is essential. Drug-withdrawal nightmares secondary to increases in REM sleep occur when REM-suppressant drugs are abruptly withdrawn, but they can also occur on the same night that the drug is given. Nightmares during nights when drugs are taken are probably rare but may occur when short-acting hypnotics are used, since REM sleep increases late in the night as the action of the drug diminishes. Nightmares may also occur on drug nights when an individual sleeps past the duration of action of the drug (nine to ten hours), as on a weekend.

Although most drug-induced nightmares are associated with REM rebound following the withdrawal of REM suppressant drugs, the actual administration of certain drugs may increase both the intensity and frequency of dreaming and REM sleep. For example, reserpine, Thioridazine, and meso-Thioridazine all produce increases in REM sleep and may intensify dreaming.

Therapy

Nonpharmacological Management and Psychotherapy

Education and reassurance are often necessary, since both the patient

and his family not infrequently consider nightmares as a psychotic symptom. Parents of children with nightmares should know that children frequently experience nightmares as part of their normal development and usually "outgrow" the disorder within a relatively short time. Preventive measures are also very helpful. Parents should know that terrifying experiences such as watching violent television programs or listening to frightening bedtime stories may be harmful. Other than eliminating these specific types of potentially traumatic experiences, however, parents should allow the child a normal range of activities and experiences.

If there is considerable psychopathology, as is commonly the case in adults who have nightmares, the nature of the intrapsychic conflicts is explored in psychodynamic psychotherapy. For example, the physician may determine that early life experiences are causing concern and a preoccupation with death. Mono-symptomatic psychiatric conditions, such as chronic fears or phobias, may be responsible for nightmares in a limited number of cases. Most often, however, a more global psychological disturbance underlies the presence of nightmares in adulthood.

In the few instances in which nightmares appear to be an isolated problem rather than symptomatic of a basic psychiatric disturbance, behavioral therapies, such as systematic desensitization, have been successful.' Nevertheless, since the underlying psychiatric condition is seldom

mono-symptomatic, behavioral treatment has only limited application; insight-oriented psychotherapies are more likely to be effective in most cases.

The psychiatrist should be aware that the nightmare sufferer often feels distrustful and alienated, and that he may consequently suddenly terminate treatment. Thus, a major consideration at the beginning of therapy is to develop a sound therapeutic relationship. A subsequent therapeutic task is to have the patient achieve a better understanding of his emotions, particularly his anger. This enables him to more efficiently cope with his emotions, rather than become excessively frustrated and, as a consequence, discharge incompletely expressed resentments in his nightmares. Finally, the psychiatrist should be alert to the presence of depression, especially in men who have nightmares, since the nightmare patient tends to avoid treatment and suicidal potential may go unnoticed for lack of follow-up.

Drug Treatment

The physician should be aware of the schizoid adjustment in the chronic nightmare sufferer's life style, but he should also understand that the patient's coping mechanisms are neurotic in nature. When overt psychotic behavior is associated with the presence of nightmares, however, the physician should proceed with antipsychotic treatment. Antidepressant medication should be used whenever depression is identified. Administration of REM-suppressant

drugs to decrease the nightmare frequency should be avoided, since their withdrawal may lead to a REM rebound associated with an increase in dream intensity and possibly nightmares.

Enuresis

Attainment of complete bladder control depends upon sociocultural and developmental factors, personality characteristics of the child, general emotional climate in the home, and parental attitudes toward toilet training. Enuresis is the term most often used to refer to bedwetting after control of the urinary bladder should have been acquired. Most children begin to develop urinary control by eighteen months of age and achieve dryness between ages two and three; some do not attain dryness until four to six years of age.

There are two types of enuresis: primary and secondary. In primary enuresis, the child has never been dry for more than one or two weeks; in secondary enuresis, he may be dry for several weeks, months, or years before enuresis begins. Primary enuresis is also called functional or persistent enuresis, and secondary enuresis may be referred to as acquired, regressed, or onset enuresis.

After age three, about 10 to 15 percent of all children are enuretic; at age twelve about 3 percent are enuretic. Studies of military recruits indicate

that in young adulthood, the prevalence of enuresis may be as high as 1 to 3 percent.

Causative Factors

Enuresis appears to be related to time of night, not to sleep stages. About two-thirds of all enuretic episodes occur in the first third of the night, and most take place during NREM sleep.' However, the enuretic episodes are distributed across the various sleep stages in proportion to their rate of occurrence during each third of the night. Thus, since NREM sleep predominates in the first third of the night, it is more likely for the episodes to occur in NREM sleep. If the child is not awakened and changed after wetting the bed, the sensation of wetness may be incorporated into a dream during the next period of REM sleep and result in dreams of wetting or being wet. This phenomenon accounts for the common misconception that enuresis occurs while the child is dreaming.

In primary enuresis, genetic and developmental/maturational factors underlie the disorder, while in secondary enuresis, psychological factors are the most important. From a developmental standpoint, children with primary enuresis have a smaller-than-normal functional bladder capacity. Secondary enuresis is most often psychogenic. Psychological factors related to secondary enuresis may reflect a need to regress or a need for excessive attention, for

example, when a new sibling is born. It should also be noted that bedwetting may be a symptom of diabetes mellitus, diabetes insipidus, nocturnal epilepsy, severe mental retardation, neurologic disorders, or other systemic conditions.

Diagnostic Considerations and Procedures

Since there are multiple causes and contributing factors in enuresis, it is critical for the physician to thoroughly evaluate and treat each patient individually rather than follow any single, stereotyped approach to management. The evaluation of the enuretic patient includes a thorough history, with specific attention paid to the history and course of bedwetting. A physical examination and urinalysis are also important.

The physician first determines whether the enuresis is primary or secondary; if secondary, he then determines the age of onset and its relation to major life-stress events. Other factors to consider are: the family history for enuresis, the child's general sleep habits, the presence of other sleep disorders, the attitude of the child and parents toward enuresis, and the degree of psychopathology in the child and family.

The physician should be careful not to misinterpret any psychopathology as the cause rather than the effect of enuresis. This is particularly true for children with primary enuresis who are commonly mishandled by their parents, sometimes with devastating psychological

consequences. On the other hand, in children with secondary enuresis, the physician needs to distinguish between psychological difficulties that cause bedwetting and the psychological problems the child may develop because of his enuresis.

When conducting the physical examination, the physician directs special attention to the child's general growth and development. The urinary stream is observed to see if it is full and forceful or if there is deviation, narrowing, or dribbling. In the urinalysis, a urinary specific gravity of 1.024 ^" eludes renal disease. A culture of the urine should be made when an infection is suspected. The baseline functional bladder capacity is also determined by having the child refrain from voiding as long as possible and then measuring the volume of the urine voided.

Therapy

Nonpharmacologic Management and Psychotherapy

The type of treatment to be used for enuresis depends mostly upon whether the disorder is primary or secondary. Education and reassurance for the parents and the child are an important aspect of treating both types of enuresis. Parents of children with primary enuresis should be counseled to be tolerant, patient, and understanding of their child's disorder. They should be aware that overreacting with harsh and punitive behavior may cause their

child to feel guilty, angry, and anxious, and that their mishandling of the situation may result in serious psychological complications.

Because children with primary enuresis often have a small functional bladder capacity, bladder-training exercises are often used to expand the bladder's capacity. These exercises consist of three major elements: (1) drinking fluids in unrestricted amounts during the daytime, (2) voluntarily withholding urination as long as possible at least once a day, and (3) recording the daily amount of urine passed after maximum holding. These exercises give the child a feeling of mastery and, by stretching the bladder, increase its capacity.

The treatment of primary enuresis in adolescents and adults starts with bladder-training exercises that help achieve initial mastery of bladder functioning. Subsequently, insight-oriented psychotherapy may help the patient to attain permanent dryness.

Since psychological difficulties usually underlie secondary enuresis, various psychotherapies are often employed in its treatment. The proper assessment of the psychological basis of the problem determines the therapeutic approach. Family therapy, seeing the parents and child separately in psychodynamic treatment, behavioral therapy, or simply educating and instructing the parents, are all helpful if appropriately applied by the

psychiatrist. When treating adults with secondary enuresis that is unrelated to any organic factors, insight-oriented psychotherapy is most effective.

Drug Therapy

Imipramine has been proven effective in reducing enuretic frequency; however, the relapse rate is high after the drug is withdrawn. Use of the drug is recommended only in older children or adolescents, and then for only limited periods of time and special situations.

The efficacy of imipramine in treating enuresis apparently is not related to its antidepressant effect since there is no evidence to suggest that enuretic children are more depressed than non-enuretic children. Also, sleep laboratory studies have shown that the reduction in enuretic frequency with imipramine is not related to any sleep stage alterations produced by the drug. It was found that imipramine produces a significant increase in wakefulness during the night. The hypothesis is that these heightened levels of arousal during the child's sleep allow a more conscious control of micturition. This factor, together with a decrease in bladder excitability, results in greater bladder capacity and fewer enuretic events, particularly early in the night when enuresis is most frequent. This allows the child to continue to sleep without micturition, and later in the night, when sleep is lighter, to be more aware of stimuli from a full bladder. Children who are being treated with

imipramine, as well as their parents, should be advised of this effect of the drug. In this way, the child is encouraged to get up and urinate when he experiences wakefulness during the latter part of the night, rather than remain in bed.

Imipramine should not be prescribed on a long-term basis since the effects of long-term administration of psychotropic medication in children have not been determined. Treatment with imipramine is initiated with a dose of 25 mg one or two hours before bedtime, and this dose is then gradually increased to a therapeutic level. The FDA-approved dose for treating enuresis is 1 to 2.5 mg per kilogram per day, or 25 to 75 mg daily.

Conclusion

Sleep disorders are frequently encountered in medical practice. Although the general physician is capable of diagnosing and treating most cases of sleep disturbance, patients who have disorders that are chronic or that present special difficulties are usually referred to specialists, most often psychiatrists and, less frequently, neurologists. Psychiatrists will most often be presented with cases of intractable insomnia, sleepwalking, night terrors, nightmares, and secondary enuresis, while neurologists are more likely to see patients with narcolepsy, the hypersomnias, and occasionally night terrors.

The clinical indications for sleep-laboratory diagnostic studies are

limited. Insomnia, enuresis, sleepwalking, night terrors, and nightmares rarely require sleep laboratory evaluations because the diagnosis nearly always can be established by taking a thorough history. In most cases, the psychiatrist's skills are far more useful in diagnosing sleep disorders than are time-consuming and expensive sleep-laboratory procedures.

There are certain specific and limited situations in which nocturnal electroencephalographic recordings or other sleep laboratory procedures can aid the psychiatrist in formulating a diagnosis. In narcoleptics who do not have cataplexy, diagnostic nap recordings to detect sleep-onset REM periods may be helpful; such studies can be performed in a clinical EEG laboratory. If sleep apnea is present or suspected, sleep-laboratory diagnostic studies are indicated to quantify the number and duration of episodes, changes in blood gases, and the degree of sleep disturbance. All-night sleep recordings may also be useful in diagnosing nocturnal epilepsy when daytime clinical sleep EEG recordings are not sufficient. Finally, psychogenic impotence can be differentiated from organic sexual difficulty by recording the occurrence of penile erections in relation to REM periods.

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