The Sleep Disorders

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Introduction

Between 12% and 15% of all people living in industrialized countries have serious sleep problems. This 12% to 15% figure excludes the 20% to 25% of the people who complain of occasional bouts of insomnia (Karacan et al, 1976). In a careful survey of a Florida county, more than 35% of the population reported having trouble sleeping at least "sometimes" and 13% said they had sleep problems either "often" or "all the time" (Karacan et al, 1976). Similar estimates have been obtained in other studies from this country, from Britain, and from Australia (Clift, 1975). Furthermore, problems with insomnia are not the only sleep disturbances of concern. Excessive daytime sleepiness is a serious problem for well over 100,000 Americans (Guilleminault et al, 1974), and problems during sleep, such as nightmares and enuresis, take a further toll.

The purpose of this review is to help clinicians effectively diagnose and treat patients who complain of sleep problems. Secondarily, it is hoped that criteria for referring patients to sleep disorders centers will be established. To achieve this goal, the review is divided into three parts: *1*) an introduction to the basic facts of sleep; *2*) a review of the factors that influence sleep; *3*) a discussion of the diagnosis and treatment of the various sleep disorders.

The Sleep Laboratory

Most knowledge concerning sleep in humans has been gathered in sleep laboratories, which usually function as follows. The subject, either a normal volunteer or a patient suffering from sleep disorders, comes to the lab one or two hours before his usual bedtime. After the subject fills out a questionnaire concerning his daytime activity and his current moods, electrodes and sensors are applied. At least eight electrodes are needed to determine sleep stages (Rechtschaffen and Kales, 1968): two central-scalp electrodes (C_3 and C_4) to record an electroencephalogram (EEG), two at the outer canthi of the eyes to record eye movements (the electro-oculogram, or EOG), two chin electrodes to record a mentalis electromyogram (EMG), and two reference electrodes on the earlobes (A_1 and A_2). However, for a *clinical* assessment of sleep disorders, many more electrodes are usually required. For the evaluation of insomnia, for example, one usually needs sensors that measure respiratory rate and breath-by-breath air flow, an oxymeter, EKG leads, and surface EMG leads over the right and left anterior tibialis muscles. On the other hand, a full clinical EEG is rarely obtained in sleep studies.

After electrode application, the subject retires to a relatively quiet and comfortable bedroom, which is separate from the equipment room but connected to it electronically and by an intercom system. A technician monitors the polysomnogram: the continuous recording of EEG, muscle tension, eye movements, and other activities being observed. Even though sleep laboratories traditionally record at only 10 to 15 mm/sec, an entire night of polysomnography generates from 1,000 to 1,500 feet of paper.

Although electronic techniques can be used to reduce the data, most sleep-stage analyses are still done by visual inspection of the polysomnogram. The classification of sleep stages depends on multichannel patterns and relationships rather than on individual EEG frequencies, and electronic techniques are still less reliable and more expensive than is the trained eye for this complex analysis.

Basic Facts on Sleep

Sleep Architecture

Less than 50 years ago, sleep was thought to be a relatively simple, uniform, and homogenous state. The concept changed in 1935, when Loomis et al described separate and distinct EEG stages within sleep. The next significant advances occurred in the mid-1950s, when Aserinsky and Kleitman (1953) reported that bursts of conjugate, rapid eye movements (REM) periodically appear during sleep and when Dement and Kleitman (1955) linked these periods of REM to dreaming. So, sleep can be looked upon as a complex and multivariable state not unlike a building with various components and structural laws relating one part to another. Sleep architecture, then, describes the components of sleep (stages, cycles) and their interrelationships.

Sleep Stages: Basically, there are two very different kinds of sleep: Non-Rapid Eye Movement (NREM, pronounced Non-Rem) sleep, also called "orthodox" sleep or "the S-state," and Rapid Eye Movement (REM) sleep, also called "paradoxical" sleep or "the D-state." Within NREM sleep, three stages are usually recognized: stage 1, stage 2, and delta sleep.

Stage 1 is a transition phase between full wakefulness and clear sleep. On the polysomnogram it is identified by a relatively low voltage, mixed-frequency EEG with a prominence of activity in the three-to-seven cps range (Rechtschaffen and Kales, 1968). In normal sleepers, stage 1 tends to be relatively short, ranging from one half to seven minutes. Reactivity to outside stimuli is diminished. For example, college students with their eyes taped open no longer "see" (that is, remember or recall) pictures presented to them in this stage (Rechtschaffen and Foulkes, 1965).

Sleepers also change mentally during stage 1 as thoughts begin to drift. Thinking is no longer reality-oriented, and short dreams often develop (Foulkes and Vogel, 1965). Nevertheless, many people subjectively feel that they are awake during stage 1. Because stage 1 is similar to sleep in some respects (lack of reactivity to certain stimuli) and similar to wakefulness in others (relatively desynchronized EEG), it probably should be considered a transition phase rather than regarded as full sleep (Johnson, 1973).

Stage 2 is marked by the appearance of sleep spindles (ie, bursts of 12 to 14 cycles per second [cps] activity lasting one half to two seconds) and by K-complexes (well-delineated, slow-negative EEG deflections that are followed by a positive component). Stage 2 is the first bona fide sleep stage, and mentation during this stage usually consists of short, mundane, and fragmented thoughts (Foulkes, 1962).

Delta sleep is defined by an EEG record showing at least 20% waves with a frequency of one half to two cps or slower and amplitudes greater than 75 microvolts (μV) peak to peak (C_3A_2 or C_4A_1). In the past, delta sleep has been separated further into sleep stages 3 and 4 depending on the number of delta waves, but this differentiation has proven unnecessarily detailed in most cases.

REM sleep alternates with NREM sleep at about 90-minute intervals. The EEG pattern during REM resembles that of stage 1 sleep, except that sawtooth waves are often seen (Figure 1). The rapid eye movements (REM), which give this stage its name, are only one of the outstanding characteristics. Muscle tonus is extremely low (especially around the neck and chin), though many small twitches are seen during REM sleep. Heart rate and respiratory rate are relatively high and very variable. More than 80% of subjects awakened

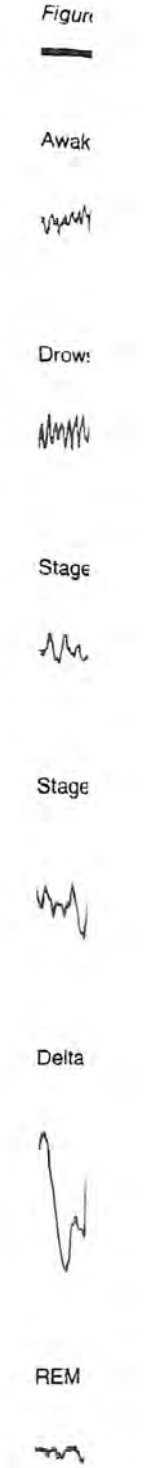
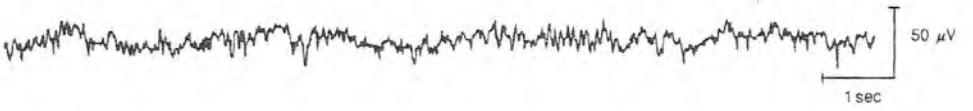


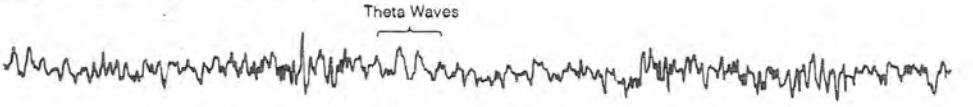
Figure 1. **Human Sleep Stages**

Awake – low voltage – random, fast

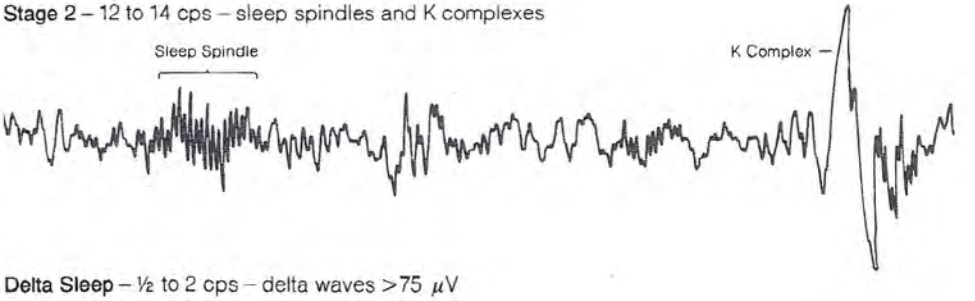


Drowsy – 8 to 12 cps – alpha waves

Stage 1 – 3 to 7 cps – theta waves

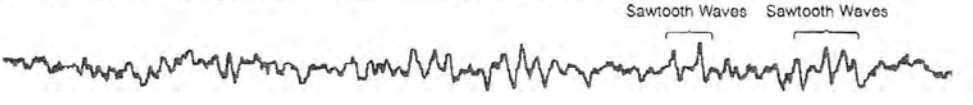


Stage 2 – 12 to 14 cps – sleep spindles and K complexes



Delta Sleep – ½ to 2 cps – delta waves >75 μV

REM Sleep – low voltage – random, fast with sawtooth waves



during REM sleep are able to recall dreams (Snyder, 1971).

Events during REM sleep are customarily divided into two classes: tonic events (such as low muscle tonus and desynchronized EEG) that form the stable base of this stage, and phasic events (such as eye movements and muscle twitches) that are superimposed in short bursts on this tonic background.

Sleep Cycles: Upon falling asleep, a subject goes through a stage of relaxed wakefulness characterized by alpha waves. Later, the sleeper passes through stage 1 into stage 2. Gradually descending deeper into sleep, most young adults enter delta sleep within 30 to 45 minutes after sleep onset. Depending on the subject's age, this sleep stage persists from a few minutes up to an hour, then it yields again to stage 2 sleep. About 70 to 90 minutes after sleep onset, the sleeper begins the first REM period of the night; this REM period usually lasts about five minutes. It is by far the least-intense REM period of the night, in terms of both the physiological manifestations of REM sleep and the psychological intensity of dreams (Figure 2).

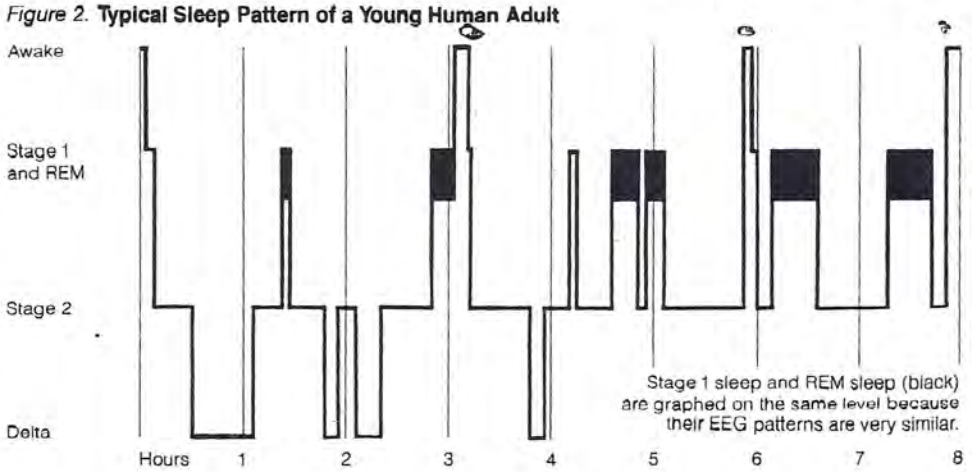
The second sleep cycle begins when stage 2 sleep recurs after the first REM period. On some occasions, delta sleep reappears, but there is usually less delta sleep in the second cycle than in the first. Following stage 2, the second REM period of the night occurs about three hours after falling asleep. This second REM period lasts about ten minutes and is more intense (both physiologically and psychologically) than the first.

Following the second REM period and until awakening in the morning, stage 2 sleep and REM sleep alternate in about 90-minute cycles. Delta sleep is rarely seen in these later sleep cycles. REM periods become more intense (both physiologically and psychologically) and longer towards morning. The mean length of a REM period is about 15 minutes, but REM periods frequently last up to one hour.

Although the separation of sleep into mutually exclusive, separate stages is convenient for data reduction, sleep stages actually merge into one another. Delta waves, for example, gradually become more abundant and gain amplitude following the onset of sleep. There is no clear threshold where one suddenly enters delta sleep (except by arbitrary definition). Similarly, the indices of REM sleep are strongest in the middle of the REM period, whereas the transition point between stage 2 sleep and REM is often quite difficult to define.

Sleep is frequently interrupted by body movements and 8 to 15 short awakenings, even in those who sleep

Figure 2. **Typical Sleep Pattern of a Young Human Adult**



well. There seems to be an optimal number of body movements (around one every 15 to 20 minutes), and Othmer (1965) feels that too few body movements might be as detrimental to good sleep as too many.

Depth of Sleep: Of the NREM stages, delta sleep is deepest and stage 1 is lightest (if it is sleep at all). However, REM sleep cannot be that easily classified on a scale of sleep depth. By measuring how much noise it takes to wake a person from REM sleep, it appears that REM in humans is about as deep as stage 2 sleep (Rechtschaffen, Hauri, and Zeitlin, 1966). In the cat, however, REM is the "deepest" sleep of the night, ie, it takes more noise to awaken a cat from REM sleep than from delta sleep. Similarly, while such things as heart rate variability and neural discharges in some parts of the brain suggest that REM sleep might be as energy consuming as alert wakefulness, a lack of reactivity in skin resistance, an exceedingly low muscle tonus, and many other measures suggest that REM sleep might be extremely deep sleep.

Because REM sleep cannot be classified as either light or deep, some researchers now believe that there are three separate stages of existence: wakefulness, sleep, and the REM state. The REM state behaviorally is similar to normal sleep (one lies in bed with eyes closed and diminished responsivity to the environment); however, it is much more similar to wakefulness in other respects (desynchronized EEG, arousal in many physiological systems). In short, REM appears to be a state as different from NREM sleep as it is from wakefulness.

Circadian Rhythm

"Circa" is Latin for "about," and "dian" means "day." Circadian rhythms are those fluctuations that take about 24 hours to complete.

The Healthy Rhythm: Normal body temperature is said to be about 36.8 C, but regular and consistent 24-hour fluctuations occur around this mean. Temperatures are low during sleep, rise in the morning, and reach a high sometime during a person's period of peak efficiency. Many other bodily functions, especially endocrine secretions and metabolism, follow similar circadian rhythms (Weitzman et al, 1968; Weitzman et al, 1976).

When human beings are removed from all indications of time, ie, being placed in an underground cave with artificial light, plenty of food, but no clock, they still maintain a circadian rhythm. However, under such constant conditions the circadian rhythm rarely lasts exactly 24 hours. Usually humans fall into rhythms between 24 and 28 hours, though sleep-wake cycles of up to 50 hours have been observed.

In order to entrain (synchronize) the circadian rhythm to the exact 24 hours imposed on us by the sun, "Zeitgebers" (ie, indicators of time) are necessary. Such Zeitgebers may be clocks, time of wakening by alarm, time of regular meals or work times, or position of the sun (Aschoff et al, 1975). Some data even suggest that naturally occurring, extremely low-frequency electromagnetic fields (at 10 cps) might be Zeitgebers (Wever, 1974).

Excessively Long Circadian Rhythms:

Case History: *Miss R., a 26-year-old, single journalist, sought help at the clinic for sleep-onset insomnia. She explained that she would often lie in bed four to six hours before falling asleep each night, and then would have extreme difficulties getting up the next morning (she had recently been fired from her job because she overslept so regularly). However, except for a certain defensiveness concerning her inability to get up on time, Miss R. seemed to be in reasonably good mental health.*

Miss R. was asked to keep a sleep log at home for two weeks. This log revealed that now, while she was unemployed, her sleep-onset insomnia had totally disappeared. Instead, each night Miss R. now went to bed four to six hours later than she had the previous day, causing her to stay up all night and sleep during the day on certain occasions. At this point, Miss R. kept

herself occupied by doing research for a book she planned to write. She was counseled to continue this activity, irrespective of the time of day or night, to go to bed only when she felt sleepy, and to sleep as long as she wished. A subsequent sleep log filled out for more than a month was astounding: Miss R. felt healthy and alert, usually working 20 to 22 consecutive hours, then relaxing for one or two hours before sleeping uninterruptedly for 10 to 12 hours (Figure 3). Realizing how well she felt under this new regimen, Miss R. decided to become a freelance writer and to continue "free running" on a 36-hour to 38-hour day. She has been on this schedule for over six months now, without ill effects.

Although most of us show circadian rhythms somewhat longer than a normal 24-hour day in a timeless environment, we are apparently able to synchronize with the sun quite easily. However, the possibility exists that some people (such as Miss R.) might be unable to compress their circadian rhythms into the usual 24-hour day.

Manipulation of Circadian Rhythms: The *position* of the peaks and troughs of the circadian rhythm can apparently be adjusted within the 24-hour day. This is often done passively by flying across a number of time zones. After a few days of discomfort ("jet lag") when our biological circadian rhythm is out of synchrony with the local time, the body adjusts to its new environment but is then out of synchrony with home (McFarland, 1975).

The position of peaks and troughs within the 24-hour clock time can apparently be manipulated by an act of will, as is done in shift work. A worker who consistently stays up all night and goes to bed at 8 AM learns to sleep until, say, 4 PM. Under these conditions, body temperature will be low at noon and high at midnight, and other circadian events will also adapt to this new time pattern. However, adjustment to the newly imposed sleep-wake rhythm may take a week or more. The problem with shift work is that on the days off, the worker tries to become a "day person" again. Thus, the Zeitgebers switch back and forth every few days, and a consistent circadian rhythm is never established.

Manipulating the sleep-wake rhythm is largely a behavioral issue. One can force only wakefulness; one cannot force sleep. If habitual oversleeping is a problem, going to bed earlier when not tired will only result in sleep-onset difficulties. It will probably not result in a phase shift. However, forcing persistent arousal at a specific time each morning will finally result in a phase shift, by causing tiredness earlier in the evening. Similarly, arousal too early in the morning cannot be corrected by trying to force oneself to sleep longer. Only by forcing oneself to remain awake later in the evening can one gradually accomplish a phase shift, over a period of weeks, and sleep longer in the morning.

Lack of Zeitgeber:

Case History: *Mr. M., a 25-year-old mechanic, had been hit by a train and was in a coma for three months. He then made a dramatic recovery, and seemed to be normal except for a slight aphasia. His lawyers, however, advised him not to work until the damage suit was settled.*

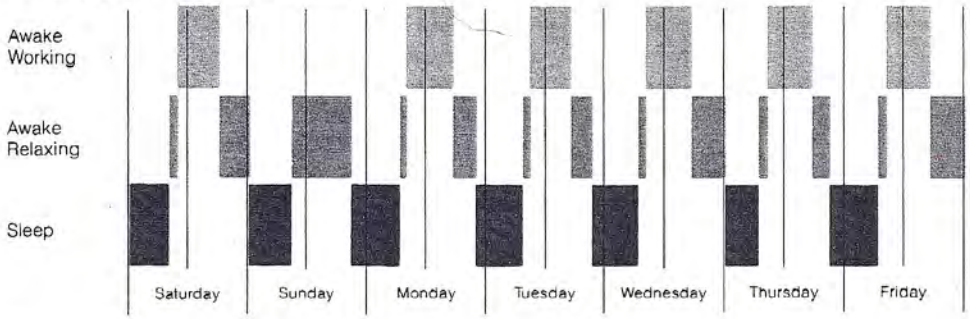
Mr. M. had always been a "loner," and he spent the next two years in a small apartment doing very little else except reading. Whenever he needed food, he would get it from a nearby 24-hour grocery store. Thus, while there were the usual Zeitgebers in his environment, Mr. M. ignored them. As a result, he soon found that he started to sleep poorly and always felt tired.

After two years on a "timeless" behavior schedule, Mr. M. was referred to the clinic as an insomniac. Twenty-four-hour recordings at the clinic revealed that he took frequent short naps throughout the day and night. To our surprise, body temperature showed no discernible circadian rhythm (Figure 4), but only wide and random fluctuations.

Mr. M. was urged to reestablish a regular day-night rhythm. When that proved impossible because he always felt tired, he hired a friend to live with him to keep him awake and active during the day, while gradually increasing his time awake between 7 AM and 10 PM. Regular mealtimes and a slow increase in exercise were also prescribed. Over a period of six very difficult months, Mr. M. reestablished a normal circadian cycle, and felt more fit during the day. After

Figure 3. **Excessively Long Sleep-Wake Rhythm**

Example of a normal sleep-wake rhythm



Free-running, sleep-wake rhythm of Miss R

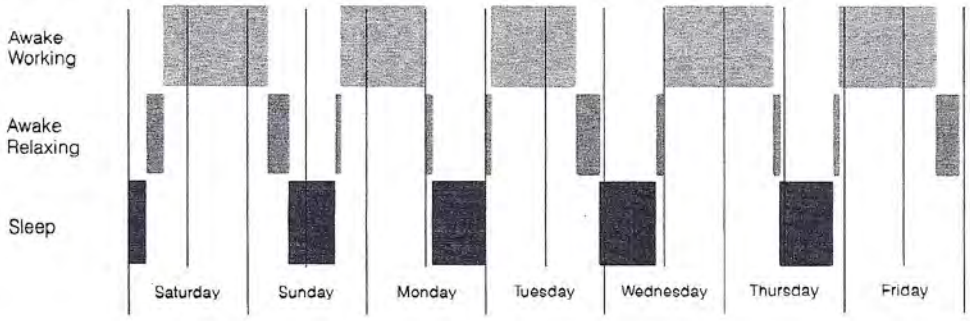
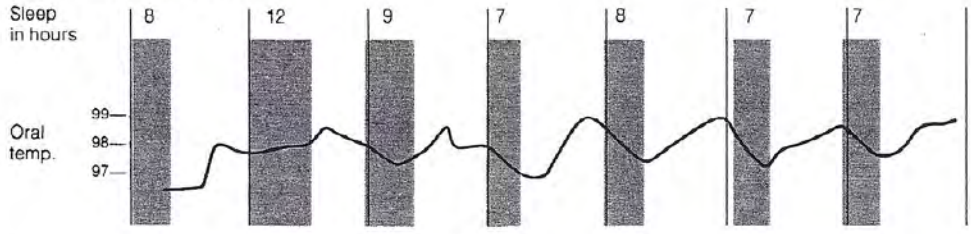
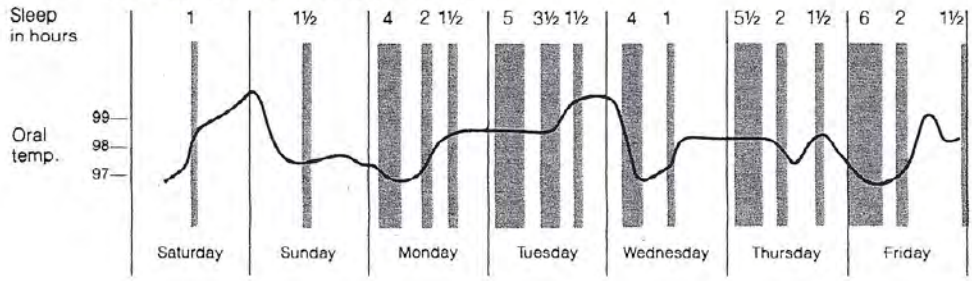


Figure 4. **"Regular" and "Irregular" Insomnia**

Regular sleep-onset insomniac



Insomniac (Mr M) lacking a consistent sleep-wake rhythm



the settlement of the lawsuit 1½ years later, Mr. M. bought a small farm in New England, which he is now managing quite effectively. The insomnia has totally disappeared.

Case History: *When Mrs. B. came to the sleep clinic, she was almost totally crippled by her chronic insomnia. She felt too weak to walk further than to the bathroom, usually went to bed around 8 PM, and then stayed in bed until 5 PM the following evening. What little sleep she got came in short episodes of one half to two hours irregularly spaced throughout the day and night. She arose only to keep her husband company during the dinner hours, and managed everything else (including a weekly women's group) while lying in bed.*

History revealed that Mrs. B. had always been a "fickle" sleeper but her main trouble started about 15 years ago when she had gone through a traumatic experience involving, within a period of six months, the death of her son, the loss of her husband's business, and her removal from her town's "high society." Sleeping poorly at night because of the trauma, she first started to escape her trouble by taking afternoon naps. Sleeping worse and worse at night, and feeling weaker and weaker, she extended her time in bed until she stayed there for 22 hours per day.

Careful medical evaluation showed no specific findings, except for muscular atrophy and weakness apparently secondary to prolonged bed rest. The depression of 15 years ago had long since lifted. However, both body temperature and endocrine function had lost the usual 24-hour cycling, both showing erratic, wild fluctuations. Mrs. B. was encouraged to increase the time out of bed very gradually and to become more involved, once more, in her husband's business and town affairs. Over a period of three years of constant counseling and support, she learned to stay up from about 8 AM to 10 PM and has regained her old position in civic affairs. Night sleep consolidated. However, Mrs. M. never became an excellent sleeper and, after very poor nights, she still takes a 1½-hour afternoon nap once or twice per week.

Both Mr. M. and Mrs. B. had lost a clear 24-hour pattern in their lives, each for a different reason. By the time the original reasons (hospitalization for Mr. M., depression for Mrs. B.) had disappeared, circadian cycling had given way to erratic fluctuations. It was then very difficult to reestablish a healthy sleep-wake rhythm and to resynchronize the various circadian cycles (Lund, 1974). Because the major Zeitgebers in our culture are social (work, meals, evening activities), scheduling problems such as the ones discussed in the above two case histories are seen mainly in people who are not under pressure to follow them rigidly, such as schizoid, withdrawn persons and those who are unemployed or self-employed.

Obviously, establishing circadian rhythms on the basis of oral temperature is scientifically weak. Measuring temperature rectally, or assessing metabolism, would be more convincing. However, for clinical purposes, oral temperature is much easier to obtain and often convincing enough. Graphing this temperature over a period of weeks gives the patient a powerful incentive to reestablish a regular sleep-wake rhythm.

The shape of the 24-hour oscillations seems partially related to personality variables. "Early birds" reach the peak of biological function and performance earlier than "night owls," who show very little increase in metabolism and performance for hours after awakening from sleep, but then reach their peak later during the day (Blake, 1965).

Neurological Basis of Sleep

The search for neurological regulators of sleep has a long and distinguished history. As early as 1890, Mauthner speculated that the area around the nucleus of the third cranial nerve might be related to sleep induction. His conclusion was based both on postmortem examination of patients who had suffered from a form of encephalitis with sleepiness and on theoretical speculations.

Reasoning from his now famous encéphale isolé and cerveau isolé experiments, Bremer (1935) concluded that the junction between the diencephalon and the brain stem must be crucial to sleep and wakefulness. Like Mauthner, Bremer also felt that sleep occurred "by default," that is, after afferent impulses

to the cortex fell to a level too low to maintain wakefulness. A modified version of this view gained widespread credence when Moruzzi and Magoun (1949) proposed the existence of the reticular activating system for maintaining arousal.

Powerful evidence contradicting the above idea that sleep was purely the absence of wakefulness was presented by Hess (1943, 1954). Hess felt that his experiments indicated the existence of an active sleep-inducing center, because he could actually "force" animals into sleep by his stimulations. Lesion experiments by Nauta (1946) supported this view. The arguments between "passive" sleep (based on a lack of stimulation) and "active" sleep (induced by activity in a sleep-promoting system) then raged for over two decades until it became clear, in the late 1960s, that both active and passive theories were partially right (as will be discussed below).

A separate breakthrough in understanding the neurophysiology of sleep came around 1960, when Jouvet and his co-workers (1959, 1962, 1965) located a REM-inducing system in the higher pons.

As understood today (Hobson, 1974), our wake-sleep-REM life depends on three neurological systems. These systems should not be thought of as specific nuclei,or centers, but rather as networks of mutually interacting and inhibitory areas or circuits.

Wakefulness seems to be maintained largely through activity in the ascending reticular-activating system. As long as activity in the system is high, there cannot be any sleep. Low activity levels in this system apparently lead to relaxed wakefulness (often indicated by alpha waves) and possibly to stage 1 (the transition phase between wakefulness and sleep).

Sleep (stage 2 and delta) requires the active involvement of a hypnagogic or sleep system *in addition to* low activity in the reticular-activating system. The location of this hypnagogic system is still debatable. It may be located either in the serotonergic nuclei of the pontine raphé system or in the medial forebrain area.

For the *REM* state to occur, the active involvement of the nucleus ceruleus (NC) and the gigantocellular tegmental field (GTF), both located in the higher pons, seems necessary (Figure 5).

Based on a series of brilliant experiments in cats, Hobson and co-workers (eg, Hobson et al, 1975) propose that cells in the gigantocellular tegmental field (GTF) produce electrical discharges spontaneously, unless such discharges are inhibited by impulses from the nucleus ceruleus (NC). As will be seen below, this inhibitory relationship between NC and GTF seems crucial for REM sleep. When GTF cells discharge, they set up spikes that travel into many different neurological systems. They are then often called pontine-geniculate-occipital spikes (PGO spikes), because they were first found to travel from the pons to the lateral geniculate nuclei, and from there to the occipital cortex.

The PGO spikes may be the origin and the prime movers for most phasic and some tonic phenomena during REM sleep. Such PGO spikes are found entering the oculomotor nuclei, possibly giving rise to the rapid eye movements of REM sleep. They appear in pathways to the middle-ear muscles, travel to the midbrain reticular formation (where they may be associated with the tonic EEG desynchronization typical of REM sleep), and they seem related to many aspects of the high variability found in the autonomic nervous system during REM sleep – for example, instability of the heart rate. They may even be involved in the psychological aspects of REM dreaming. Discharges coming from the lateral geniculate nuclei may be interpreted by the occipital cortex as visual pictures coming from the retina.

The *biochemical regulation* of human sleep probably involves numerous different neurotransmitter substances. Some tantalizing bits of evidence have

already emerged. In the cat, the neurotransmitter involved in the hypnagogic circuitry emanating from the pontine raphé is serotonin. Although things seem more complicated in humans (Wyatt, 1972), Hartmann et al (1974) have shown that as little as one gram of tryptophan, the precursor of serotonin, can reduce sleep latency.

The neurotransmitter substance that inhibits gigantocellular tegmental field activity seems to be aminergic (norepinephrine or serotonin, or both). This would explain why medications that increase aminergic activity at the relevant pontine sites usually decrease REM sleep. When such drugs are withdrawn abruptly, the neurochemical balance is usually disturbed in the opposite direction, and there is more REM sleep per night for up to four weeks after withdrawal from certain drugs. This REM sleep is often more intensive, both physiologically and psychologically (Oswald, 1969). The frightening dreams occasionally experienced after abrupt withdrawal from drugs that influence the aminergic system have been explained as being results of this REM rebound.

Physicians might do well to caution their patients about the possibility of such frightening dreams when withdrawing them abruptly from drugs that increase aminergic activity (eg, hypnotics, stimulants, antihistamines, antidepressants).

Most of the neurophysiological and neurochemical details remain to be worked out, but nobody questions the existence of three neurological systems governing wakefulness, sleep, and REM. From the interaction between the sleep and the wake systems, it follows that insomnia can occur either through excessive activity in the wake system (ie, the reticular-activating system) or through some inadequacy in the sleep circuitry. Although scientific data are lacking, it appears that the occasional insomnia most of us experience around stressful events relates almost exclusively to arousal in the reticular activating system. Chronic insomnia, however, especially when it has existed since early infancy, might not always be related to excessive arousal. Rather, one might suspect a direct weakness in the sleep-inducing circuitry or in its biochemical components, as will be discussed later.

In addition to the work on neurotransmitters, Pappenheimer (1976) and Monnier et al (1972) seem to have isolated two different complex chemicals in the CNS and blood that, when injected into recipients, apparently cause sleep. How these chemicals relate to the neurological mechanisms discussed above is unknown at present.

Age Relationships

Age seems to be the single most powerful determinant of a person's sleep pattern (Williams et al, 1974). Total time spent in bed drops from about 17 hours per 24 hours near birth to about 8½ hours at age 12 and to about 7½ hours between the ages of 25 and 45. From then on, total time spent in bed rises again, until it reaches a mean of about 8½ hours in old age.

The average time asleep per 24-hour period also drops from about 16 hours per day for neonates to about 8 hours at the age of 12 before leveling out at about 7 hours between the ages of 25 and 45. From then on, total sleep time continues to decline gradually, until it reaches about 6½ hours in old age (Figure 6). Comparing total sleep time with total time in bed, it is clear that sleep efficiency (time asleep/time in bed) remains relatively stable up to about 45 years of age, but then starts to drop. The older people get, the longer they stay in bed but the less sleep they obtain per 24-hour period. With advancing age, sleep also becomes more fragmented – more awakenings during the night and more difficulties returning to sleep once awakened.

The proportion of time spent in various sleep stages also changes quite dramatically over a lifetime. Delta sleep

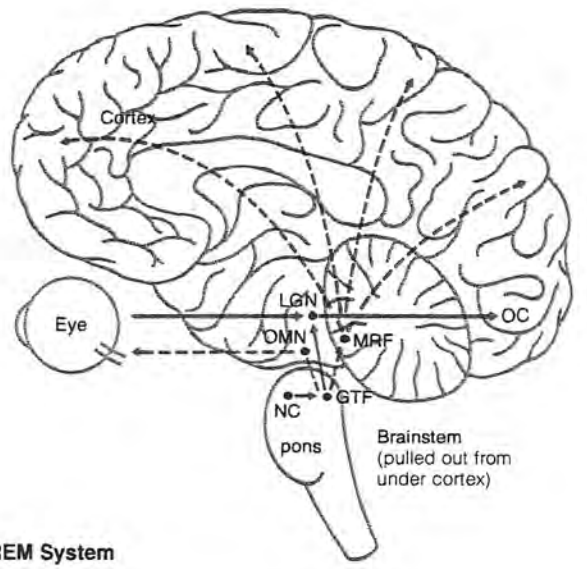


Figure 5. **Schematic Diagram of the REM System**

From the Nucleus Ceruleus (NC) to the Gigantocellular Tegmental Field (GTF)

a. Discharges from the NC usually inhibit discharges from the GTF during waking and NREM sleep.

b. The neurotransmitter involved in this inhibition appears to be aminergic.

c. This inhibition breaks down just before and during REM, liberating the GTF cells to discharge spikes.

From GTF via LGN to OC; The PGO Spike

a. Discharges from the gigantocellular tegmental field (GTF) enter the lateral geniculate nucleus (LGN), a halfway station on the visual pathway from the retina to the occipital cortex (OC).

b. Via the LGN, these discharges then enter the OC, where they are apparently interpreted as coming from the retina (seen as pictures in our dreams).

c. Because the pathway GTF–LGN–OC was the first one discovered, the discharges are called pontine-geniculate-occipital spikes, or PGO spikes.

Other discharges from GTF

The discharges from the GTF enter a multitude of other neuronal structures, apparently causing many of the typical phenomena related to REM sleep. Only two are outlined here:

a. Discharges enter the oculomotor nuclei (OMN) and cause eye movements.

b. Discharges enter the midbrain reticular formation (MRF) and cause general desynchronization of the cortex typical for REM sleep.

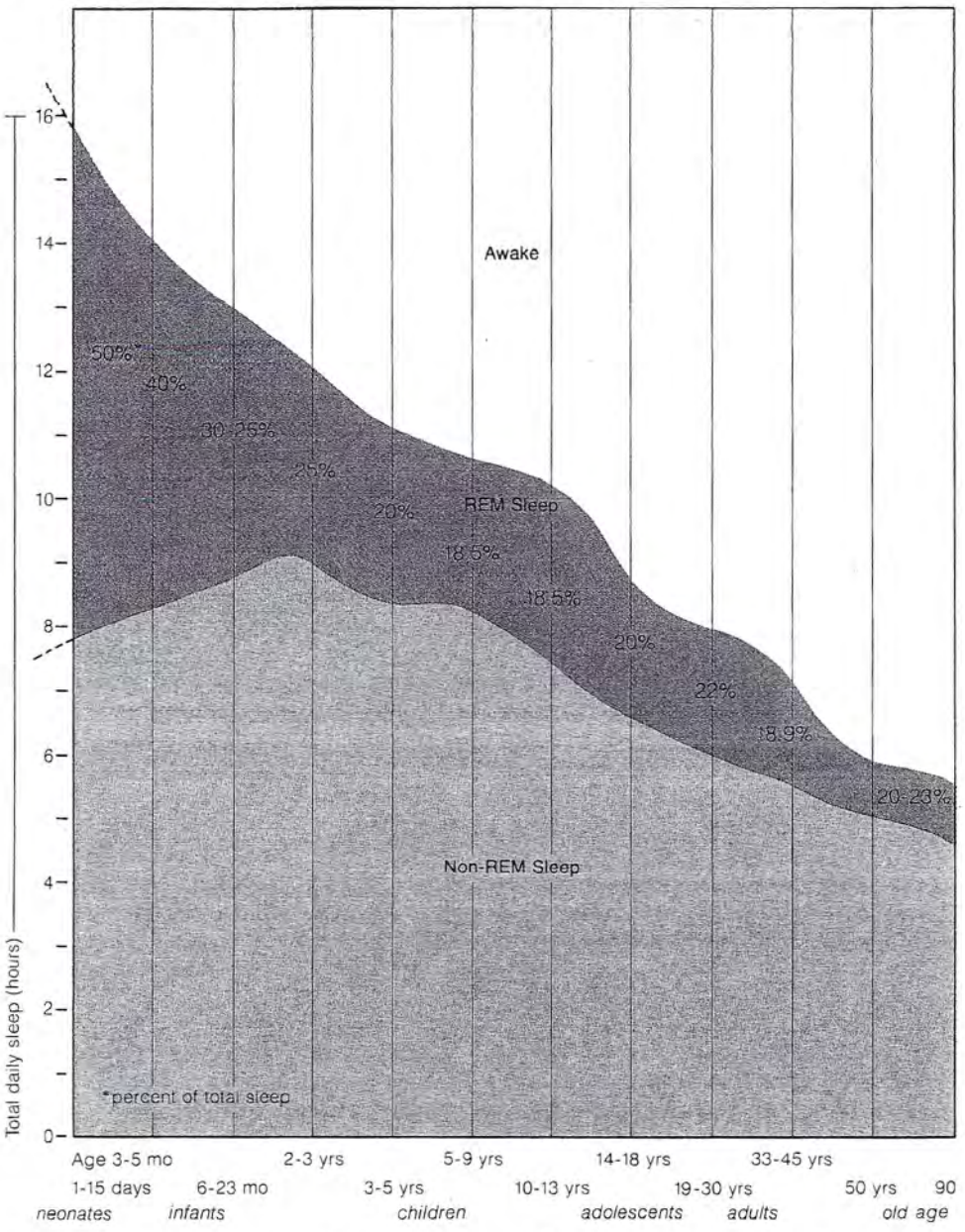
hovers around 15% to 20% up to the age of about 20 but then gradually decreases, until there is practically no delta sleep left by the age of 60. Conversely, stage 1 "sleep" increases from about 5% in childhood to about 15% of sleep in old age. Both of these changes imply that sleep becomes lighter and less efficient as one grows older.

REM sleep changes more dramatically than any other stage during childhood, dropping from about 50% at birth to about 20% by puberty. From that time on, REM sleep remains reasonably constant throughout life, although there seems to be a slight percentage increase during young adulthood and a slight percentage decrease, at least in males, during old age. The meaning of these percentage changes in REM sleep is unknown, but they have given rise to many speculations concerning our "need to dream" and the function of REM sleep.

Sleep Needs

Individual Requirements: The preceding section established the average levels of sleep requirements over a lifetime. However, not all people need the same amount of sleep, and the acceptable

Figure 6. **Mean Development of Human Sleep Over a Lifetime**



This graph shows changes (with age) in total amounts of daily sleep, daily REM sleep, and in percentage of REM sleep. Note sharp diminution of REM sleep in the early years. REM sleep falls from 8 hours at birth to less than 1 hour in old age. The amount of Non-REM sleep throughout life remains more constant, falling from 8 hours at birth to 5 hours in old age. In contrast to the steep decline of REM sleep, the quantity of Non-REM sleep is undiminished for many years. Although total daily REM sleep falls steadily during life, the percentage rises slightly in adolescence and early adulthood. This rise does not reflect an increase in amount; it is because REM sleep does not diminish as quickly as does total sleep.

(Reprinted by permission from Dr. H. Roffwarg, Montefiore Medical Center, NY, revised since publication in *Science 152*:604-619, 1966.)

range seems to be surprisingly wide. Some healthy adults sleep three hours or less per night without apparent ill effects, and a few rare ones have been recorded who habitually sleep one hour or less (Jones and Oswald, 1968; Meddis et al, 1973). At the other extreme, some healthy adults sleep 10 to 12 hours per night and feel sleep-deprived and tired when they can obtain "only" seven hours' sleep per night. This wide range in the sleep needs of apparently normal adults makes it necessary to define many sleep problems individually and subjectively. *A person has insomnia if his inability to sleep interferes chronically with efficient daytime function, regardless of how many hours he sleeps each night.* Thus, some insomniacs might sleep seven hours, whereas some normal sleepers might sleep only three. This definition is important, because some people complain bitterly about their insomnia when, in fact, they just have a low need for sleep.

Case History: *Mrs. C. was 71 years old when referred to the sleep clinic because she had never slept more than three hours per night throughout her adult life. She felt well during the day, although she speculated she might feel even better if she could obtain a "normal" amount of sleep. She was physically well, still cross-country skiing every day of the winter, and hiking frequently during the summer. An extensive medical workup, as well as neuropsychological testing, was totally unremarkable except for some slight deterioration commensurate with her age.*

Laboratory recordings confirmed an average of three hours' sleep per night. Surprisingly, Mrs. C. spent more than a full hour in delta sleep, ie, her sleep was much more sound than would have been expected for her age. Interviewing Mrs. C. in detail indicated that she felt unconcerned about her "insomnia" but had agreed to a sleep evaluation at the urging of her husband and her physician, both of whom had thought that she was an insomniac. She was reassured that she was a "healthy hyposomniac" rather than an insomniac and seemed pleased with this diagnosis.

It is not known why humans vary so widely in their sleep needs. Hartmann (1973) claims that long sleepers appear to be worriers, nonconformists, with a variety of psychological and social problems. In contrast, Hartmann's short sleepers, like Mrs. C., appeared to be "successful, outgoing, and healthy."

How much sleep does a "normal" individual need? Anyone can establish his or her own required quota by experimenting with different sleep lengths, providing each specific length is tried for at least a few weeks before trying another length. If any specific length does not permit adequate sleep, one will become excessively tired over the course of a week or two, ready to drop off to sleep when not stimulated. With too much sleep, one will find oneself lying in bed, developing symptoms of insomnia. This, however, assumes that the person is free from significant sleep pathology. As we will discuss later, some patients with excessive daytime sleepiness remain sleepy no matter how many hours they rest.

Extreme differences in the need for sleep characterizes children as well as adults. Under no circumstances should the average sleep times discussed in the section on age relationships be taken as absolute norms for individual children. Many two-year-old children need less sleep than ten-year-old children. The yardstick for adequate sleep in childhood is that the child remains alert and energetic over weeks and months. Should the child become excessively tired or doze off at school or play in response to sleep curtailment, he or she apparently needs more sleep. However, should even drastically increased amounts of sleep time not bring alertness and well-being after a few nights, the diseases of excessive daytime sleepiness (eg, sleep apnea, narcolepsy, to be discussed later) need to be investigated in such a child, as they would in the adult.

Manipulating Sleep Needs: Are we chronically sleep deprived? Webb and Agnew (1975) found that students now seem to sleep about 1½ hours less per 24-hour period than they did in 1910, but that many would be willing to sleep

longer than their customary time if given the opportunity. Webb and Agnew believe that sleep is under increasing pressure from modern living, jammed tightly between rigid work schedules, increased opportunities to socialize in the evening, television, and electric lights – and they suggest that most of us might do well to sleep longer.

While this view might apply to the busy, successful fellow who can never find enough time to "catch up on his sleep," sleep clinicians usually warn about the contrary problem – staying in bed longer than is necessary, which can lead to behavioral insomnia (see page 58). "Sunday night insomnia" is a common complaint. After having slept excessively long on Saturday and Sunday mornings, it apparently becomes difficult for many people to fall asleep at their regular bedtime on Sunday night. Perhaps keeping a little "pressure" in the sleep system would facilitate ease of falling asleep. Assuming that most humans have an inherent circadian rhythm somewhat longer than 24 hours, this would make good sense: getting up somewhat before the system is ready to get up might help to squeeze our inherently longer circadian cycle into the 24 hours required by the clock.

Can we decrease our need to sleep? The answer seems to be a qualified "yes" (Mullaney et al, 1976). Three couples who habitually slept eight hours per night and one who usually slept 6½ hours were asked to reduce their total sleep time by 30 minutes every two weeks until they reached six hours, and by 30 minutes every three weeks from then on. One couple terminated the experiment at 5½ hours, two stopped at 5 hours, and one at 4½ hours. They were then allowed to sleep *ad lib* again. At a one-year follow-up, six of the volunteers still slept 1 to 2½ hours less than they had before the beginning of the experiment. Although there were no visible signs of distress at the one-year follow-up, the long-range effects of curtailing sleep are unknown. We do not know, for example, the effect of sleep curtailment on psychological development, resistance to stress, or the cardiac system.

Total Sleep Deprivation

Effects: Can some people exist without *any* sleep? Many who claim to do this actually take short naps when studied in the laboratory. However, in certain very rare forms of organic brain disease (eg, Morvan's chorea), sleep seems totally abolished (Fischer-Perroudon et al, 1974), and the patients survive, at least for a few months.

Keeping healthy, young volunteers awake for an entire night makes them very sleepy but has remarkably little effect on their performance during the next day (Johnson, 1969; Wilkinson, 1969; Morgan, 1974). Initially, this was a very surprising finding because, after a sleepless night, mood deteriorated markedly and volunteers presumed that they were performing poorly. Except for extremely monotonous tasks, one can apparently "rally" one's reserves and function about as well after a sleepless night as after a good night's rest.

After two or three nights of total sleep deprivation, however, small "microsleeps" (beginning with a few seconds' duration) intrude into wakefulness. These microsleeps cause occasional, short inattention to the task at hand, and performance then deteriorates even under high motivation. If the task allows for such short periods of inattention (eg, self-paced tasks), the performance decrement after extended sleep deprivation is smaller than if the task requires constant vigilance. Microsleeps become longer and more frequent as total sleep deprivation continues beyond three days. After about ten days, microsleeps are so intertwined with wakefulness that it becomes almost impossible to determine whether a person is actually awake or asleep, even if he performs functions usually associated with wakefulness (eg, walking). Apart from extreme sleepiness

and these microsleeps, however, there is remarkably little abnormal pathology associated with extended stints of sleeplessness. This was demonstrated by Randy Gardner, who showed no demonstrable abnormality after staying awake for 264 hours (Dement, 1972).

The fact that laboratory volunteers can function quite well after one to three nights of sleep deprivation contrasts with the feelings of patients with insomnia, who believe that they cannot function at all without their full quota of sleep. Psychological components seem to play an important role in this difference. The volunteer enters a sleep-deprivation experiment at a reasonably convenient time prepared for the sleeplessness. Besides the excitement, there is the challenge to perform well after sleepless nights and to see how long one can remain awake. In real life, however, sleepless nights usually come at the most inopportune times, for example, before an important test or before a board meeting. The night is spent in frustration, anxiety, and anger at oneself. This psychological handling of a sleepless night seems to contribute markedly to the general feeling of malaise the next day. Similarly, if sleeplessness is caused by a medical problem (eg, a developing cold, an allergy), the concurrent effects from the illness contribute to the general feeling of malaise from sleeplessness.

Obviously, chronic sleeplessness is not the same as spending a few nights in a sleep-deprivation experiment. Chronically poor sleepers facing anew a totally sleepless night might already have depleted their reserves and might be less able to "rally" a second wind. Nevertheless, it seems important to let insomniac patients know that they can function more or less adequately after one poor night, even though they feel moody and not up to par. This knowledge often helps them sleep better by eliminating some of the panic poor sleepers experience when facing a sleepless night.

Recovery: Recovery sleep after long wakefulness shows an increase in delta sleep and, on the first recovery night, often shows a decrease in REM sleep. Recovery sleep need not be equal in length to total sleep loss, and it usually cannot be taken all at once. Even after ten days of total sleep deprivation, a person rarely sleeps more than 14 to 20 hours at any one stretch, and two or three nights of extended recovery sleep usually will return a healthy volunteer to normal (Kales et al, 1970; Williams et al, 1964).

In summary, an occasional night of very poor sleep (or no sleep at all) seems of little medical concern in healthy individuals. However, short-term sleep losses may be of concern in certain types of epilepsy (lack of sleep appears to lower seizure threshold), in medically marginal patients, in jobs where an occasional lapse of attention might be critical (eg, air-traffic controllers), and possibly in jobs that depend on one's mood (eg, receptionists).

Therapeutic Deprivation: Surprisingly, although total sleep deprivation disturbs the mood of normal individuals, in patients who suffer from endogenous depression, a totally sleepless night imposed by the therapist seems to cause psychiatric improvements (Schulte, 1959; Van den Burg and Van den Hoofdakker, 1975). Unfortunately, however, such improvements last only a few days.

Why should a night of total sleep deprivation work as a treatment for endogenous depression? The reason is currently unknown. Perhaps, like other "shocking" procedures, loss of sleep temporarily startles the patients, rallies their reserves, and snaps them out of their depression. On the other hand, Pflug (1972) points out that both serotonin and norepinephrine imbalances are related to endogenous depression as well as to sleep. Possibly, a dramatic alteration of sleep might cause a temporary change towards the better in the disturbed neurotransmitter balances of endogenous depression, whereas the same alteration might wreak havoc with

the optimal tuning of the balances in normals.

Deprivation of Specific Sleep Stages

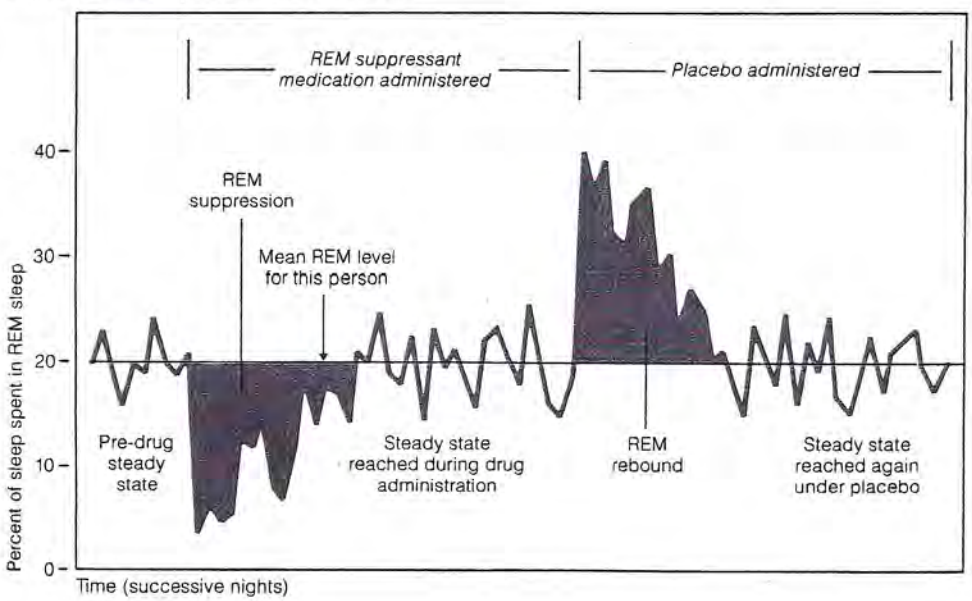
REM: When volunteers are REM-deprived by awakening them each time they start a REM period, "REM pressure" seems to build up. Upon falling asleep again, they usually wait less than the usual 1½ hours before they start REM sleep again (eg, only 45 minutes after the first REM awakening and 30 minutes after the second). They also make up the lost REM time as soon as they are permitted to do so (Figure 7). In addition, REM sleep becomes more intensive (eg, more phasic eye movements) when it is under pressure, and on awakening, certain behavioral changes occur.

Case History: *Mr. D., a college student, had volunteered for a study of REM deprivation in our lab. He was an extremely conscientious person making A + grades (which he earned mainly by studying exactly what was required for each course, handing all assignments in on time, etc.). He seemed to have total control of himself and to give himself no time at all for pleasurable, nonacademic pursuits. Mr. D. was REM-deprived for three consecutive nights by being awakened whenever the polygraph indicated the onset of REM sleep. On the fourth evening, he did not appear at the lab. Because he had reliably come to the lab exactly on time, an extensive search for him was undertaken. Two hours later, he was finally found attending a nearby burlesque show. According to him, he had never before even considered enjoying himself in that way. He claimed that after the three nights of REM deprivation, he suddenly felt different and didn't care what was going to happen or what he was supposed to do.*

In the early 1960s, it was thought that total deprivation of REM sleep for a few nights might lead to psychosis. This is not true. However, as was true of Mr. D., people who are REM-deprived seem to become more agitated, more impulsive, and less superego-controlled, whereas people deprived of delta sleep do not show these signs (Agnew et al, 1967).

Observing less superego control in normal individuals deprived of REM sleep, and considering that most antidepressant medications severely restrict REM sleep, Vogel et al (1976) suggested that the build-up of pressure for REM sleep might be the critical factor in the treatment of endogenous depression.

Figure 7. **REM Deprivation and Rebound**



Vogel et al deprived 17 hospitalized, endogenous depressives of REM sleep for three weeks by awakening them at the onset of each REM period. An equal number of similar patients were awakened the same number of times from NREM sleep. The REM-deprived group improved significantly more (were less depressed) than did the NREM-deprived group. On the other hand, patients with reactive depression did not respond very well to REM deprivation. Vogel et al also present evidence that endogenous depressive patients who do not respond to REM deprivation fail to respond to imipramine as well but can be treated with electroconvulsive therapy (ECT). Follow-up data suggest that Vogel's "REM-deprivation cure" is at least as efficient as are the usual drug treatments.

Not only do Vogel's studies suggest new ways of treating depression, they also raise theoretical questions concerning the mechanisms involved both in depression and in REM sleep. In addition, Vogel's studies demonstrate that "normal" sleep might not always be the best kind for everyone. This is important to remember in the evaluation of drug effects on sleep. It is commonly assumed that drugs that do not disturb the balance between sleep stages are preferable to those that do, but Vogel's work challenges that *a priori* assumption.

Delta: Volunteers deprived of delta sleep react differently than do volunteers deprived of REM sleep. Rather than becoming agitated and more impulsive, as occurs after REM deprivation, delta-deprived persons become physically uncomfortable, withdrawn, and manifest concern over vague physical complaints and changes in bodily feelings (Agnew et al, 1967). Also, muscles become more sensitive to pressure, and volunteers can withstand less musculoskeletal pain after delta deprivation than they could on the preceding evening (Moldofsky et al, 1976). This suggests that delta sleep might be related to musculoskeletal recovery, whereas REM sleep seems related to psychological recovery.

Dream Content

Dream analysis is beyond the scope of this review. Nevertheless, a few basic points concerning dream content might be made, because patients often insist on telling bothersome dreams to their physicians.

No matter how strange, a dream is created by the dreamer's own fantasy and thoughts. Thus, a dreamer might do well to take responsibility for his dreams, try to understand them, not fear them or laugh them off as "nonsense."

We seem to dream mainly about issues that concern us at the present time, such as fears, wishes, plans, hopes, worries. Thus, dreaming that a friend is dead does not necessarily signify a latent death wish but might well mean that one is concerned for the friend's health.

Dreams often relate to "unsettled business" stirred up during the day. For example, a glimpse of a girl may remind someone, for a split second, of a former girl friend. If engaged in conversation, the person may disregard this split-second thought. That night, however, issues relating to the girl friend may prompt dreaming, even though the waking stimulus for the dream cannot be recalled. If the issues related to the girl friend are still a matter of concern or are otherwise not properly "settled," it is even more likely that such a dream will take place.

The language of dreams is often symbolic and distorted. An apple might stand for a forbidden object; a wild tiger for rage. Disentangling the "real" meaning of the dream, therefore, needs detailed and intimate knowledge both of the dreamer and of dream mechanisms in general. Nevertheless, it is often beneficial to ask patients who insist on telling their dreams how they interpret them. In this way important concerns that the patient is unable to voice directly often surface and can be dealt with.

Sleep Hygiene

Folklore abounds with do's and don'ts concerning ideal sleep arrangements. Should we sleep on a hard mattress, in a cold room, on an empty stomach, alone, or with a partner? Before looking into these issues, some words of caution are necessary.

Most of us sleep poorly in an *unfamiliar environment.* This is known to sleep researchers as the "first-night effect." It means that sleep is usually disturbed in good sleepers on the first night in the laboratory. On the other hand, poor sleepers often sleep excellently on the first night in the lab, and some reasons for this paradoxical finding will be discussed on pages 58-63. What seems more surprising than the initially disturbed sleep in a new situation is the speed with which good sleepers can adapt to unfamiliar environments. In any case, before deciding whether a certain arrangement is disturbing or helpful to sleep, the sleeper needs to be thoroughly adapted to it.

A second problem relates to *individual differences.* One man's relaxation is another man's stress. Some need absolute quiet to fall asleep, whereas others do best when listening to music or when the TV is blaring. Robinson (1969) found that good sleepers slept even better after two hours of studying just before bedtime. On the other hand, the sleep of usually poor sleepers deteriorated even further after such studying. Individual differences appear to be especially prominent if the various environmental factors take on personal meanings. Many "fresh-air fiends" sleep well with windows wide open on a 40-degree spring night, but let the house get that cold in the middle of the winter, and they awaken from sleep and complain bitterly about freezing.

A third problem relates to the self-fulfilling prophecies that are so common in sleep work. A patient who expects to sleep poorly in a certain environment often worries about sleep and does poorly as a result of it. Expecting a good night's sleep in a certain environment reduces worry and this, in itself, contributes to good sleep.

Effect of Exercise

Everyone knows how soundly people sleep after exercise. Or do we? The question is more complex than originally thought, but an answer is important for theoretical as well as practical reasons. If sleep "restores our batteries," as many believe, we would seem to need more, or more efficient, sleep after exercise than after relaxation. Here are the facts as we know them today:

A chronic, constant amount of exercise does seem to benefit sleep, since athletes seem to have more delta (deep) sleep than nonathletes (Baekeland and Lasky, 1966; Zloty et al, 1973). Increasing exercise loads for one day in nonathletes does *not* seem to increase delta sleep on the following night (Hauri, 1968; Zir et al, 1971), but denying athletes their customary exercise decreases

the amount of their delta sleep (Baekeland and Lasky, 1966).

Exercise seems most effective in influencing sleep if it is done in the afternoon or early evening. Exercise in the morning has little effect, possibly because the ensuing wakefulness is sufficient for physiological recovery (Horne and Porter, 1976). Excessive exercise immediately before sleep seems to cause arousal and stress, which might interfere with possible beneficial effects of sleep (Hobson, 1968).

The amount of peripheral, physiological arousal following exercise influences neither sleep onset nor sleep stages in healthy young adults. Studying the effects of evening activities on subsequent sleep, Hauri (1968, 1969, 1970) had students exercise intensively for six hours on one evening and relax for six hours on another evening. Surprisingly, there was no difference in the ease with which subjects fell asleep after the two evening activities, nor in the development of sleep stages through the first three hours of the night. Sleep came just as easily after exercise as it did after relaxation, even though heart rate, respiratory rate, and rectal temperature were still significantly higher at sleep onset after exercise than after relaxation.

No condition seems as diametrically opposed to exercise as prolonged bed rest, yet two studies (Ryback et al, 1971a, 1971b) have shown that bed rest of five weeks' duration *increased* the amount of delta sleep per night. This startling finding contradicts all commonsense notions about a direct relationship between delta sleep and physiological recovery after exercise.

Keeping in mind that these studies were performed on healthy volunteers, there seem to be two possible explanations. First, there are only two ways of dramatically changing muscle mass, either by hypertrophy (following exercise) or by disuse atrophy. Perhaps a similar intensity of physiological recovery and repair (as evidenced by delta sleep) is needed after each of the two conditions. Second, one might speculate that slow EEG waves are merely an epiphenomenon, not the "essence" of the deep, sound sleep we have come to associate with the delta stage. Slow EEG waves and some presumably more basic processes of delta sleep might usually show a high correlation, yet the association between the two events might be lost under atypical conditions such as sensory deprivation or bed rest (Zubek et al, 1963). In any case, there are conditions, such as certain organic brain syndromes, where slow EEG waves do not imply delta sleep.

The issue does not seem resolvable at this time. Nevertheless, in terms of sleep hygiene, the following recommendations can be supported by current research findings:

- A steady amount of exercise is probably beneficial for sound sleep, but one cannot force sleep on any given night by exercising excessively during the preceding day. Rather, such excessive exercise may result in increased aches and pains that might detract from sleep on the following night.
- If one exercises in order to sleep well, such exercise should probably come in the afternoon or early evening, not in the morning or just before sleep.

Effect of Environment

Noise: How much noise can we tolerate before our sleep becomes disturbed? To answer this question, one needs to qualify it in at least six different ways.

First, the awakening threshold depends on the stage of sleep. Stage 1 is the lightest stage; that is, it takes the least amount of noise to cause full arousal, whereas stages 2 and REM are deeper, and delta sleep is deepest (Rechtschaffen et al, 1966). However, human REM sleep can be very deep if the external noise is somehow incorporated into the dream content. Bradley and Meddis (1974) measured an average REM arousal threshold of 60 decibels on the A level (dBA) in their sleepers when the noise they fed into the sleeper's room was not

incorporated into the dream but an average threshold of 70 dBA when it was incorporated. They gave the following example of a dream that incorporated the noise and which had, therefore, a high arousal threshold:

We had to ask for more stationery, and you have to fill in a form to ask for it and that noise started and my father did not know how to stop it, and he picked up a file and flicked through it, looking for this form and when he came to the form that would stop the noise... but there wasn't a form in the file. So that noise wouldn't stop.

Second, the auditory awakening threshold depends on how much sleep a person has already accumulated during the night. The same stage, later in the night, is lighter than it was earlier in the night (Rechtschaffen et al, 1966).

Third, individual differences in sensitivity to noise seem very important. Some people awaken more easily than others, even from the same sleep stage. In the study by Rechtschaffen et al (1966) mentioned above, the average awakening threshold throughout the night was 15 dBA for one sleeper and more than 100 dBA for another.

While examining individual differences in arousal thresholds, Zimmerman (1970) found that light sleepers showed faster heart and respiratory rates than did deep sleepers, higher body temperature during sleep, more spontaneous awakenings, and more body movements while asleep. These differences between light and deep sleepers are similar to those observed by Monroe (1969) between poor and good sleepers. Zimmerman, therefore, speculated that his light sleepers might be the population "at risk" for developing insomnia later on.

Fourth, the specific meaning of the noise seems important. The mother who sleeps through thunderstorms but awakens when the baby fusses in the next room is well known. Apparently, a "night light of consciousness" is still burning during sleep, discriminating among incoming auditory stimuli and deciding on appropriate responses. Morgan (1970) discusses such a mechanism in detail, but there has been little factual research carried out on this point.

Fifth, the age of the sleeper seems crucial. Although hearing usually becomes less acute with age, the sleep of older patients seems to be more sensitive to noise than does that of younger people. Lukas and Kryter (1970) present evidence that older sleepers awaken much more frequently to sonic booms than do younger sleepers. Similarly, Roth et al (1972) found that the same noise level that awakened 70 year olds caused only a temporary shift toward stage 1 sleep in 25 year olds.

Finally, noise sensitivity is different in men than in women. Dobbs (1972) established that women awaken more easily to airplane noise than do men.

Keeping these qualifications in mind, we can now examine the effect of noise, particularly truck and aircraft noise, on sleep. Most studies have used young adults, who may be subjected to simulated aircraft noise or truck noise during some laboratory nights, to no noise during other nights. Unfortunately, this procedure resembles having a country man suddenly try to sleep close to a busy airport. Rarely is enough time given for adequate adaptation. Nevertheless, Chiles and West (1972) found no evidence that one sonic boom per hour during sleep produced any measurable consequences on a complex task performed the next day. On the other hand, LeVere et al (1972), using nine simulated jet aircraft overflights per night (20-second duration, maximum intensity 80 dBA), found that daytime performance on a complex monitoring task declined after the flyover nights, in spite of the fact that normally the noise had not awakened the subjects. In the morning, many of them were unable to recall a single flyover. Nevertheless, the sleep EEG of these subjects characteristically had shown a marked desynchronization in response to the noise, indicating a movement towards lighter sleep stages.

This desynchronization lasted, on the average, for five minutes after the noise had disappeared. In a comparison between sonic booms and truck noise, Berry and Thiessen (1970) found that the longer-lasting truck noises at 70 dBA produced more awakenings than did the shorter and more impulsive sonic booms at 120 dBA.

Herbert and Wilkinson (1973) stimulated ten subjects during one of five nights with clicks of variable loudness (65, 75, 80, 90 dBA) administered every 20 seconds. These clicks caused a significant increase in stage 1 and wakefulness and a trend towards less REM and less delta sleep. However, the effect of such a sleep disturbance on awakening performance (measured with vigilance tasks and addition tests) was relatively small and confined to the early waking hours.

Studying the problem of human adaptation to noise for extended periods, Cantrell (1974) had 20 young males confined to a dormitory for 55 consecutive days. During 30 of these days, high-pitched tones at 80, 85, and 90 dBA were emitted through omnipresent loudspeakers at 22-second intervals, 24 hours per day. Although the volunteers complained that the 85 and 90 dBA tones interfered with their ability to fall asleep, this complaint could not be substantiated in the laboratory. However, delta sleep did decrease during this relatively prolonged and stressful time. Townsend et al (1973), in a similar experiment, found similar results.

The most direct answer to the question of whether chronic noise interferes with normal sleep comes from Globus et al (1973), who studied the sleep of long-term Los Angeles residents in their own homes. Half of the volunteers in this study lived adjacent to Los Angeles International Airport; the other half resided in quieter Los Angeles neighborhoods. Even though each of the subjects had resided in the same neighborhood for at least six years, those close to the airport still showed less delta sleep, more wakefulness, and more stage 1 sleep than did residents living in quiet neighborhoods. In fact, residents close to the airport recorded 45 minutes less "useful" sleep (stage 2, delta, and REM) per night than did residents living in quiet neighborhoods, and the disruptions and awakenings in their sleep could be directly traced to aircraft flyovers.

Would it help to attenuate the noise by the use of earplugs or to mask it by a constant noise such as an air conditioner? Otto (1972) found that fiberglass earplugs (with about 20 dB attenuation) helped solidify sleep in the face of normal street-traffic noises. As for screening, Scott (1972) found that a constant white noise of 93 dBA decreased REM and increased stages 1 and 2 sleep, although it did not change the number of awakenings or the amount of delta sleep when compared with quiet nights. Because Scott studied only one night, it is unclear whether one might gradually habituate to the white noise over successive nights. That such *habituation* can occur during sleep has been established (Johnson et al, 1975; McDonald and Carpenter, 1975), although adaptation is much slower during sleep than it is during wakefulness.

In summary, loud noise does disturb sleep, even in people who do not awaken from it, do not remember the noise in the morning, or supposedly are habituated to it. Noise usually shifts sleep away from delta and away from REM, and it increases the number of awakenings and the amount of stage 1 sleep. Muffling excessive noise that can reach the bedroom seems to be a good idea for most people. However, whether a specific noise is disruptive to the sleep of a specific person depends largely on individual differences, such as age, sex, the meaning of the noise, and the physical properties (pitch, duration, and amplitude) of the noise.

Physical Surroundings: Throughout history, bed preferences have ranged

Table I **Ten Rules to Better Sleep Hygiene**

1. Sleep as much as needed to feel refreshed and healthy during the following day, but not more. Curtailing time in bed a bit seems to solidify sleep; excessively long times in bed seem related to fragmented and shallow sleep.
2. A regular arousal time in the morning seems to strengthen circadian cycling and to finally lead to regular times of sleep onset.
3. A steady daily amount of exercise probably deepens sleep over the long run, but occasional one-shot exercise does not directly influence sleep during the following night.
4. Occasional loud noises (eg, aircraft flyovers) disturb sleep even in people who do not awaken because of the noises and cannot remember them in the morning. Sound attenuating the bedroom might be advisable for people who have to sleep close to excessive noise.
5. Although an excessively warm room disturbs sleep, there is no evidence that an excessively cold room solidifies sleep, as has been claimed.
6. Hunger may disturb sleep. A light bedtime snack (expecially warm milk or similar drink) seems to help many individuals sleep.
7. An occasional sleeping pill may be of some benefit, but the chronic use of hypnotics is ineffective at most and detrimental in some insomniacs.
8. Caffeine in the evening disturbs sleep, even in persons who do not feel it does.
9. Alcohol helps tense people to fall asleep fast, but the ensuing sleep is then fragmented.
10. Rather than trying harder and harder to fall asleep during a poor night, switching on the light and doing something else may help the individual who feels angry, frustrated, or tense about being unable to sleep.

from the hard floor to the extremely soft beds of the 18th century in which one slept in a near-sitting position. Similarly, many Europeans today sleep with the head part of the mattress elevated by a special triangular supplement, whereas Americans sleep on flat mattresses. There is no evidence that such differences affect sleep once one is accustomed to them.

Are the shape and the quality of the mattress important to sleep? Kleitman (1963, p 309) pointed out that the curvature of the spine does not seem to be influenced by how straight one lies in bed: "Aside from the fact that the spine has a couple of natural curves, there is no evidence that the spine curvature is changed in the cat, which sleeps curled up, in the Japanese, who sleep on the ground, or in sailors, who often sleep in bona fide hammocks with a considerable sag in the middle." Well-constructed mattresses may be important for people with orthopedic problems; they seem unnecessary for most normal sleepers.

It has been established that excessively hard surfaces (ie, wooden floors) can cause more body movements, more awakenings, and more stage 1 sleep than do softer surfaces (Kinkel and Maxion, 1970). Beyond this, however, sleep seems intrinsically very little affected by the characteristics of the sleeping surface, except for matters of personal preferences and style. Even such exotic situations as air-fluidized bead beds seem to have few lasting effects on sleep in normal subjects (Dement et al, 1971; Shurley, 1971; Rosekind et al, 1976).

If the type of mattress used does not influence sleep, as long as it is reasonably comfortable, why should patients sleep better on air-fluidized beds (Kline et al, 1974) or waterbeds? As shall be discussed later (p 60), some patients are conditioned *against* relaxing in their own beds. In these patients, the more the new sleep situation differs from the old one, the less conditioning can carry over from the old to the new situation. Add to this

a healthy dose of placebo when one changes to a more expensive mattress, and it may well be that sleep improves quite dramatically in specific cases.

In a study conducted by Monroe (1969), couples who were good sleepers and habitually slept in the same bed were studied in the lab. They slept together on some nights, apart on others. Monroe found more delta sleep when the couple slept apart, and somewhat less REM sleep. Sleeping together may be good for marital bliss, but sleeping apart does seem to deepen sleep by preventing disturbances when the partner changes position.

Temperature: Ideal sleep temperatures differ for different species: the rat apparently sleeps best at 30 to 32 C (86 to 90 F) (Valatx et al, 1973), whereas cats appear to sleep longest at around 22 C (72 F) (Parmeggiani and Rabini, 1970). Unfortunately, an ideal temperature for humans has not been determined to date. A study of sleep differences in psychiatric patients sleeping at 18 C, 23 C, and 29 C (64 F, 74 F, 84 F) found no consistent trends toward better sleep in a particular temperature (Presley et al, 1973). The old belief that sleeping is best when the bedroom is cool, therefore, still awaits scientific validation. It has been determined, however, that room temperature will influence dream content. As the room temperature was decreased from 22 C (72 F) to 17 C (63 F) and later to 12 C (54 F), dreams became more emotional and more unpleasant (Ziegler, 1973).

Sleeping in a hot room (temperature much above 24C [75F]) seems to be bad for humans: they wake up more, move more in their sleep, and both REM and delta sleep are decreased (Schmidt-Kessen and Kendel, 1973; Otto et al, 1971). This is probably why Kleitman, as early as 1937, found more body movements and more interruptions in the sleep of his Chicago subjects during the summer than during the winter.

It appears that during REM sleep, thermoregulation is seriously impaired, and animals as well as humans become poikilothermic (Baker and Hayward, 1967; Shapiro et al, 1974). Sleeping in extreme temperatures might, therefore, have less effect in the early part of the night when REM periods are short, but it might possibly become disastrous later on when REM periods become longer.

Weather: Popular thought has long maintained that specific weather conditions could influence sleep. Sleep is often said to be especially poor when a front is approaching or when particularly dry winds prevail, such as the Santa Ana in Southern California or the föhn in Switzerland and Austria.

Some scientific evidence now supports the popular association between weather and sleep. Raboutet et al (1959) found a statistically significant relationship between barometric pressure and sleep onset as assessed in routine EEGs done on aviation personnel: the higher the barometric pressure was, the fewer were the people who fell asleep during a clinical EEG. Webb and Ades (1964), following up on Raboutet's lead, found that both extremely high and extremely low barometric pressures were associated with increasing sleepiness during clinical EEGs.

Effect of Food

Caloric Intake: Losing or gaining weight often has surprisingly profound influences on sleep. In general, weight gain is associated with long, uninterrupted sleep, whereas weight loss is associated with short and fragmented sleep. This has been demonstrated most convincingly by Jacobs and McGinty (1971) in rats: the less food their rats received, the less they slept, until after 6 to 11 days of food deprivation, virtually all sleep disappeared. The survival value of a food-sleep association is obvious: Starving animals ought to be awake and looking for food rather than sleeping; satiated animals are probably safest if they can hide and sleep for long periods. Pa-

rentheticaly, one wonders whether in our weight-conscious society a fair amount of poor sleep might be directly related to semistarvation diets.

Evidence for an association between food intake and sleep in humans comes mainly from psychiatric clinics. Irrespective of their diagnosis, psychiatric outpatients who have recently lost weight usually complain about poor sleep and more awakenings, especially in the second half of the night. Patients who recently have increased their weight usually sleep long and soundly (Crisp and Stonehill, 1973). In addition, insomnia is a common symptom in anorexia nervosa (Lacey et al, 1975); when such patients are fed, their sleep improves. Similarly, in depression, it appears that most patients both lose weight and become insomniacs, although some depressed patients increase their weight and sleep for excessively long times (Michaelis and Hofmann, 1973).

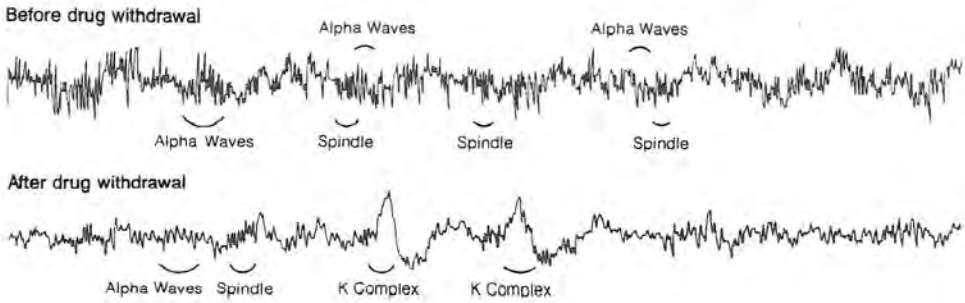
Why should food intake be related to sleep? Fara et al (1969) introduced milk or fat directly into the duodenum of cats and observed improved sleep. They postulated that sleep was induced either by the direct, central action of a released gastrointestinal hormone or by indirect neurogenic mechanisms triggered through the stimulation of duodenal receptors. On the other hand, it seems quite possible that some of the association between food and sleep might be secondary to thyroid function. Hyperthyroidism is associated with weight loss and short but intensive sleep (much delta), whereas hypothyroidism results in weight gain and long sleep with a relative lack of delta (Kales and Kales, 1974).

The association between food intake and sleep is not invariably present; some studies have been unable to confirm the above findings. Nevertheless, chronic insomniacs and those awakening too early in the morning might do well to try a midnight snack in an effort to solidify their sleep.

Tryptophan and Sleep: Postprandial drowsiness after a good meal is a common phenomenon. Similarly, warm milk, Ovaltine, and Horlicks (a Scotch malt drink) all improve sleep significantly when taken before bedtime (Kleitman et al, 1937; Brezinova and Oswald, 1972). It appears that these phenomena might not only be related to caloric intake (as discussed above), but specifically could affect the ingestion of L-tryptophan, the serotonin precursor, that exists in many foods. As discussed before, serotonin appears to be related to sleep mechanisms, and Hartmann et al (1974) have demonstrated that as little as 1 gm of tryptophan can clearly improve sleep in some patients.

The production of serotonin depends on the availability of tryptophan which is

Figure 8. **Stage 2 Sleep Patterns Before and After Withdrawal From Hypnotics (Mr. D.)**
Note the mix of spindling and alpha waves before withdrawal, and the relative absence of alpha waves and the reappearance of K complexes after withdrawal.



carried into the brain on a transport protein. Unfortunately, the transport protein has a lower affinity for tryptophan than for four major competing amino acids. Thus, increased amounts of tryptophan will enter the brain only if blood concentrations of tryptophan are increased relative to the concentration of the competing amino acids (Wurtman and Fernstrom, 1974; Kolata, 1976).

Sleep Medication

Hypnotics are not entirely harmless. Carelessly prescribed over months and years, they apparently can destroy sleep, as the following case history illustrates.

Case History: *Mr. D., 58 years old, operated a small business. He complained of inability to sleep even though he took at least 40 mg of diazepam, 90 mg of flurazepam, and 400 mg of secobarbital each night. When this mixture did not help, he often used up to a fifth of whiskey to induce sleep. He was referred to the sleep clinic after his fourth "blackout episode." According to Mr. D., blackouts occurred on the nights when he took "whole handfuls" of sleeping pills in a desperate attempt to find some rest. Four times after such episodes, his wife found him lying on the floor unconscious and was unable to arouse him; she then rushed him to the hospital. On one occasion, he remained in a coma for five days before recovering.*

A sleep evaluation revealed that Mr. D.'s habitual nightly dose of hypnotics produced absolutely ***no*** *normal sleep. Although the EEG did slow somewhat for about two hours, and a few eye movements around 3 AM suggested a weak attempt at a REM period, nothing in Mr. D.'s record looked like normal sleep. Nevertheless, Mr. D. claimed that his lab sleep had been "much better" than a normal night at home.*

Mr. D. related that he had been a good sleeper until two weeks after Pearl Harbor, when he had become plant manager. Being ill-prepared for the job, he then started to worry and to sleep poorly, and a barbiturate was prescribed. Over the next 30 years, his sleep gradually deteriorated, and he required more and more hypnotics and alcohol to reach a state of oblivion.

Mr. D. was admitted to the hospital and gradually withdrawn from all hypnotics. He was taught electromyogram (EMG) biofeedback and relaxation exercises. Alcoholics Anonymous became involved because of his drinking, and social ties to family and friends were re-established in joint therapy. A low-grade chronic depression was treated with 100 mg doxepin h.s. Six months later Mr. D. started to function adequately once more. He still sleeps poorly but does get up more refreshed. Laboratory studies now show about five hours of normal sleep but still a high number of awakenings (Figure 8).

Mr. D. obviously suffered iatrogenic insomnia. His case is not rare. Most sleep clinicians know patients who received a hypnotic for a temporary disturbance (such as surgery) but from whom the drug was never withdrawn. Over the years, dosages gradually crept upwards until natural sleep became impossible (Kales et al, 1974).

Franklin (1969) demonstrated that an entire psychiatric admission ward can be managed quite well with very little use of hypnotics, once the staff has changed its attitude towards sleeping pills. Unfortunately, in the United States, the average complaint of insomnia is dealt with in less than three minutes by prescribing hypnotics (Dement, personal communication). This is worrisome, because many patients who initially complain about sleeplessness do not suffer from insomnia when evaluated in the lab. In these instances, extensive probing may be necessary to uncover their basic psychiatric or medical reasons for seeking help. Others, although true insomniacs, suffer from problems not treatable with hypnotics (as shall be discussed below).

Although the overprescription of hypnotics may be an important cause of chronic insomnia, the informed use of hypnotics is essential in the management of sleeplessness. Some rules for such a use of hypnotics will therefore be developed here without, however, discussing the details of each prescription hypnotic and over-the-counter sleeping pill. Careful reviews comparing specific hypnotics have recently been published (Johns, 1975; Greenblatt and Miller, 1974). In controlled studies, hypnotics are more alike than different. Most show basically the same course (Figure 9): an initially

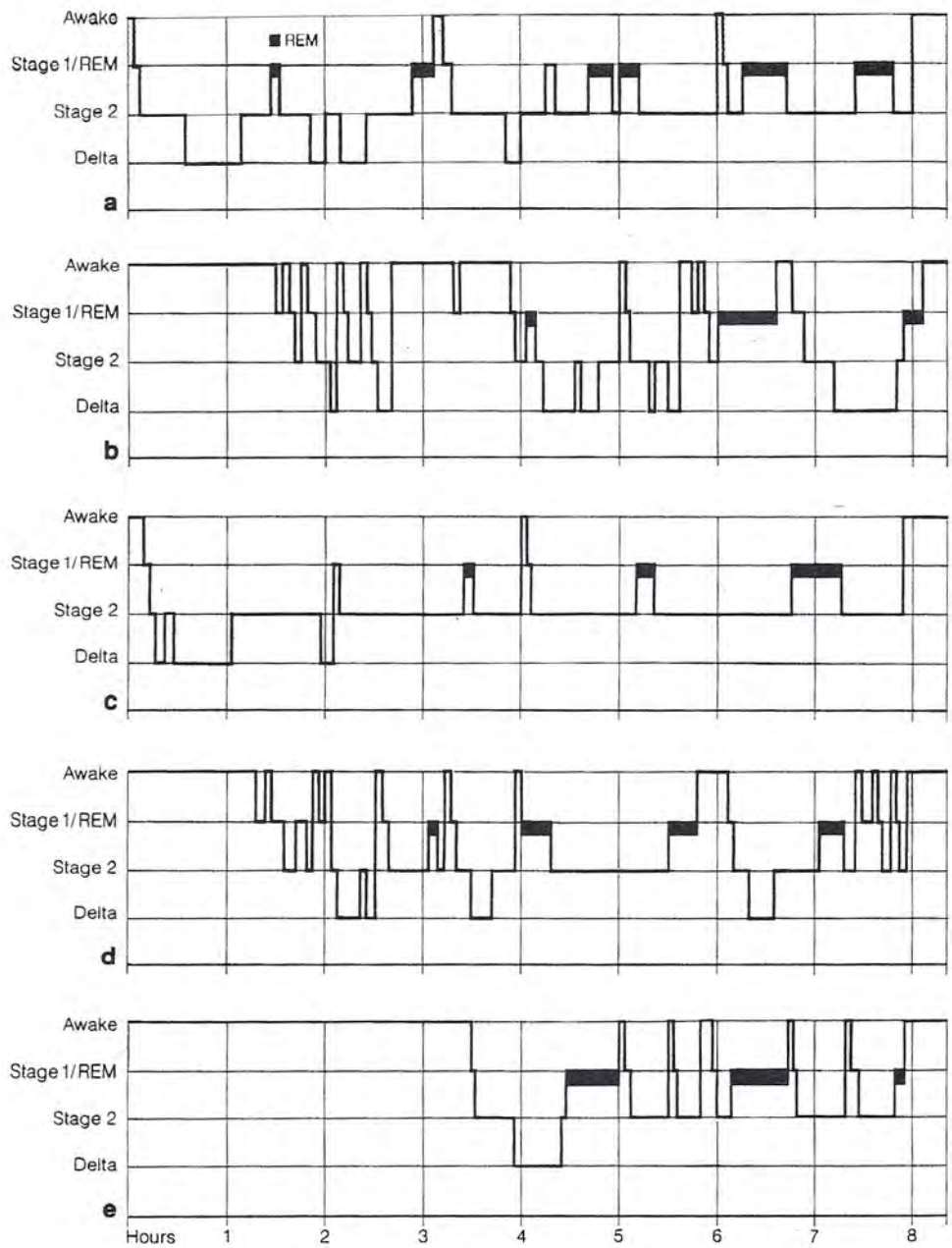


Figure 9. **Development of Sleep in a 17-Year-Old Insomniac on 100 mg Secobarbital**

a. Typical sleep cycle of a young adult (taken from Figure 2).
b. Sleep of the 17-year-old insomniac on placebo – long sleep latency, fragmented sleep.
c. Sleep on the first drug night – note solid sleep and long delay of REM sleep.
d. Sleep on fifth drug night – insomnia has returned: long sleep latency, fragmented sleep.
e. Sleep on the first drug-withdrawal night: sleep latency of 3½ hours, fragmented sleep.

satisfactory reaction that diminishes over time. Also, most patients show a "rebound insomnia" when withdrawn from their hypnotic. Such a pattern is graphed in Figure 9. A young adult with sleep-onset insomnia was recorded in the clinic, then 100 mg secobarbital was prescribed. While on this medication, the patient seemed to sleep well the first night, but after as few as five nights, the insomnia returned. When the drug was discontinued after one week of chronic use, the patient initially slept even more poorly than before the medication was given.

Clinical Efficacy: More important than the seemingly objective efficacy of a hypnotic is the subjective feeling of having slept well, the ability to function well during subsequent wakefulness, and the lack of any medical and psychological damage that might be caused by the hypnotic. A drug that could consistently produce a good feeling during wakefulness, while maintaining sound psychological as well as medical health on a long-term basis, would be the ideal drug. Thus, the *main* questions when evaluating hypnotics are "Does the patient feel better and more alert during the day following the use of hypnotics? Does he do so without incurring a debt in terms of side effects and/or medical and psychological sequelae?"

The following five efficacy questions are also crucial when evaluating a hypnotic (see Figure 10):

1) *How effective is the drug on the first night?* What is the minimum adequate dose for securing a night of adequate sleep, and how close is this dose to a toxic one?

2) *How long is the drug effective?* Some drugs (eg, secobarbital, 100 mg) lose their sleep-inducing potency within one week of chronic administration (Kales et al, 1976). Others (eg, flurazepam) lose efficacy more gradually, although they are still better than is the placebo after one month of chronic administration (Kales et al, 1975). In addition, the shape of the curve seems important. Many hypnotics are most effective on the first night of use, but some, with long half-lives of their active components, are more effective on the second drug night.

3) *What level of sleep will be produced with chronic use?* In Mr. D.'s case (above), the chronic administration of hypnotic drugs eventually seems to have produced less sleep than would have been obtained without drugs. Other drugs apparently level out at the patients' average sleep time, letting the insomniacs sleep about as poorly with the chronic use of a hypnotic as without it. No drugs have yet been shown conclusively to improve sleep permanently over baseline conditions.

4) *What is the immediate effect of withdrawal?* When a hypnotic that has been taken chronically is precipitously withdrawn, total sleep times of less than one half hour per night are common. It is then often imagined that such a poor sleep would be the patient's lot forever should he withdraw from the medication. This erroneous assumption prolongs unnecessary dependency on hypnotics. However, care is indicated when withdrawing medication. A precipitous withdrawal from certain hypnotics can cause serious medical sequelae, such as the seizures induced by sudden barbiturate withdrawal.

5) *How long do the withdrawal effects last?* Most studies document withdrawal effects only for three or four days, but patients often complain of sleeping poorly after drug withdrawal for up to a month. In the laboratory, Haider and Oswald (1970) found that overdosages of certain hypnotics resulted in abnormal sleep patterns for up to two months after complete withdrawal.

Nobody has yet documented the long-range effects of hypnotic drugs when used for months, years, or decades, even though this may be a serious health question. Obstacles to studying this problem are enormous. It is difficult to assess pre-drug levels of sleep and psychological function in those who have taken hyp-

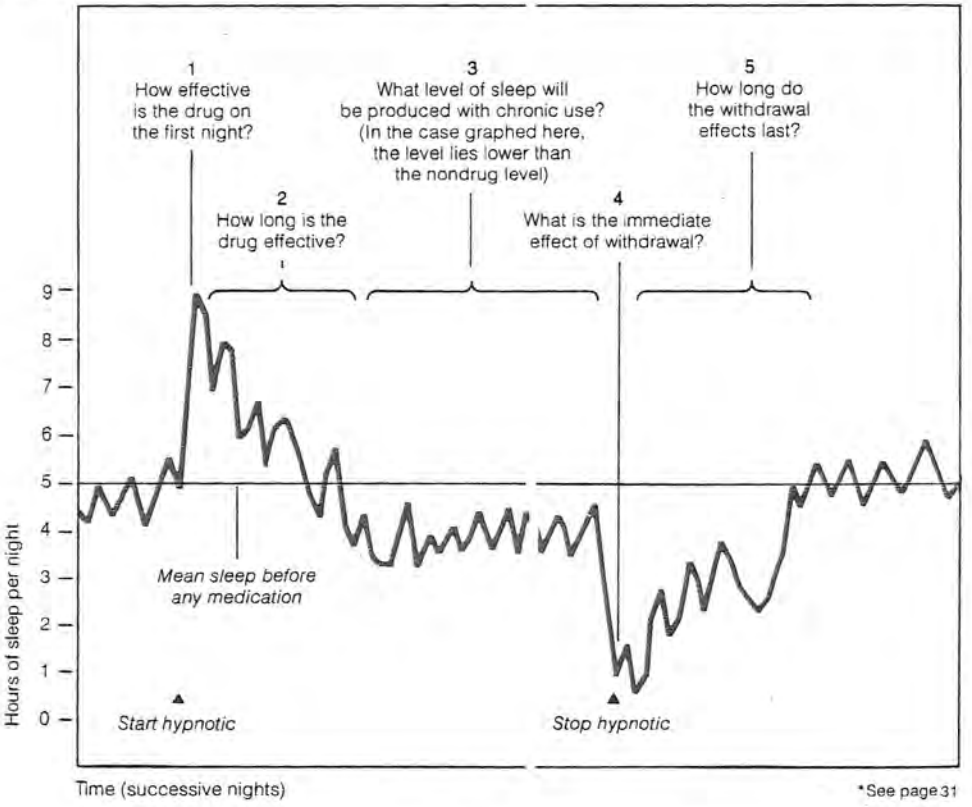
notics over a long period of time, and to do the needed prospective study would strain finances and resources considerably. Nevertheless, the costs of not knowing the answer may, in the long run, far outweigh the costs of finding out, both for individuals and for society as a whole.

Before using a hypnotic drug, each patient should be familiar with the basic curve as outlined in Figure 10. A patient might be advised, for example, to take the hypnotic each night for the next two weeks because of specific job pressure, providing he can then take some days off from work to accommodate the "insomnia-rebound" which inevitably will follow withdrawal. Similarly, an insomniac might decide to take the medication during weekdays but not on weekends, or on alternate days, or only during two out of three weeks.

While these procedures make intuitive sense, no acceptable research has been done to date on this topic of "drug holidays," a serious and glaring blind spot in our knowledge.

A careful evaluation of the effects hypnotics have on the different sleep stages is not included in this discussion. According to Johnson (1973), there is little difference in the recovery value of different sleep stages, at least in terms of subsequent performance. Similarly, some humans can apparently function quite adequately without any REM sleep (Wyatt et al, 1973), and the well-documented lack of delta sleep following the administration of benzodiazepines does not seem to have much effect on wakefulness, either. The only documented concern in the area of hypnotics and sleep *stages* is the REM rebound. Withdrawal

Figure 10. **The Five Efficacy Questions for Hypnotics***



*See page 31

from most hypnotics usually results in an increase in and intensification of REM sleep. This REM rebound may cause increased and unpleasant dreaming, even nightmares, and patients should be warned about this possibility when withdrawing from hypnotics.

Agents Used to Induce Sleep

Although many different hypnotics are currently marketed, the general trend recently has been towards using the benzodiazepines whenever possible. This trend has a rational basis, although it should be clearly understood that the benzodiazepines are not yet the ideal drugs, and we do not know the effects of these drugs when used for months or years. Also, benzodiazepines do not induce sleep in all insomniacs.

Some of the rational reasons for using benzodiazepines are:

- Long-range effectiveness (up to one month) as a hypnotic has been demonstrated for some benzodiazepines, but not for the other sleeping pills.
- Fatal overdoses are much less common with benzodiazepines than they are with such drugs as barbiturates, glutethimide, ethchlorvynol, chloral hydrate.
- Benzodiazepines as hypnotics are easier to withdraw than are other hypnotics, apparently in part because the half-life of some active metabolites in this type of drug extends beyond 24 hours. Thus, sleep is still somewhat aided by the benzodiazepines on the first withdrawal night.
- Barbiturates, glutethimide, and possibly ethchlorvynol potentiate certain liver enzymes and therefore influence the activity levels of drugs given concomitantly. Antihistamines potentiate anticholinergic drugs. Chloral hydrate influences protein binding. Benzodiazepines, on the other hand, do not seem to interact significantly with other medications.

Sedating tricyclic antidepressants have been advocated by certain clinicians because they appear to maintain effectiveness longer than do the hypnotics. Such antidepressants seem especially indicated if the patient is depressed as well as insomniac, and several authorities have suggested giving all of the antidepressant medication in one dose at bedtime (Karacan et al, 1975). However, Flemenbaum (1976) warns that combining all antidepressant medication into one single dose at bedtime apparently did lead to some terrifying nightmares in his patients. Such nightmares are rare, however, if very low doses of sedating antidepressants (eg, 25 mg amitriptyline) are administered for the sole purpose of solidifying sleep or abolishing alpha intrusion into sleep, as will be later discussed (see page 51).

Over-the-counter medications advertised as soporiphics usually contain scopolamine and/or methapyrilene, which make a patient groggy. If the patient's main problems are worries about not sleeping, feeling this grogginess apparently relaxes him because he thinks that "the sleeping pill has already started to work." This thought alone will make it easier for many to fall asleep naturally. However, over-the-counter drugs do not induce sleep directly and are useless in more-serious insomnias (Kales et al, 1971).

Aspirin is widely used to induce sleep. In some cases, simple reduction of pain may be the explanation. However, aspirin also increases the proportion of free tryptophan in the blood. Inasmuch as free tryptophan can bind to the transport protein and cross the blood-brain barrier, and tryptophan bound to serum albumin cannot, it seems quite possible that aspirin might directly improve sleep by increasing brain serotonin levels. No adequate study of this hypothesis has been done to date.

Although most hypnotics are unsatisfactory in the long run, some breakthroughs may well be expected in this area within the next few years. Hopefully, we shall learn more specifically how to alter certain neurotransmitter balances at the appropriate sites, rather than attempting to induce sleep by an

overall depression of CNS activity. Hopefully, we shall also learn better how to get the desired drug directly to specific areas of the brain rather than having to saturate the entire body.

Four Rules for the Use of Hypnotics

- If a person is under short-term stress mainly manifested by sleeplessness, a hypnotic drug to help him sleep for a few nights seems indicated. However, care should be taken to withdraw this hypnotic either when the stress has terminated, or no more than two to four weeks after the initial prescription, depending on the type of hypnotic prescribed. Should the stress manifest itself in daytime anxiety, a general tranquilizer seems more appropriate than the often-prescribed hypnotic.
- Many insomniacs have a tendency to panic when they feel a series of poor nights descending. A prophecy of a poor night then becomes self-fulfilling as the panic increases agitation. Such patients should be given a few effective sleeping pills but admonished not to take more than one or two per week. The mere possession of a hypnotic has a very relaxing and reassuring effect in such patients, and the sleeping pills will often not be needed.
- Each patient using hypnotics should be familiar with the basic relationships as outlined in Figure 10 so that he can use his hypnotics wisely.
- Many patients use low doses of hypnotics for years without undue side effects and without needing to increase the dosage. The placebo effect of such sleeping medication is apparently enough to induce sound sleep. There seems to be little reason to categorically withdraw such patients from all hypnotic use. However, patients who continually need to increase their medication while remaining dissatisfied with their sleep probably should be withdrawn from all hypnotics and treated behaviorally, by other pharmacological means (such as antidepressants) or by psychotherapy.

Specific Sleep Disorders

Nosological Considerations

Only during the past ten years have the problems of sleep pathology been attacked by the technology of sleep research (eg, Kales and Kales, 1970; Karacan et al, 1971; Oswald, 1969). Numerous ways of classifying sleep problems have been suggested since then, but none have yet gained universal acceptance. Currently a subcommittee of the Association of Sleep Disorders Centers is working on the classification of sleep disorders, but their deliberations are not yet concluded. The classification system used in this report follows the most commonly used nosological categories, but adds a category of "disorders secondary to behavioral problems," which has not been previously proposed.

Until recently, sleep disorders were classified into the *insomnias* (not enough sleep), the *hypersomnias* (too much sleep), and the *dyssomnias* (miscellaneous disturbances occurring during sleep, such as enuresis and sleepwalking). Logical as such a classification might seem, the more one works with it, the less satisfactory it becomes. Issues that should be grouped together (eg, central sleep apneas, resulting primarily in insomnia, and upper airway apneas, resulting primarily in hypersomnia) are split apart in such a system. In addition, many sleep disorders belong in more than one category. Narcolepsy, for example, is usually classified as a form of hypersomnia because excessive and inappropriate sleep attacks during the day are its main symptoms. However, most narcoleptics also sleep very poorly at night and might just as well be classified as insomniacs. Worst of all, excessive daytime sleepiness, the hallmark of hypersomnia, is also an expected outcome of chronic insomnia. Thus, hypersomnia and insomnia have

the same chief complaint of excessive daytime sleepiness, and this has led to unnecessary semantic arguments.

The nosology used here first separates problems into *primary and secondary sleep disorders.* Primary sleep disorders (as defined by Williams et al, 1974) include the disorders in which disturbances or abnormalities of sleep are the *principal* or only symptoms of the problem (eg, sleep apnea, narcolepsy, primary insomnia). Secondary sleep disorders are those in which the sleep problem is only part of a symptom complex belonging to a more widely ranging clinical problem.

Among the secondary sleep disorders, the current review classifies them along traditional lines: sleep disorders secondary to psychiatric problems, secondary to medical problems, or secondary to behavioral problems. Clearly, these three categories are not mutually exclusive. Poor sleep secondary to alcoholism, for example, could be classified into any one of the three categories (it has arbitrarily been put into psychiatric problems in this review). Also, for purposes of simplification, sleep-exacerbated disorders are included among the sleep disorders secondary to medical problems, mainly because this review does not focus heavily on this type of problem.

A third category, the parasomnias, includes those activities that would be more or less normal if executed appropriately during waking, but are pathological during sleep (eg, sleepwalking, nightmares, enuresis, bruxism). These disorders are often called dyssomnias, but *Dorland's Illustrated Medical Dictionary* (1975) has preempted that term by defining dyssomnia as any disorder of sleep.

Two further terms need clarification: *Insomnia,* as discussed under Sleep Needs (page 17), is defined as a chronic inability to obtain the amount of sleep necessary for efficient daytime functioning. Sleep needs vary, and insomnia cannot be defined in terms of absolute hours. A person who needs nine hours, but is chronically unable to obtain more than seven, has insomnia, but a person who needs and obtains five hours of sleep each night is not an insomniac.

Excessive Daytime Sleepiness (EDS) has replaced the term "hypersomnia" in most circumstances. This is so primarily because many people formerly classified as hypersomniacs do not actually sleep much longer than normals when measured over 24 hours. Rather, their main problem is that they chronically feel sleepy, no matter how many hours they allot to their sleep.

As will become clear, knowledge needed to treat sleep disorders involves general medicine, endocrinology, neurochemistry, neurology, otolaryngology, and cardiology, as well as psychiatry, psychology, and other behavioral sciences. Historically, these disciplines have shown little interest in the clinical problems of sleep. A new type of specialist has therefore appeared, during the past few years, who is primarily concerned with the diagnosis and treatment of sleep-related disorders.

It seems too early to tell whether or not we are now witnessing the emergence of a new clinical subspecialty concerned exclusively with problems of sleeping and waking. In any case, there are a growing number of sleep disorder centers in this country that attempt to use scientific knowledge of sleep and sleep-wake rhythms to diagnose and treat sleep disorders. (In 1972, there were three or four centers; now, there are many more, as listed in Table II.)

Primary Sleep Disorders

Primary sleep disorders are those conditions in which various abnormalities related to sleep are the cardinal (and often the only) sign or symptom of the problem from which the patient suffers (Williams et al, 1974). Presumably one or more of the neurophysiological/neurochemical mechanisms involved in regulating the wake/sleep/REM states are malfunctioning in these primary sleep disorders.

Narcolepsy:

Case History: *Mr. R., a 36-year-old widower, responded to an advertisement for research volunteers suffering excessive daytime sleepiness. It developed that Mr. R. had felt the need to nap two or three times per day since about the age of 17. He had been told "all his life" that he was lazy, and nobody had ever seen anything abnormal other than laziness in his frequent naps. He was a self-employed farmer, and his repeated 10- to 15-minute naps did not interfere to any great extent with his work.*

During the work-up, Mr. R. casually related another problem, not associated in his mind with his frequent naps. When his children made him very angry and he tried to discipline them, he often felt very weak in his knees and needed to sit down. On several occasions, when he had remained standing, he had collapsed on the floor from the "clumsiness that occasionally overcomes me when I am mad at my kids." Two years earlier he had sought psychotherapy for this problem, thinking that he must have "some hang-up" about disciplining his children. He was concerned about these spells of weakness because they seemed to increase in frequency, but his physician had not found anything wrong with his muscles and had concurred with his therapist that the clumsiness must stem from some psychological problem.

As expected, Mr. R. showed a long REM period immediately upon falling asleep in the lab. The rest of his lab sleep was uneventful, except that Mr. R.'s sleep showed many awakenings and excessive amounts of stage 1 sleep. Both Mr. R. and his physician were then advised that the patient had classical narcolepsy. Sleep attacks were treated with 5 mg methylphenidate b.i.d., and his "muscle weakness" (cataplexy) yielded to 25 mg imipramine t.i.d. This regimen produced excellent results in the beginning, but habituation to the imipramine occurred within about six months. Mr. R. was then advised to select periods of low work pressure when he could "afford" cataplectic attacks and to refrain from taking imipramine during those times. In

Table II **Some Referral Centers for Patients with Sleep Disorders***

Department of Psychiatry
Albany Medical College
Albany, NY 12208
ATTN: Vincenzo Castaldo, MD

Sleep Disorders Center
Stanford University
Medical Center
Stanford, CA 94305
ATTN: William C. Dement, MD

Sleep Disorders Clinic
c/o Dept. Neurosurgery
University of Arkansas
for Medical Sciences
Little Rock, AR 72201
ATTN: Herman Flanigan, MD

Dartmouth Sleep Clinic
Dartmouth Medical School
Hanover, NH 03755
ATTN: Peter Hauri, PhD

Boston State Hospital
591 Morton Street
Boston, MA 02124
ATTN: Ernest Hartmann, MD

Sleep Disorders Center
Department of Psychiatry
Baylor College of Medicine
Houston, TX 77030
ATTN:
Ismet Karacan, MD (Med) DSc

Sleep Evaluation Center
Western Psychiatric Institute
and Clinic
3811 O'Hara Street
Pittsburgh, PA 15261
ATTN: David J. Kupfer, MD

Sleep/Wakefulness Clinic
College of Medicine and
Dentistry
100 Bergen Street
Newark, NJ 07102
ATTN: James Minard, PhD

Department of Clinical
Neurophysiology
Clarke Institute of Psychiatry
Toronto, Canada
ATTN: Harvey Moldofsky, MD

Sleep Physiology Laboratory
VA Hospital (183A)
921 NE 13th Street
Oklahoma City, OK 73104
ATTN: William C. Orr, PhD

UAB Sleep Clinic
University of Alabama
Medical Center
University Station
Birmingham, AL 35294
ATTN: Vernon Pegrem, PhD

Sleep Clinic
Peter Bent Brigham Hospital
Boston, MA 02115
ATTN: Quentin R. Regestein, MD

Sleep Disorders Evaluation Center
Ohio State University
473 West 12th Avenue
Columbus, OH 43210
ATTN: Helmut S. Schmidt, MD

Sleep-Wake Disorders Unit
Department of Neurology
Montefiore Hospital and
Medical Center
111 East 210th Street
Bronx, NY 10467
ATTN: Elliot D. Weitzman, MD

University of Cincinnati
Sleep Disorders Center
Christian R. Holmes Hospital
Eden and Bethesda Avenue
Cincinnati, OH 45219
ATTN: Frank Zorick, MD

* This listing does not constitute an endorsement or recommendation of those named by the publisher.

this way, potency was restored during periods of higher need.

Classical narcolepsy is a highly specific sleep disorder. Usually, the patient suffers from two or more of the following four symptoms (the narcoleptic tetrad):

• *Short, but almost irresistible daytime sleep attacks.* This is the primary and most disabling symptom of classical narcolepsy. Many of the sleep episodes occur during totally inappropriate times (eg, while driving or eating). Usually, these attacks last 10 to 15 minutes, but if the patient is lying on a couch when the attack occurs, he may sleep for two or three hours.

In addition to the short sleep attacks, narcoleptic subjects usually suffer from general, chronic drowsiness. They very rarely feel fully alert, even after prolonged periods in bed.

• *Cataplexy.* This symptom can range from a very transient weakness in the knees to total paralysis of all voluntary muscles while the patient is fully conscious. Attacks of cataplexy are often triggered by emotions (laughing, crying, excitement). Usually such attacks last only seconds. A sudden loss of muscle tonus is observed and is either generalized or limited to specific muscle groups.

• *Sleep paralysis.* Either when falling asleep or upon waking, the patient feels that he cannot move any muscles except those controlling his eyes. However, respiration is not affected. Sleep paralysis usually lasts from a few seconds to several minutes and is very frightening. Patients can occasionally terminate the attacks by first vigorously moving the eyes, then the eyelids, then the facial muscles, and gradually spreading the areas of movement further and further away from the eyes. The paralysis is broken immediately if somebody touches the patient. Some cases of sleep paralysis without any of the other narcoleptic symptoms have been observed, and such cases do not invariably lead later to classic narcolepsy.

• *Hypnagogic hallucinations.* When falling asleep, narcoleptic patients occasionally experience vivid dreams while still fully conscious. However, both sleep paralysis and hypnagogic hallucinations are less frequent than the first two symptoms of narcolepsy, and are not necessary to diagnose the problem.

In the laboratory, a classic narcoleptic patient often shows a full-blown REM period occurring immediately at sleep onset (Rechtschaffen et al, 1963; see Figure 11). In addition, nocturnal sleep is usually fragmented and poor, showing more stage 1 and less stage 2 and delta sleep than is normal. REM sleep varies among narcoleptic subjects: some have excessive amounts of REM sleep, others have much less than normal. Surprisingly, despite their chronic daytime sleepiness, when narcoleptic subjects are asked to try to sleep as long as possible, they cannot sleep longer than do normal patients (Hishikawa et al, 1976).

In general, the disease seems best understood as a disturbance in the balance between the REM system, the sleep system, and the system maintaining wakefulness. Narcoleptic sleep attacks during the day frequently start with an intrusion of REM sleep into wakefulness. Cataplexy can be understood as the inappropriate triggering of that part of the REM system that is responsible for muscle atonia during REM. Sleep paralysis and hypnagogic hallucinations are manifestations of REM sleep that either occur at sleep onset (just before waking consciousness has ceased) or linger inappropriately after awakening from REM sleep.

There is a fair amount of confusion in the current literature concerning the term "narcolepsy." Following the discovery of Rechtschaffen et al (1963) that many narcoleptic subjects had sleep-onset REM periods, some authors tried to distinguish "classical" narcolepsy (involving REM sleep) from "idiopathic" narcolepsy (involving NREM mechanisms). However, it seems conceptually much clearer to limit the term narcolepsy to the sleep disorder discussed above

(involving sleep-onset REM, and at least one auxiliary symptom, such as cataplexy, in addition to the sleep attacks), while reserving the term hypersomnia for disorders involving excessive sleep attacks without the other aspects of narcolepsy.

Narcolepsy is not a rare disease. The incidence rate in the general U.S. population is estimated to be around .07% (Dement et al, 1973). It appears that this disorder has a genetic component in many cases (Leckman and Gershon, 1976), even though the disorder often does not manifest itself until the second decade of life. However, neurological insults in later life seem to lie at the roots of some other forms of narcolepsy (Hobson, 1975).

The excessive daytime sleepiness and the inappropriate napping of the narcoleptic patient can be treated with stimulants, mainly methylphenidate, pemolene, or dextroamphetamine, in that order of preference. However, there is some evidence that the chronic use of amphetamines occasionally aggravates the condition. Cataplexy, on the other hand, usually yields either to imipramine or to protriptyline. The dosage and administration of both the stimulants and the suppressants of cataplexy need to be titrated carefully. For example, there is no reason to give imipramine in cases in which cataplexy is not a serious problem (Zarcone, 1973; Guilleminault et al, 1974).

No drug currently available is entirely satisfactory. Habituation may be a serious problem, as is the occasional side effect of impotence in males treated with tricyclics. "Drug holidays" may be necessary to restore potency and to evaluate the possibility that the chronic use of amphetamines has started contributing to the problem rather than helping it.

Sleep apneas:

Case History: *Mr. K. was a 45-year-old, severely obese (313 lb) male with a primary complaint of excessive daytime sleepiness. This problem started when the patient was in his early 20s. Although Mr. K. seemed willing and intelligent, and had made good grades in college, he had never held any job for more than two or three weeks since then, and he had usually been fired for "laziness" (falling asleep five to ten times each day). He had had numerous medical work-ups during the past 20 years, involving most medical specialties from endocrinology to neurology and psychiatry. When he was first seen at the sleep clinic he had already spent more than $20,000 to find a cause for his sleepiness. However, except for gross obesity, essential hypertension, and some enlargement of the right ventricle, all of Mr. K.'s work-ups had been essentially normal.*

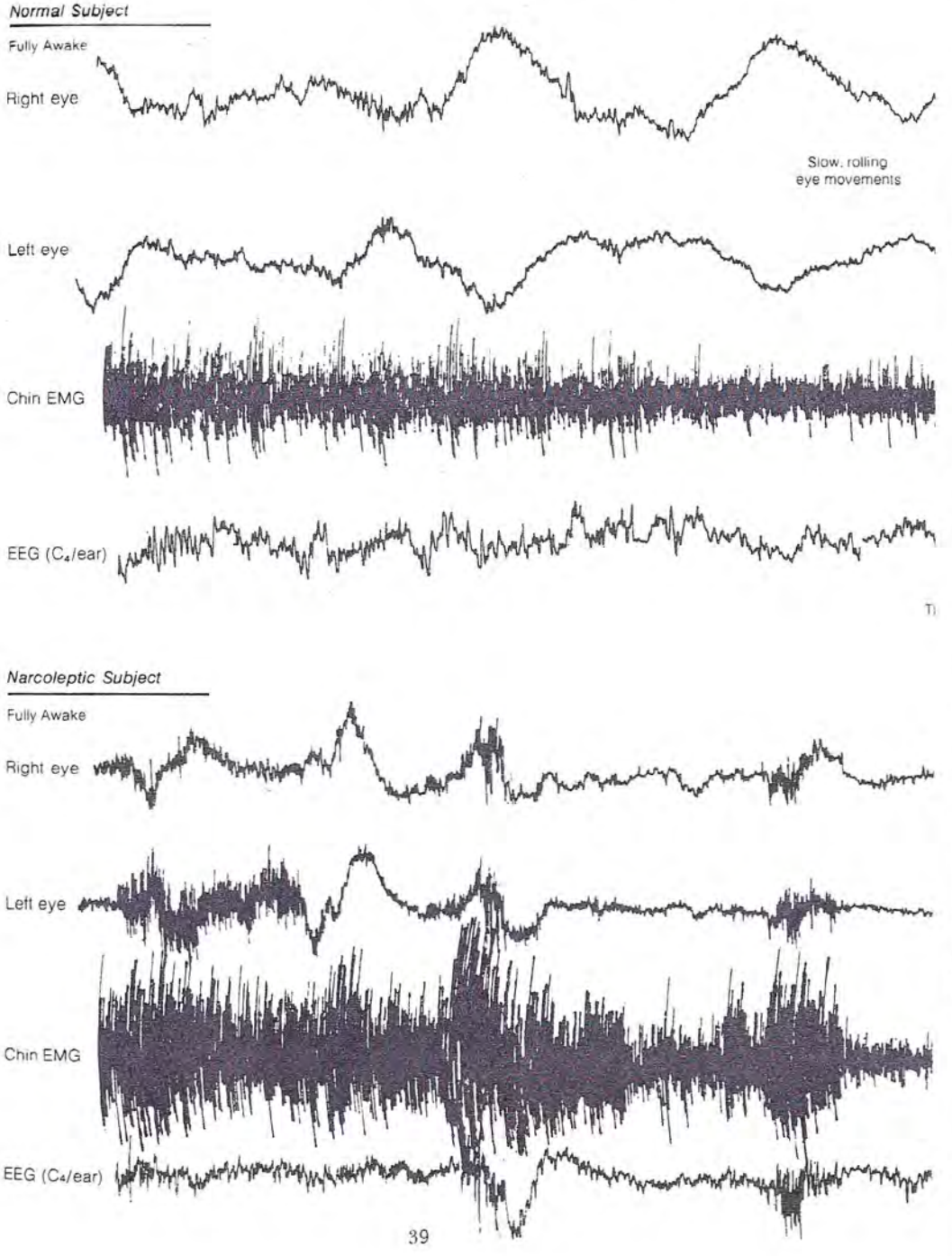
Although Mr. K. came from a relatively well-to-do family, he was destitute, when seen in the lab, because of his high medical bills and his inability to remain gainfully employed. His personal life was in ruins – two wives had left him (preferring divorce to living with a chronically sleeping, obese, heavy snorer), and he had been unable to maintain adequate social relationships with friends and peers because of his excessive sleepiness.

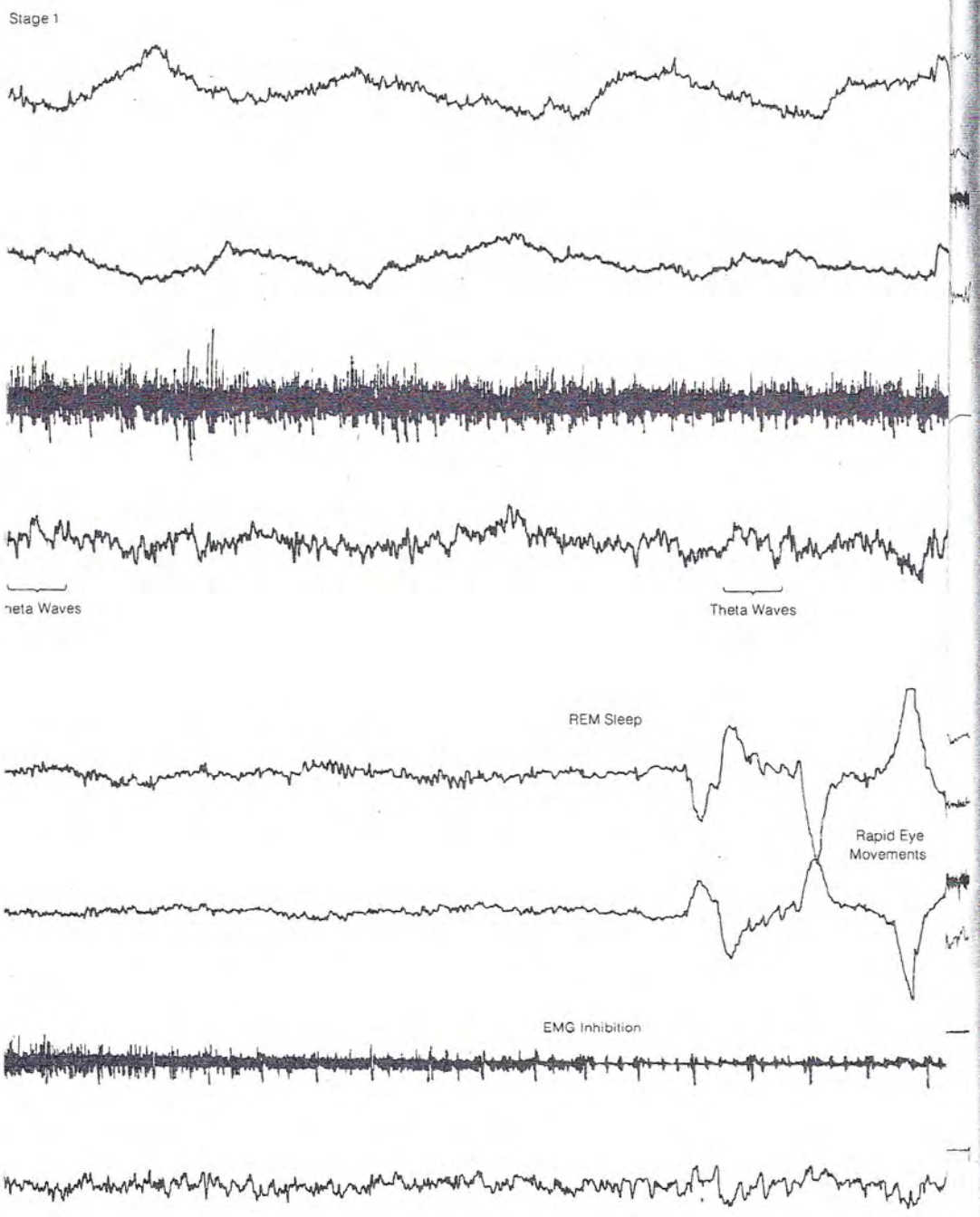
In the lab, Mr. K. was pleasant and polite. He fell asleep within five seconds after "lights out," but as soon as he fell asleep, his breathing stopped. He wakened 35 seconds later gasping for air. This cycle then repeated itself for the next ten hours of "sleep." As soon as Mr. K. showed signs of stage 1 sleep, he stopped breathing, then awakened 20 to 80 seconds later gasping for air. Throughout the night, he did not sleep for more than three uninterrupted minutes. By morning he had totaled 562 individual arousals from sleep, and more than 75% of his "sleep" time had been spent in sleep apneas. Visual observation of the patient indicated that during the periods of sleep apnea his chest heaved and he was straining, but no air passed through the upper airways (Figure 12).

In the morning, Mr. K. stated that he had had a "normal night of sleep" but that he was now more tired than he had been the previous night. He guessed he had awakened "five to eight times" during the night, and he was totally unaware of his very labored breathing, the heavy snoring, and the more than 500 sleep-apnea awakenings.

Mr. K. was told that he suffered from upper airway apneas and that the only effective treatment for this condition was a special form of permanent tracheostomy (to be closed during the day, but opened at night to allow breathing during sleep). Although the entire problem and its treatment were carefully explained to both Mr. K. and his private physician, both declined to consider a permanent tracheostomy for a "mere

Figure 11. **Sleep Onset in a Normal Subject and in a Narcoleptic Subject**
Note how the normal subject goes from awake to stage 1 (slow eye movements, gradual reduction of chin EMG, theta waves in the EEG) and then to stage 2 (K complexes and sleep spindle). The narcoleptic changes immediately from wakefulness to REM sleep (rapid eye movements, sudden decrease in chin EMG and sawtooth waves in the EEG).




Stage 1
heta Waves
Theta Waves
REM Sleep
Rapid Eye
Movements
EMG Inhibition

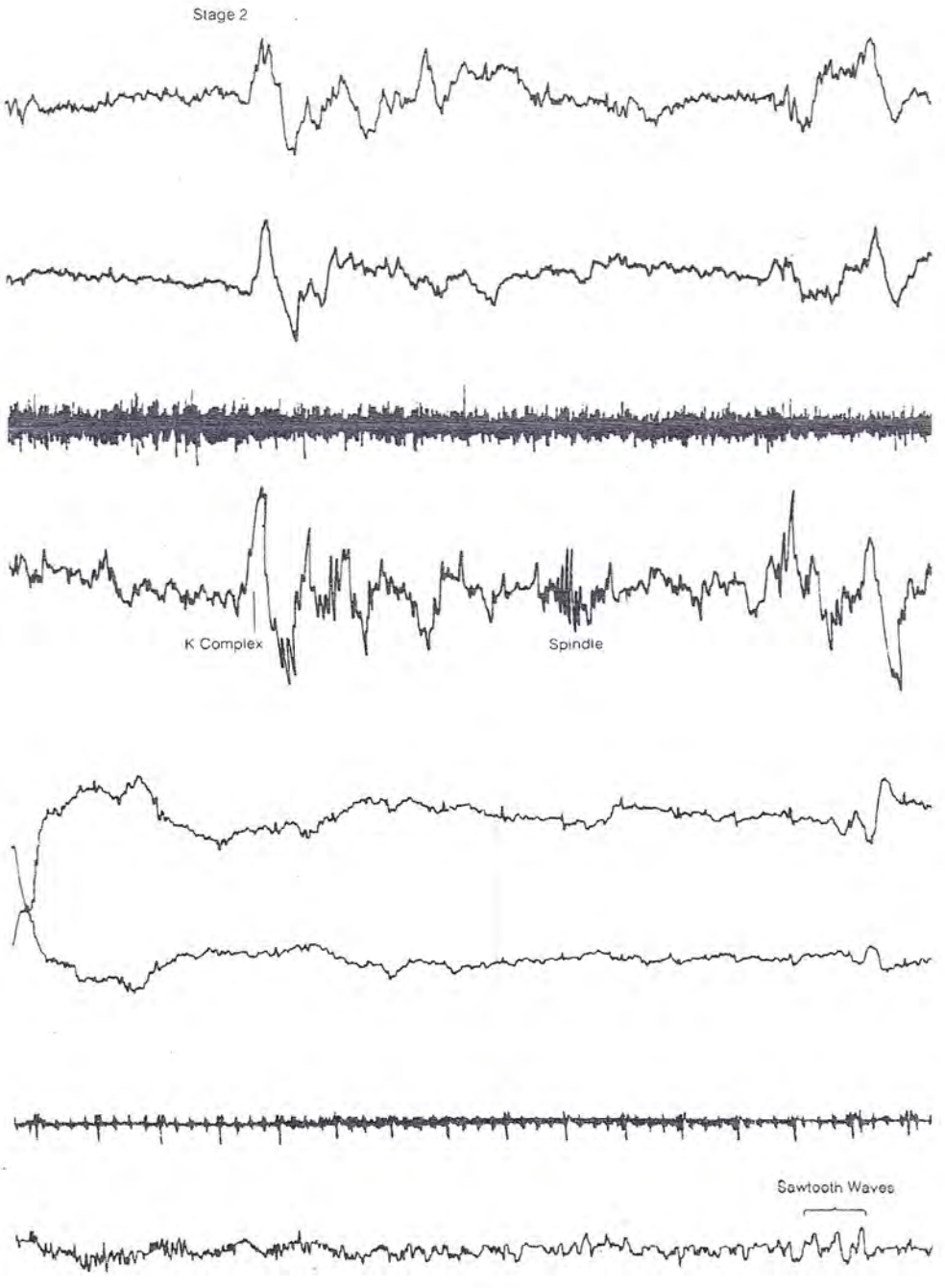

Stage 2
K Complex
Spindle
Sawtooth Waves

Figure 12. **Two Episodes of Sleep Apnea**

The patient is fully awake at point 1 (alpha waves in the EEG). At point 2 he is starting to fall asleep. Notice the slowing in his EEG (theta waves and the shallow breathing). At point 3 the patient is fully asleep. Notice the relaxation in the chin EMG, the high-amplitude theta waves, and the absence of breathing. At point 4 the patient wakes up and breathing restarts shortly thereafter.

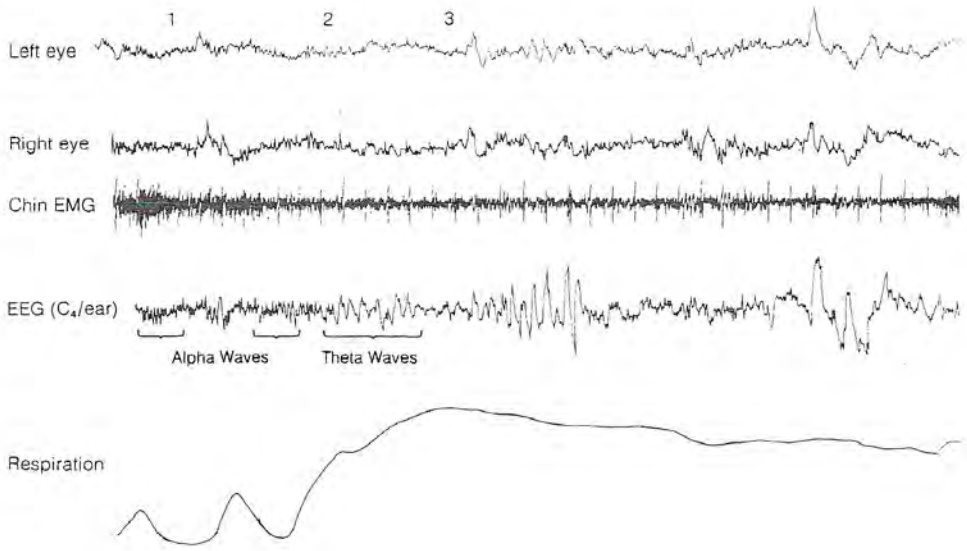
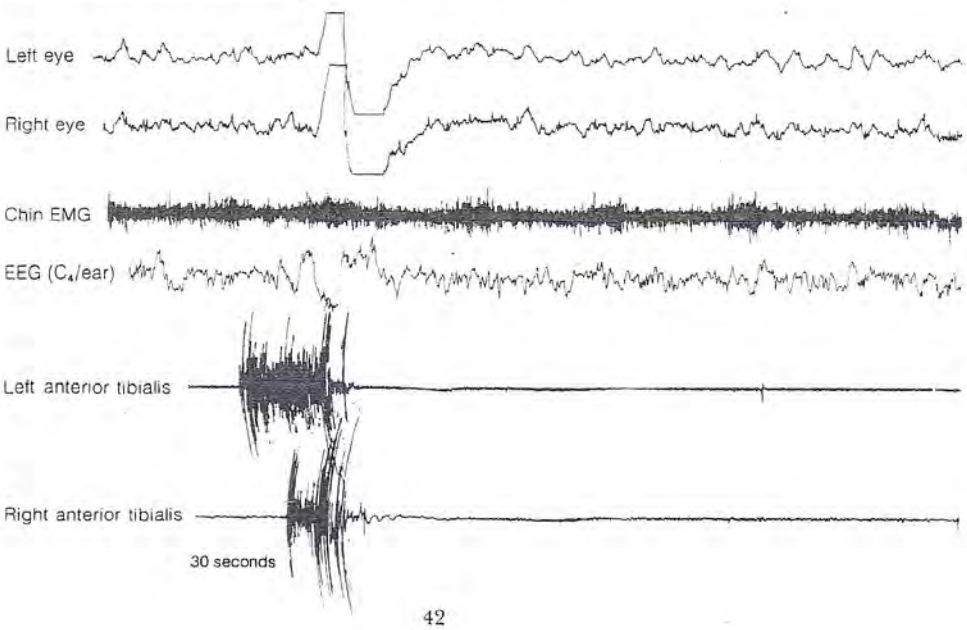
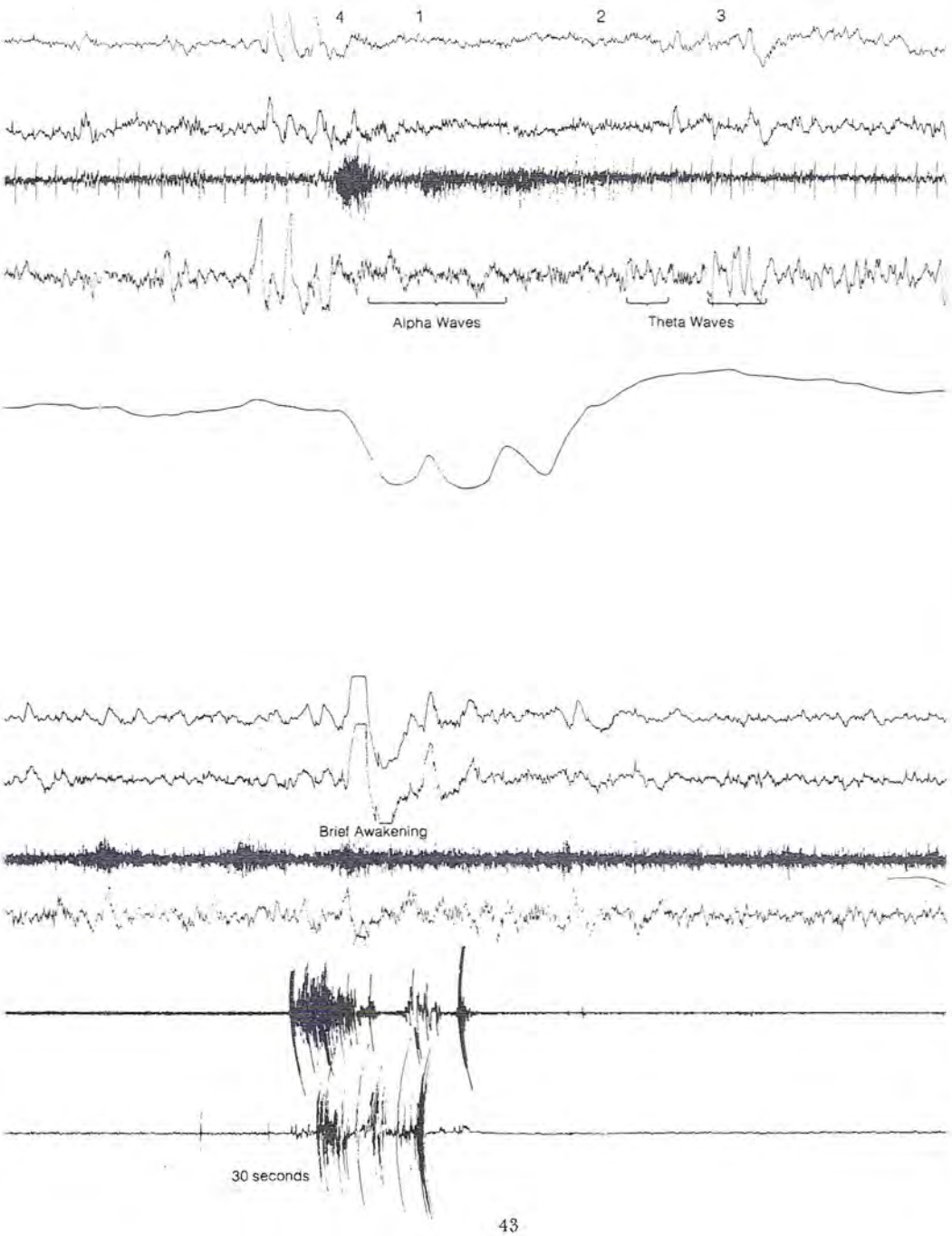
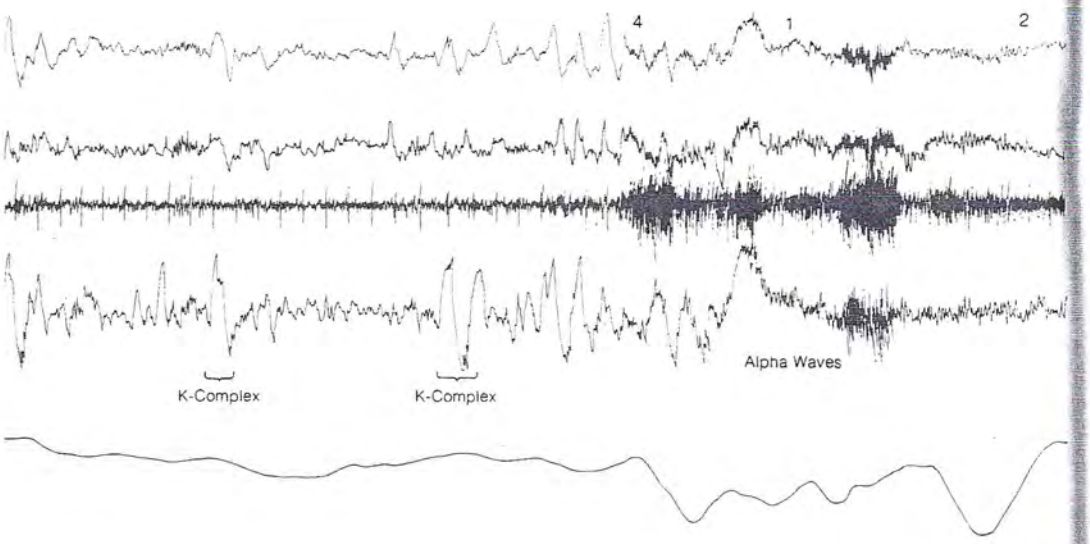


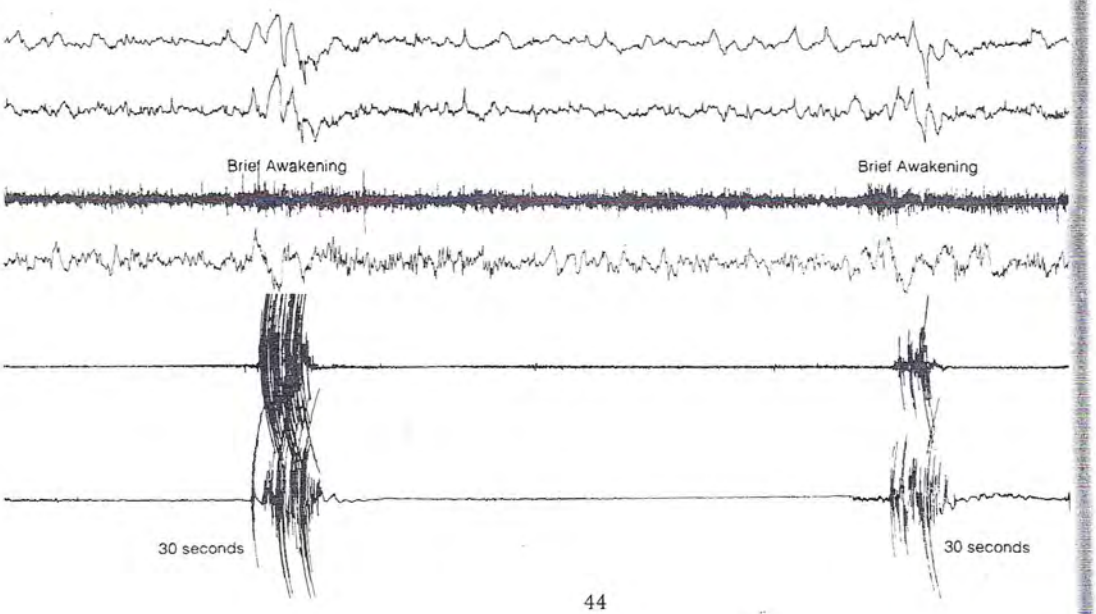
Figure 13. **Nocturnal Myoclonus**

A stereotyped twitch in the anterior tibialis muscle. This twitch occurs about every 20-40 seconds for periods of up to three hours. The EEG indicates stage 2 sleep. The second, third, and fourth twitches cause brief awakenings.




4
1
2
3
Alpha Waves
Theta Waves
Brief Awakening
30 seconds


4
1
2
K-Complex
K-Complex
Alpha Waves


Brief Awakening
Brief Awakening
30 seconds
30 seconds

sleep problem." Rather, they hoped that weight loss would cure the difficulty.

In the four months that followed, Mr. K. became an alcoholic, and seven months later he was caught in an attempt to steal liquor. Eleven months later, he died "of unknown causes" in a state prison.

Sleep apneas have been described only recently (Gastaut et al, 1965). With this disease, patients literally stop breathing whenever they fall asleep. Sleep then lightens to the point where breathing resumes, after 10 to 180 seconds, or the patient may wake up. Patients are never aware of their sleep apneas, even when they are awakened literally hundreds of times each night. Episodes of repeated sleep apneas may last from a few minutes to a few hours, alternating with apparently normal sleep, or they may take up practically the entire night of a patient (as they did in the case of Mr. K.). The Stanford Sleep Disorder Clinic has collected a large series of sleep apnea patients to study this disease in detail, and the following discussion will therefore follow their conceptualization of this problem.

Three types of sleep apneas are typically observed:

Central apneas are defined by an absence of any respiratory effort. Although the upper airway seems open, no air flows through it. The diaphragm stops moving.

Upper airway apneas involve a collapse of the upper airway. Respiratory effort persists, but there is no air exchange.

Mixed apneas are a combination of the two types. The apnea starts with a lack of respiratory effort. The upper airway then seems to collapse, and a few seconds later ineffective respiratory efforts resume (see Figure 14).

Additional medical complications are usually associated with severe sleep apneas. Some complications appear to be secondary to the extreme respiratory effort of trying to breathe against the upper airway obstruction; others seem to be the result of chronic hypoxemia associated with apnea, or they may be related to the same CNS dysfunction that initially caused the apneas. Hemodynamic complications are also prevalent (Guilleminault et al, 1975). During sleep, most apnea patients show marked elevation of blood pressure, and they gradually may develop essential hypertension during wakefulness as well. Cor pulmonale seems to be a possible end result. Other complications involve the heart. According to Guilleminault et al (1976), a number of severe arrhythmias are commonly observed in association with sleep apneas, such as severe sinus arrhythmia, second-degree heart block, ventricular tachycardia, and sudden asystoles (lasting up to six seconds). One might wonder how many patients with conditions such as essential hypertension or cardiac problems suffer from an undiagnosed sleep apnea syndrome.

The following three clinical subtypes of the sleep apnea syndrome are currently distinguished (Guilleminault et al, 1976):

a. Hypersomnia-hypoventilation syndrome – In this syndrome, upper airway apneas are predominant. Patients complain mainly about excessive daytime sleepiness, no matter how long they sleep at night. They take frequent, involuntary, and unrefreshing naps during the day, often at totally inappropriate times. They show extreme grogginess when awakening in the morning, occasionally associated with "sleep drunkenness" or severe headaches. Although some patients suffering from this syndrome are overweight and might be called "pickwickian," others are of normal weight. Severe depression is a frequently encountered corollary of this syndrome.

b. Insomnia-sleep apnea syndrome – Central sleep apneas seem to predominate in this syndrome, possibly because blood-gas changes are not as extreme in this condition as they are in the hypersomnia-hypoventilation syndrome. Patients are often aware that they awaken frequently during the night, but they

rarely suspect that they are having sleep apneas. Most have taken sleeping pills, which invariably make matters worse. While these people are excessively tired during the day, they are rarely forced into taking inappropriate naps.

c. Sleep apnea in children – Guilleminault et al (1976) have reported on eight children who suffered from severe, upper airway sleep apneas. According to these authors, excessive daytime sleepiness, decreased school performance, abnormal daytime behavior, recent enuresis, morning headaches, abnormal weight gain, and the progressive development of hypertension all suggest the possibility of a sleep apnea syndrome if they are associated with loud and irregular snoring. It seems likely that "Ondine's Curse" (Severinghaus et al, 1962) is a special variant of central sleep apnea. Much has been said about a possible relationship between the infant sudden death syndrome and sleep apneas (Weitzman and Graziani, 1974). Although such a relationship seems quite likely (Steinschneider, 1972), the final data are not yet in.

The incidence of sleep apneas in the general population has not been determined. On the basis of preliminary data, Dement (personal communication) estimates that there are about 50,000 serious sleep apnea patients in this country. However, should some disorders of currently unknown etiology (eg, specific nocturnal arrhythmias) be found to be rooted in this syndrome, the incidence of sleep apnea might be considerably higher.

Practically all sleep apnea patients are heavy snorers. Thus, snoring in a patient who complains either of excessive daytime sleepiness or of fragmented sleep is usually the most obvious sign suggesting a sleep apnea syndrome. Indeed, Lugaresi et al (1975) found sleep apneas in a great number of snorers who did not complain of any other sleep problem. When the apneic episodes in these heavy, noncomplaining snorers were repetitive, alveolar hypoventilation developed and systolic arterial pressure rose and remained above the physiological level found during wakefulness.

A presumptive diagnosis of sleep apnea can be made in the physician's office if there is either heavy snoring or abnormal behavior during sleep, and if the bed partner or a parent can document the apneic pauses. Alternatively, the patient might be observed during a nap in the physician's office, although patients with less-severe apneas are occasionally able to sleep for two or three hours with regular breathing. This makes repeated observations necessary.

If a presumptive diagnosis of sleep apnea syndrome is made, a work-up in a sleep center equipped to measure such things as blood gases and pulmonary function is mandatory. Although such work-ups are relatively expensive, costs are minimal when compared to the extensive and often unrevealing medical work-ups most of these patients have undergone in the past. In addition, sleep apneas severely damage one's health, and the risk of sudden death during sleep is high in undiagnosed sleep apnea patients.

It appears that upper airway sleep apneas can occasionally be based on purely mechanical abnormalities such as excessive fat deposits, abnormally thick soft palate, micrognathia, and retrognathia. Other sleep apneas are secondary to a variety of known neurological conditions (Guilleminault et al, 1975). Most often, however, upper airway apneas seem related to a sudden, dramatic, and apparently reflexive collapse of the upper airways, a finding that points to a specific CNS dysfunction. Similarly, in central apneas, the sudden cessation of any respiratory effort suggests a CNS dysfunction, possibly some lesion or abnormality in the lower respiratory centers.

If upper airway apneas are predominant, potential obstructions in the airway might be investigated first. If the patient is obese, massive weight loss has occa-

sionally alleviated the syndrome (Fisher et al, 1976). In severe cases, however, especially when conditions such as severe cardiac arrhythmias or cor pulmonale threaten the patient's life, a special type of permanent tracheostomy seems to be the treatment of choice and gives dramatic, immediate relief. The opening of the tracheostomy is closed during the day for normal breathing, but opened before sleep. Excessive daytime sleepiness disappears within a few days following tracheostomy, cardiac arrhythmias gradually improve, and blood pressure returns to normal within two or three months. However, the medical problems reappear almost immediately if the tracheostomy is plugged during sleep.

No adequate treatment for central apneas has been found. Imipraminic drugs, especially the investigational drug clomipramine, have helped some patients, but there are problems of adaptation and potential side effects. Some children have found some relief with chronic phrenic nerve stimulators, but the results have not been impressive.

Primary insomnia, primary hypersomnia

Case History: *Miss O. was a 29-year-old secretary who had apparently been an insomniac since birth. In fact, her hospital chart bore a note from the newborn nursery commenting on the remarkable lack of sleep in this baby. Miss O.'s earliest memories were of many preschool nights when she would sit quietly by her window for hours while her parents slept, watching the deserted street below, exhausted, but unable to sleep. She remembered grade school only as a constant struggle, always tired, rarely sleeping well. Although intelligent, she barely scraped by in school.*

Starting with high school, Miss O. noticed that she could not take cola drinks or coffee after lunch, because it would keep her up "all night." Most available hypnotics and stimulants had

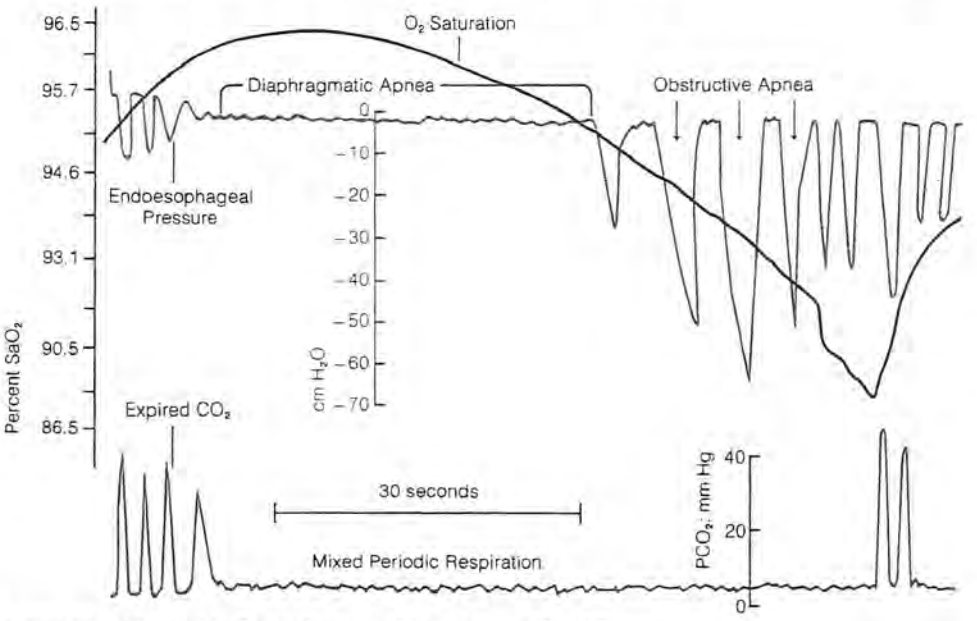


Figure 14. **Polygraphic Recording of a Sleep Apnea, Mixed Type**

This 49-year-old male patient is breathing normally during the first 5 seconds. He then falls asleep (EEG not shown). A central apnea of about 40 seconds' duration ensues (see endoesophageal pressure). On the right side of the recording, obstructed attempts to breathe result in wide fluctuations of endoesophageal pressure, until breathing resumes. CO_2 is expired, and O_2 saturation increases again.

Courtesy of Guilleminault et al, *West J Med* 123:7-16, July 1975.

been tried at some time. Almost all had helped for a few days or weeks, but none had brought any lasting relief, either from her insomnia or from her chronic, excessive daytime fatigue. Extended evaluations at some of this nation's leading medical centers were negative, except for comments on Miss O.'s general lack of stamina and emaciated condition. Recent psychological testing showed some excessive concern about somatic function and a generalized feeling of chronic malaise, but no noteworthy psychopathology beyond that. Specifically, no clear signs of depression could be found.

In the lab, Miss O. slept very fitfully for three or four hours each night, and she was very easily aroused by even the slightest click on the intercom. When aroused, she was immediately and fully awake. Most remarkable were her very "weak" sleep patterns: almost no delta waves, and only very poorly defined, irregular sleep spindles that occurred once every five to eight minutes or so. However, no respiratory or muscular abnormalities could be found during sleep, and the alternations between REM and NREM sleep seemed normal.

Miss O. was given sensorimotor rhythm (SMR) training, a procedure designed to increase activity in the 12 to 15 cps EEG range. After about 40 hourly sessions, she seemed able to produce somewhat more SMR. Concomitantly, sleep at home improved slightly, and Miss O. was delighted. Postbiofeedback evaluations showed about 40 minutes' more sleep per night after biofeedback, fewer awakenings, and, possibly, some better-formed sleep spindles. However, sleep still was extremely poor after biofeedback, and Miss O.'s daytime fatigue improved only marginally.

Case History: *Mrs. M. was a 52-year-old nurse who sought help from the lab for her excessive daytime sleepiness. She had suffered from this problem since her teenage years, and she described vividly how in high school she would accept any active dates (such as horseback riding and dancing) but would have to avoid quiet dates (such as movies) because she would fall asleep within a few minutes after sitting down. Throughout her life she had slept from 12 to 16 hours per day. When her children were little, she would lie down on the floor to feed them, for fear of dropping them should she fall asleep during nursing. Struggling against her sleepiness, she was able to take her children bowling, swimming, or roller skating, but she could rarely play cards with them or supervise their homework, because she would fall asleep as soon as she sat down. She barely managed to finish nursing school, and, from then on, always requested general duty work in overcrowded wards because this kept her constantly busy throughout her eight-hour work shift. Occasionally assigned lighter duty, she would often fall asleep on the job, and this had led to her being fired on three different occasions.*

On the first laboratory night, Mrs. M. fell asleep within two minutes after "lights out." She then slept for eleven consecutive hours without awakening, almost without body movements. In the morning, she labeled this sleep "somewhat shorter than average." On another lab night, when she was worried about a sick child at home, she slept only nine hours and felt excessively tired in the morning, "like an insomniac after a sleepless night." No sleep apneas, nocturnal myoclonus, or nocturnal seizures could be recorded. Sleep was essentially normal except for its unusual length.

Mrs. M. was placed on low doses of amphetamines. Follow-up data one and two years later revealed that Mrs. M. now slept about eight hours per night and felt quite awake during the day. The patient and her family were extremely grateful that she was now "an awake mother," and the required amphetamine dosage has now been stable for at least two years. No addiction developed; when Mrs. M. was abruptly withdrawn from amphetamines on two occasions, there were no serious withdrawal effects except that excessive daytime sleepiness reemerged.

On purely theoretical grounds, one would suspect that there must be patients in whom the complex neurophysiological/neurochemical balance between sleep and wakefulness is maladjusted in one way or another. Either the sleep or the wake system might be excessively weak or excessively strong purely because of the normal range of variability in any biological system or because one or both of the systems might be damaged (either anatomically or biochemically). The preceding two cases appear to illustrate such problems. Miss O. shows primary insomnia, whereas Mrs. M. apparently suffers from primary hypersomnia.

Although most insomniacs show psychiatric, medical, or behavioral etiology, a minority (about 10% to 15% at the Dartmouth Sleep Clinic) do seem to

show demonstrably poor sleep in the laboratory in the absence of significant medical, psychiatric, or behavioral psychopathology. In some of these cases, we probably just have not learned where to look for the pathology. However, it seems likely that others in this category are showing primary insomnia. In addition to their shortened and fragmented sleep, such patients often show a number of atypical wave forms during sleep, such as a lack of sleep spindling or frequent intrusions of alpha bursts into sleep (Phillips et al, 1975).

No direct treatment is currently available for primary insomnia. The recently developed SMR biofeedback training, however, does hold some initial promise (Feinstein et al, Jordan et al, 1976). In this training, one attempts to strengthen 14 cps activity over the sensorimotor cortex by biofeedback. The hope is that the strengthening of this rhythm will improve sleep spindling during the night, as it apparently does in cats (Sterman et al, 1970). Hopefully, this will, in some as yet unexplained action, strengthen the sleep system, deepen sleep, and avoid excessive arousals.

Similar to insomnia, most cases of excessive sleep show medical or psychiatric etiology, or they can be traced to side effects of chronic drug usage. Others, however, do seem to be primary, or they might be based on very subtle abnormalities such as biochemical disturbances that are still undiagnosable with current technology. Indeed, some patients at the Stanford Sleep Disorder Center who showed excessive daytime sleepiness were found to have elevated levels of 5-hydroxyindoleacetic acid (5-HIAA) in their cerebral spinal fluid. This is an interesting result, because the production of 5-HIAA in the brain is thought to be proportional to serotonergic neuronal activity (Aghajanian et al, 1967). It is currently unknown how widespread excessive CSF 5-HIAA is in hypersomnia, because most laboratories do not have the facilities to make the determination.

The treatment of primary insomnia and primary hypersomnia is still very much in its infancy. Amphetamines are still the most widely used treatment for primary hypersomnia. Whether the inherent risks in such treatment are worth the benefit depends on the seriousness of the hypersomnia. Fortunately, the danger of becoming seriously addicted to amphetamines seems to be somewhat lower for hypersomniacs than it is for people who take amphetamines for weight reduction or for "kicks." Hyper-

Table III **Three Questions to Ask when Patients Complain of Excessive Daytime Sleepiness (EDS)**

Have you ever had any unusual muscular experiences?	*Reason:* If patients report either cataplexy or weaknesses (especially knees) during excitement, suspect narcolepsy. This question helps to diagnose about 65% of all patients with EDS.
Do you snore?	*Reason:* If "yes," especially if snoring is irregular, investigate sleep apneas. This question helps to diagnose an additional 10% to 15% of patients with EDS.
What medications have you used chronically during the past few months or years?	*Reason:* Chronic use of stimulants, hypnotics, and some other drugs can result in EDS. This question helps to diagnose an additional 5% to 10% of patients with EDS.

According to Dr. W. Dement, Symposium on Sleep Pathology, Boston, June 7, 1976.

somniacs do not usually need ever-increasing dosages of amphetamines to obtain the same effects, and they do not show excessive withdrawal effects when the amphetamines are taken away. Other medications that might be tried in selected cases are the stimulating antidepressants such as protriptyline (Schmidt et al, 1976).

Nocturnal myoclonus and restless legs syndrome:

Case History: *Mr. M. was a 35-year-old electrician sent to the sleep clinic with a diagnosis of "inadequate personality" after two years of psychiatric treatment. His main complaints were that he felt "washed up" in the morning and that, occasionally, he had difficulties falling asleep because his "legs were nervous." By this he meant that he felt uncomfortable but not painful sensations creeping deep inside his calf. The urge to move his legs then became so strong that he had to get up and walk for 10 to 20 minutes before he could try to sleep again. Occasionally, this problem continued until 4 AM or 5 AM. Detailed waking neurological evaluations and laboratory work-ups had been negative, and a sleep study was performed.*

Each night in the lab, Mr. M. got up once or twice after "lights out" to "walk off his legs." When he finally fell asleep, episodes of nocturnal myoclonus were recorded. For periods of up to one hour, Mr. M.'s legs jerked violently every 30 to 40 seconds, and almost each time this happened, the EEG showed a 5- to 15-second arousal (see Figure 13, page 42). In the morning Mr. M. was not aware that he had been aroused 300 to 400 times by nocturnal myoclonus, and he knew only that he felt tired, as if he "had not slept very much."

Mr. M. was placed first on 20 mg, later on 40 mg, diazepam h.s. He reported having fewer problems with his "nervous legs" before the onset of sleep and experiencing a more refreshing sleep. Objectively, when recording in the lab three months later, he did not get up after "lights out" to "walk off his legs." The episodes of nocturnal myoclonus were about the same length, but each discharge seemed much weaker than before. Instead of 300 to 400 times each night, he now was aroused only 20 to 40 times, and he felt better during the day.

A diagnosis of nocturnal myoclonus is made when highly stereotyped leg twitches repeat themselves every 20 to 40 seconds. Usually, myoclonus is recorded from both the right and the left anterior tibialis muscle. Some myocloni are bilateral; others are unilateral. Episodes of such myocloni generally last from five minutes to two hours, and they alternate with periods of normal sleep. Usually, periodic leg movements are not seen during wakefulness, and the myoclonic leg twitches are never associated with epileptic discharges in the EEG.

Nocturnal myoclonus should not be confused with the occasional "hypnic jerks" or startle movements, which awaken most of us occasionally just as we fall asleep. Although bed partners usually can describe nocturnal myoclonus in detail, patients themselves are rarely aware of their leg movements and typically complain of their fragmented and unrefreshing sleep.

Neither the incidence nor the etiology of nocturnal myoclonus has been resolved. Guilleminault et al (1973) postulate that phasic events, occurring every 20 to 40 seconds, seem improperly regulated in nocturnal myoclonus, and that these events then lead to "postexcitatory rebounds." Similarly, Lugaresi et al (1972) relate nocturnal myoclonus to a number of vegetative and somatic phenomena that tend to oscillate periodically every 20 to 30 seconds (such as systematic arterial pressure and peripheral motor neuron excitability).

At present, there is no adequate treatment for nocturnal myoclonus. Diazepam seems to mask the problem. The myocloni are less violent after diazepam and, therefore, seem to arouse the sleeper less often. Methysergide maleate has been proposed as a treatment of nocturnal myoclonus in some cases, and some promising results have been reported with γ-aminobutyric acid analogs (Guilleminault et al, 1975).

Many patients with nocturnal myoclonus also complain of the restless legs syndrome (Ekbom, 1945). This syndrome occurs mainly before one falls asleep and at other times of inactivity while

awake. Uncomfortable, but not really painful, sensations occur deep inside the calf (occasionally also in thighs and feet), sensations that can only be ameliorated by moving the legs vigorously.

Some cases of the restless legs syndrome have been linked to motor neuron disease (Frankel et al, 1974). Others seem to have been secondary to deficiencies in iron, calcium, or vitamin E. Chronic uremia is often associated with the restless legs syndrome, as well as with nocturnal myoclonus. However, in the majority of cases no cause for restless legs can be found. Boghen and Peyronnard (1976) suggest that about one third of all cases involving restless legs have a familial incidence and that this syndrome may be transmitted as an autosomal dominant trait.

Treatment of the restless legs syndrome obviously involves the removal of any deficiency that may be found. If none can be found, diazepam or oxycodone has been recommended, and carbamazepine occasionally seems quite effective in serious cases. Also, it appears that an adequate exercise program, coupled with some form of deep muscle relaxation (eg, meditation) during other times of the day, has brought relief to some restless legs syndrome sufferers.

Nonrestorative sleep:

Case History: *Mr. A., a 32-year-old executive, felt that his sleep was relatively adequate in length, but very shallow. He also awakened each morning with excessive fatigue and muscle stiffness, as if he suffered from rheumatoid arthritis. However, a work-up for arthritis proved negative. The muscle aching and stiffness usually disappeared gradually during the morning, but Mr. A. felt somewhat tired all day, as if he had not slept enough.*

When studied in the laboratory, Mr. A. slept a total of about seven and one half hours each night. However, throughout NREM sleep, high amplitude, slow alpha waves continuously intruded into his sleep EEG, making it very difficult to score the record.

Mr. A. was put on a regimen of 50 mg of amitriptyline h.s. Subjectively, he then slept much more soundly, and the morning stiffness disappeared within a few days. A nine-month follow-up in the laboratory showed fewer alpha waves intruding into his sleep, and somewhat longer sleep. When the amitriptyline was withdrawn, alpha returned, and so did his muscular stiffness.

Chronic alpha intrusion into sleep (Hauri and Hawkins, 1973) seems to be related to a general feeling of somatic malaise and fatigue in the face of relatively adequate amounts of sleep. Recently, Moldofsky et al (1975) have found that many patients with fibrositis also show chronic alpha intrusion into NREM sleep. Fibrositis is a poorly defined symptom complex that consists of acute or chronic aching, soreness, and stiffness in the absence of objective abnormalities. Moldofsky et al (1975) suggest that the observed alpha intrusion into the sleep EEG could be related to a "nonrestorative sleep syndrome" associated with fibrositis. However, care seems indicated; the chronic use of hypnotics and/or stimulants may also result in an excessive intrusion of alpha waves into sleep, without relating to fibrositis.

No treatment for nonrestorative sleep has yet been validated, and not everyone who suffers from excessive fatigue in the morning shows alpha intrusion into sleep. According to Moldofsky (personal communication), neither L-tryptophan nor thorazine is effective in abolishing alpha intrusion or excessive muscular stiffness in the morning. Occasional patients have benefited from low doses of sedating antidepressants, but to date the numbers are far too small to warrant definite conclusions.

"Pseudoinsomnia"

Many patients claim that they sleep little or not at all, even though observers (ie, bed partners, nurses in hospitals) find them to be soundly asleep throughout the night. Such patients are usually suspected of malingering, supposedly to gain sympathy. Undoubtedly, malingering is a possible explanation in some patients, and many insomniacs exaggerate their sleep problems. Nevertheless,

subtle EEG and physiological abnormalities have been found in a number of patients with pseudoinsomnia, even in the absence of gross sleep pathology. Some patients, for example, show hundreds of "mini-arousals" throughout the night, each arousal lasting for only a few seconds. Others seem to be thinking very intensely throughout the night, even when objectively they are in NREM sleep, or they may show excessive amounts of stage 1 sleep. Thus, pseudoinsomnia should be diagnosed conservatively, and the daytime waking behavior of such patients should show personality traits commensurate with a diagnosis of pseudoinsomnia. If not, the patient might well be given the benefit of the doubt. There are many sleep disorders recognized today that were unknown just ten years ago, and others will undoubtedly be discovered in the future.

Miscellaneous Primary Sleep Problems

Periodic hypersomnia: Some patients sleep excessively for days, weeks, or even months, alternating with periods of normal or even excessively short sleep. In most cases, such persons are found to be suffering from recurrent depressive episodes or from manic-depressive psychosis. In other patients, periodic hypersomnia points to disorders such as endocrine problems or difficulties in the regulation of hormones, or a relationship with menstruation (Billiard and Passouant, 1973; Billiard et al, 1975).

Kleine-Levin syndrome: This disorder is much more rare than its extensive coverage in the literature indicates. In the Kleine-Levin syndrome, periods of excessive sleepiness are coupled with increased appetites for food and sex. Periods of hypersomnia alternate with periods of short sleep and decreased appetites. This condition occurs mainly in young males (Critchley, 1962). The increased sleep is often disturbed by excessive theta waves at sleep onset and by lack of sleep spindles. No treatment has yet been developed, but some patients seem to outgrow the disease with age.

Neutral state syndrome: This syndrome involves excessive daytime sleepiness, usually associated with automatic behavior, blackouts, and a general lack of attention. Polygraphic recordings show hundreds of "microsleeps" (lasting 10 to 60 seconds each) during the day, coupled with hundreds of "microwakes" during the night. It appears that neither sleep nor wakefulness can be maintained over adequate lengths of time. There is no known treatment.

REM-interruption insomnia: REM sleep is very light sleep in humans, and frequent awakenings from REM are commonplace. It now appears that some patients awaken chronically just after REM onset or even slightly before REM is due to begin, and then have difficulties returning to sleep. This condition is suspected when a patient habitually awakens 1½ to 2 hours after sleep onset.

According to Greenberg (1967), REM-interruption insomnia might be explained as a consequence of experiencing repeated, extremely unpleasant dream episodes. Some patients showing this problem might conceivably have learned to avoid REM sleep because of these poor dreams. Greenberg has found that chlordiazepoxide (50 mg to 125 mg) was reasonably successful in inhibiting REM-interruption insomnia, whereas diazepam (5 mg to 20 mg) had less effect. Thus, the former medication might be tried if a patient complains of awakening every 1½ to 2 hours per night. However, the unpleasant dreams, which the REM-interruption insomnia had presumably attempted to avoid, might then return, and psychotherapy might then become necessary.

Painful nocturnal erections: All males regularly have penile erections during each REM episode, unless the dream content is extremely anxiety-provoking (Karacan, 1975; Fisher, 1965). Occasionally, these REM erections are painful, awakening the sleeper. No direct treatment is available, but the patient should be reassured that REM erections are

normal and that they do not indicate any abnormality in his sexual apparatus. (Parenthetically, Karacan et al (1976), as well as others, now measure these nocturnal erections during REM sleep in patients complaining of impotence, in an attempt to distinguish organic from functional cases of impotence.)

Sleep Disorders Secondary to Medical Problems

Neurological disorders, including epilepsy: Obviously, any disorder that affects the balance between the neurological wake/sleep/REM systems (see page 13) will manifest itself in sleep disturbances. Disorders that have been documented as causing sleep abnormalities (either directly or through a change in stimulus-input levels to the wake/sleep/REM system) include damage to the spinal cord; subcortical and cortical lesions; brain traumas and infections; and degenerative conditions. A detailed discussion of these disorders is beyond the scope of this review; the reader is referred to Williams et al (1974) for many references.

It is well known that some forms of epilepsy are aggravated by sleep. Indeed, in some relatively rare cases, epilepsy may manifest itself exclusively during sleep. For example, in a series of 645 epileptic patients studied by Gibberd and Bateson (1974), 38 had seizures exclusively during sleep. This is true for children as well as adults. Patients might then complain about poor sleep, or occasionally they may wet their beds without anyone being aware of their nocturnal seizures. Therefore, it would seem wise to record the sleep EEGs of patients who (1) have sporadic nocturnal enuresis, (2) have nightmares without known precipitants, (3) awaken with a bitten tongue or cheek, (4) have unexplained blood on their pillow, (5) awaken with a very disordered bed, (6) show postictal phenomena, (7) or are described by witnesses as possibly having epileptic attacks during sleep (Boller et al, 1975). According to Livingston and Pauli (1974), phenobarbital should be tried first in such cases of nocturnal epilepsy but, in their view, d-amphetamine sulphate most effectively treats sleep seizures. Others have suggested phenytoin or carbamazepine for the treatment of nocturnal epilepsy.

Sleep deprivation, as well as sleep itself, is used as an activating procedure in some EEG labs, especially in Europe (Scollo-Lavizzari et al, 1975). According to Gibbs and Gibbs (1971), among patients who suffer from clinically diagnosed epilepsy, approximately one third show seizure activity in the awake EEG recording, but in sleep and drowsiness recordings, 80% to 90% show seizure activity.

A problem with using sleep to activate seizures is that it may be "too effective," showing "abnormal" waves even in clinically sound humans. Several spike phenomena (eg, the 12 and 6 cps positive spikes, the small sharp spikes, and the 6 cps spike and wave complexes) appear in the EEG almost exclusively during drowsiness or sleep, but are not, in themselves, sufficiently reliable to diagnose a seizure disorder. According to Passouant et al (1975), the occurrence of a generalized seizure may be facilitated by NREM sleep, whereas petit mal paroxysms and the brief, generalized discharges of certain myoclonic epilepsies are activated by REM sleep. However, this finding has not been replicated to date.

Thyroid dysfunction: Abnormal thyroid function clearly affects sleep. Hyperthyroidism causes fragmented, short sleep with excessive amounts of delta sleep. The recovery of sleep after successful treatment of thyrotoxicosis is very gradual, and it may take up to a year before normal sleep patterns are achieved (Dunleavy et al, 1974). Hypothyroidism, on the other hand, seems to cause excessive sleepiness and a lack of delta. A return to euthyroid conditions by treatment with desiccated thyroid gradually, but very slowly, normalizes

the amount of delta sleep (Kales et al, 1967).

Chronic renal insufficiency: The sleep of uremic patients is short, fragmented, and disorganized (Passouant et al, 1970). Dialysis or kidney transplants improve sleep somewhat, but do not restore it to normal levels (Karacan et al, 1972). Williams et al (1974) speculate that uremia may be accompanied by irreversible neuronal changes that manifest themselves in abnormal sleep patterns.

Iatrogenic problems: The fact that the chronic intake of hypnotics and/or stimulants occasionally seems to destroy sleep has been discussed on page 29. However, other medications can have similar effects. These effects are often quite atypical or idiosynchratic, as the following case illustrates:

Case History: *Miss T., a 36-year-old nurse, complained that she had developed chronic, severe insomnia following a required three-month service on the night shift. Although she had returned to day work two months previously, her insomnia had not yet abated as had been expected.*

On the assumption that Miss T. had developed some form of behavioral insomnia or a circadian rhythm disturbance, she was treated with behavior therapy for a month, to no avail. In a more careful interview, Miss T. revealed that about three weeks prior to her switch to night work she had been placed on an oral contraceptive, but had failed to mention this in her initial interview.

Although insomnia was not a side effect listed in the Physicians' Desk Reference for this contraceptive, the drug was withdrawn, and Miss T. started to sleep better within a week's time. The contraceptive obviously was related to Miss T.'s insomnia. However, it was unclear whether, in this patient, the insomnia was related to the drug per se, or whether it was related to the psychological implications of taking the medication.

Excessive stimulants: The absolute amount of food intake may have a profound influence on sleep: Insomnia may develop from starvation; excessive sleep from overeating (see page 27). Less obvious, but apparently quite well documented (Bell et al, 1976), is the fact that certain common food allergies also manifest themselves in excessive daytime sleepiness.

Coffee, tea, and cola drinks are now so common in our culture that their stimulant properties are often forgotten. However, both Brezinova et al (1975) and Karacan et al (1975) have demonstrated that coffee disturbs sleep, apparently even in those who claim to be unaffected by it. A careful medical history needs to include a query concerning these issues. Some people habitually drink 20 cups of coffee a day and then complain about their serious insomnia.

Miscellaneous medical problems:

Case History: *Mrs. V., a 52-year-old chronic insomniac, claimed that she could never sleep more than two or three hours per night and that she had suffered from a state of chronic malaise for the past five years. Besides her insomnia, she also complained about vague aches and pains that moved unpredictably from one part of her abdomen to another. Extensive medical, neurological, and endocrine evaluations, done in two reputable hospitals, were essentially normal.*

While being interviewed, Mrs. V. readily admitted to a fair amount of marital stress. In addition, both psychiatric interviewing and psychological testing suggested a hypochondriacal reaction. The only piece that did not fit such a diagnosis was the fact that Mrs. V. actually did sleep very poorly in the lab, showing two hours of fragmented shallow sleep on the first night and four hours on the second, with no delta sleep and very little REM.

According to numerous consultants, the basic cause of Mrs. V.'s problems seemed to be neurotic. Psychotherapy was therefore recommended. However, no progress was made in this treatment, and Mrs. V. soon lost confidence. She then "hopped from hospital to hospital," insisting that her problem must have some medical origin. A few months later, to everyone's surprise, a "median arcuate syndrome" (marked stenosis of the celiac axis secondary to hypertrophy of the arcuate ligaments of the diaphragm) was diagnosed. Once this stenosis was corrected, abdominal pains gradually disappeared, sleep improved, and her "hypochondriasis" vanished.

The above case history demonstrates the fact that almost *any* medical disturbance can cause sleep problems, either directly or through the accompanying

pain and malaise. Occasionally, the discomfort is the result of assuming a horizontal position; in other cases, one only becomes aware of the pain when other, more intense sensations of wakefulness have diminished. Besides pain, fever has also been demonstrated to fragment sleep, causing many awakenings and reducing both delta sleep and REM sleep. Karacan et al (1968) speculate that these sleep changes might be related to a mechanism that involves the arousal system.

Sleep-exacerbated disorders: In these conditions, disorders do not seem to affect sleep but they become worse when a person falls asleep. Williams et al (1974) suggest that these disorders should be classified separately from the sleep disorders secondary to medical problems. They are grouped together in this review mainly for the sake of convenience.

The assumption of a horizontal posture, even without sleep, will cause hemodynamic shifts of potential importance (eg, paroxysmal nocturnal hemoglobinuria, Marks, 1964) as well as profound changes in respiratory performance (eg, nocturnal dyspnea and orthopnea related to congestive heart failure). Other sleep-exacerbated disorders are related to the physiological changes that occur during sleep in general or during specific sleep stages (eg, increase and greater variability in heart rate, respiration, and blood pressure during REM).

In addition to the ones mentioned above, a partial list of sleep-exacerbated disorders includes nocturnal angina and myocardial infarctions (Karacan et al, 1969); nocturnal headaches (Dexter and Weitzman, 1970); nocturnal asthma (Kales et al, 1968); emphysema (Trask and Cree, 1962); night cramps (Simpson, 1969); "tired-arm syndrome" (Ford, 1956), and many other neuromuscular disorders. Williams et al (1974) provide detailed references for many of these sleep-exacerbated problems.

In summary, a very careful medical evaluation is essential if the sleep disturbance is not ameliorated by customary treatment. Preferably, this evaluation should be done by a physician familiar with the sleep disorders. The pathophysiology of sleep is markedly different in many areas from the pathophysiology of wakefulness, encompassing difficulties and disorders that are unknown or unimportant during wakefulness or that cannot be assessed when the patient is in the waking stage.

Sleep Disorders Secondary to Psychiatric Problems

Classified here are disorders in which the sleep disturbance is part of a larger psychiatric problem. Distinctions concerning what might be psychiatric and what might be medical or behavioral are fairly arbitrary at present. Occasionally, the underlying psychiatric problem is fairly well hidden, and the sleep disturbance seems to be the chief complaint, as the following case history demonstrates.

Case History: *Mrs. S., the 48-year-old wife of a local businessman, had been referred to the lab for chronic insomnia after unsuccessful trials with many different hypnotics. At the first interview at the clinic, she sat in the office chair, weak from lack of sleep, with red, bloodshot eyes, but quite in control of herself. She responded appropriately, with a polite, pleasant smile. It developed that she had always been a "fickle" sleeper, but "had managed until last fall." She could not think of any reason why she should not sleep now, and said that for the first time in her life she now had everything she had ever wished for and really should be happy.*

This last statement aroused suspicion. When the interview stalled, Mrs. S. was given a Rorschach test. Her answers were clearly suggestive of a serious but well-hidden depression, even though the referring physician had "ruled out" that possibility. After another hour of interviewing, she finally started to realize that for the first time in her life she had felt increasingly useless. Her youngest son had gone to college two years ago, and some neighbors who had always leaned on her for support had moved away. According to her previously unconscious appraisal of her life, now verbalized for the first time, there was nothing left for her to do other than to wait for old age and death and to bear it graciously.

Mrs. S. was hospitalized for a work-up. Her sleep was recorded in the laboratory and shown to be seriously fragmented and curtailed. Although it fluctuated widely from night to night, Mrs. S. averaged about 35 awakenings per night, and she rarely slept for more than about four hours, often less. Much of this sleep was stage 1, with no delta sleep.

Treatment with a sedating antidepressant improved sleep in the hospital almost immediately. While undergoing therapy, Mrs. S. explored other ways of becoming useful again, and finally became involved in a relatively unknown charity organization for a minority group.

On a follow-up nine months later, Mrs. S. was pronounced "remitted" by all concerned; she was off all drugs and functioned quite well, both at home and in her social life. Five nights of sleep in the lab, however, still revealed some insomnia. Although sleep had improved dramatically when compared with sleep during her hospitalization, she still showed more awakenings, more stage 1 than expected from someone her age, and practically no delta sleep. In addition, relatively adequate nights fluctuated with very poor nights in an apparently random manner. However, Mrs. S. was satisfied. According to her, this was the way she had slept since her early 20s.

With all the new research on sleep disorders, emotional and psychiatric problems still lie at the roots of many sleep disturbances. It is difficult to determine just how large this group is. Figures vary from about 30% of all sleep problems (estimated by a neurologist-sleep clinician) to approximately 85% (estimated by a psychiatrist-sleep clinician). Estimates are made more difficult by the fact that psychopathology and poor sleep potentiate each other. Everything looks worse, more hopeless, and more threatening after a series of sleepless nights. When things look that bad, it becomes progressively more difficult to sleep, and a vicious circle is established.

It should be remembered that a patient who presents a chief complaint of insomnia (when there is evidence of substantial emotional difficulties) has already made an important diagnostic statement about himself. He has difficulties seeing himself as having emotional problems. Not surprisingly, many insomniacs are "repressors" and are unaware of what is going on inside themselves. Psychotherapy for such people differs from therapy for a patient who presents with a chief complaint of emotional distress. Kales and Kales (1973) have commented on the fact that insomniacs need a very firm, direct approach, not unlike the one best used in other "psychosomatic" problems.

According to some clinicians, psychological stress interferes mainly with the process of falling asleep but not with the ensuing sleep. This is simply not true. Many patients under predominantly psychological stress fall asleep quickly, apparently from pure exhaustion. They then wake up often and early, as soon as their excessive psychological tension and arousal overcome a still-very-strong need to sleep. Sleep is not a stuporous coma; there can be a fair amount of mental activity during REM sleep and, especially in poor sleepers, often during NREM sleep (Rechtschaffen and Monroe, 1969).

Depression: Fragmented sleep is a well-known sign of most types of unipolar depression. However, it is important to realize that some seriously depressed patients sleep quite normally (Hauri and Hawkins, 1973) and others sleep for excessively long times during their depressive episodes (Michaelis and Hofmann, 1973). Thus, poor sleep is a frequent but not a universal sign of depression, both reactive and endogenous.

Traditionally, difficulties in falling asleep have been associated with reactive or neurotic depression, whereas frequent awakenings and early-morning insomnia have been related to endogenous depression. Laboratory sleep studies are unanimous in rejecting this claim (eg, Hawkins and Mendels, 1966; Kupfer and Foster, 1973). Although laboratory evaluations cannot objectively differentiate reactive from endogenous depressions on the basis of awakenings, the subjective *complaint* of early-morning

awakenings is statistically associated with endogenous depression (Haider, 1968). Endogenously depressed patients feel worse in the morning, and awakenings at that time may be more offensive to them than to other patients.

Delta sleep is consistently curtailed in most forms of depression. Short and fractured sleep alone is not the only reason for this lack of delta sleep; Kupfer et al (1973) report low-delta sleep even in a group of depressives who slept an adequate amount. However, low delta sleep is *not* diagnostic of depression as has been claimed; it is also seen in uremia, asthma, hypothyroidism, pregnancy, and many other conditions.

Contrary to early reports, REM sleep is *not* curtailed in depression (Mendels and Chernik, 1972). However, a phenomenon unique to serious depression is very short REM latencies in some patients (Kupfer and Foster, 1972). It is not unusual to see certain depressed patients go into a REM period 5 to 15 minutes after the onset of sleep. Coble et al (1976) hope that this shortened REM latency, together with other sleep parameters, might be developed into a psychobiological marker for the classification and treatment of affective disorders.

Although sleep improves dramatically upon recovery from depression, most remitted patients still sleep very poorly nine months after hospital discharge (Hauri et al, 1973). It could be speculated that this poor sleep might index some very basic biochemical problem in "depression-prone" individuals. It also seems possible that patients with a primary sleep disturbance might be at a higher risk for depression than good sleepers, possibly because they cannot "recharge their batteries" during sleep as well as normal sleepers do.

In summary, a clinician will do well to search carefully for signs of depression (either overt or covert) when he is confronted with relatively intractable insomnia. If depression is found or suspected, a vigorous antidepressant regimen will be vastly preferable to treatment with hypnotics, especially if the main dose of a sedating antidepressant can be given at bedtime.

Manic-depressive psychosis: Most patients suffering from bipolar depression (manic-depressive syndrome) sleep more when they are depressed, less when they are manic (Hartmann, 1968). In addition, during depression the percentage of time spent in REM sleep is often increased and during mania it is decreased. However, not all bipolar patients follow this pattern; it appears that the traditionally good sleep in the depressive phase can "flip over" into insomnia in some patients, as it appears to do in unipolar depression.

Occasionally, a patient will complain of suffering from repetitive bouts with insomnia and/or hypersomnia alternating with long periods of normal sleep. If such repetitive bouts cannot be associated with other events (eg, seasonal allergies, "anniversary" depressive reactions), manic-depressive mechanisms might be at play much more often than is the frequently suspected, but indeed very rare, Kleine-Levin syndrome (page 52).

Schizophrenia: Sleep is usually disturbed during any acute psychotic turmoil. Chronic schizophrenics, on the other hand, sleep surprisingly well (Kupfer et al, 1970). In particular, the amount of REM sleep is within normal limits. This was a very disappointing finding, because many had hoped to find a key to schizophrenia in its subjective similarity to the dreaming phase of sleep.

A more careful analysis of REM sleep does show subtle abnormalities in schizophrenic patients. When acutely ill schizophrenic patients are REM deprived, they show *little or no* REM rebound (Zarcone et al, 1975). When schizophrenic patients are REM-deprived after remission of their symptoms, they seem to show an *excessive* REM rebound (Zarcone et al, 1968). Dement (1972) links these findings to a possible serotonergic defect

in the acutely ill schizophrenic patient, but the basic mechanisms for this possibility are not known.

Alcoholism: Millions of Americans take a glass of red wine or some other drink before going to bed, claiming that it helps them sleep. What is the evidence? Small doses of alcohol do relax tense people, helping them to fall asleep more easily; however, sleep after alcohol intake is disturbed (more awakenings, more sleep-stage changes). Alcohol also depresses REM sleep, with a compensatory increase of REM during withdrawal. Delta sleep seems to react inversely: more delta during intoxication, less during withdrawal (Johnson et al, 1970; Gross et al, 1971). Depending on the dose and on the speed of alcohol metabolism, a compensatory REM rebound might occur either later that night or during the next night (Rundell et al, 1972).

Chronic alcoholic patients show a "prematurely aged" sleep (Johnson et al, 1970) manifested in many awakenings, many stage changes, little or no delta sleep, and usually somewhat-decreased REM sleep. Because sleep in chronic alcoholic patients has become fragmented and shallow, total time in bed is usually increased. The sleep-wake rhythm is blurred, and the chronic alcoholic patients often show excessive daytime sleepiness and take numerous naps (Mello and Mendelson, 1970). During heavy alcoholic binges, sleeping becomes almost totally abolished, and there is the possibility that delirium, the cardinal feature of delirium tremens, might be mainly the result of prolonged, almost total sleep deprivation (Bates, 1972). Others have speculated that delirium tremens might be a breakthrough of the REM mechanisms into wakefulness after prolonged suppression of REM during alcoholic bouts (Greenberg and Pearlman, 1967).

Insomnia is a frequent, but not universal, correlate of alcohol withdrawal. Similarly, the REM rebound after alcoholic bouts seems related to individual differences. Some show almost continuous REM sleep after abrupt withdrawal from high alcohol intake (Greenberg and Pearlman, 1967), and others show little or no REM rebound (Gross and Hastey, 1975).

Even after one or two years of total abstinence, the sleep of many alcoholic patients remains disturbed. Delta sleep is low, sleep is still excessively fragmented, and alcoholic patients may continue to have problems falling asleep (Adamson et al, 1973). It is unclear how much of this sleep disturbance is related to permanent and irreversible damage caused by the alcoholism and how much of this poor sleep was there all along. Many insomniacs feel driven to alcohol in an attempt to self-medicate their sleep problems.

Neurotic problems: Neurotic patients often have severe insomnia. Occasionally a sleep problem is the main, if not the only, complaint of such patients. Unfortunately, nobody has extensively compared the different types of neurotic sleep problems in the laboratory. This is surprising, because the various types of neurotic disorders probably constitute a large proportion of all insomniacs.

When clinically important psychiatric problems are associated with insomnia, these should be treated first. The sleep problem will then often disappear. If it does not, it seems likely that during the course of the psychiatric problem some "maladaptive sleep behaviors" have been established, which can be treated as discussed in the next section.

Sleep Disorders Secondary to Behavior Problems

Behavioral factors apparently play a role in many sleep disorders including enuresis and depressive "escape into sleep." However, such factors have been studied most thoroughly with regard to insomnia, and therefore shall be discussed here mainly as they apply to poor sleep.

Behaviorally based insomnias are obviously not as dramatic or life-threatening

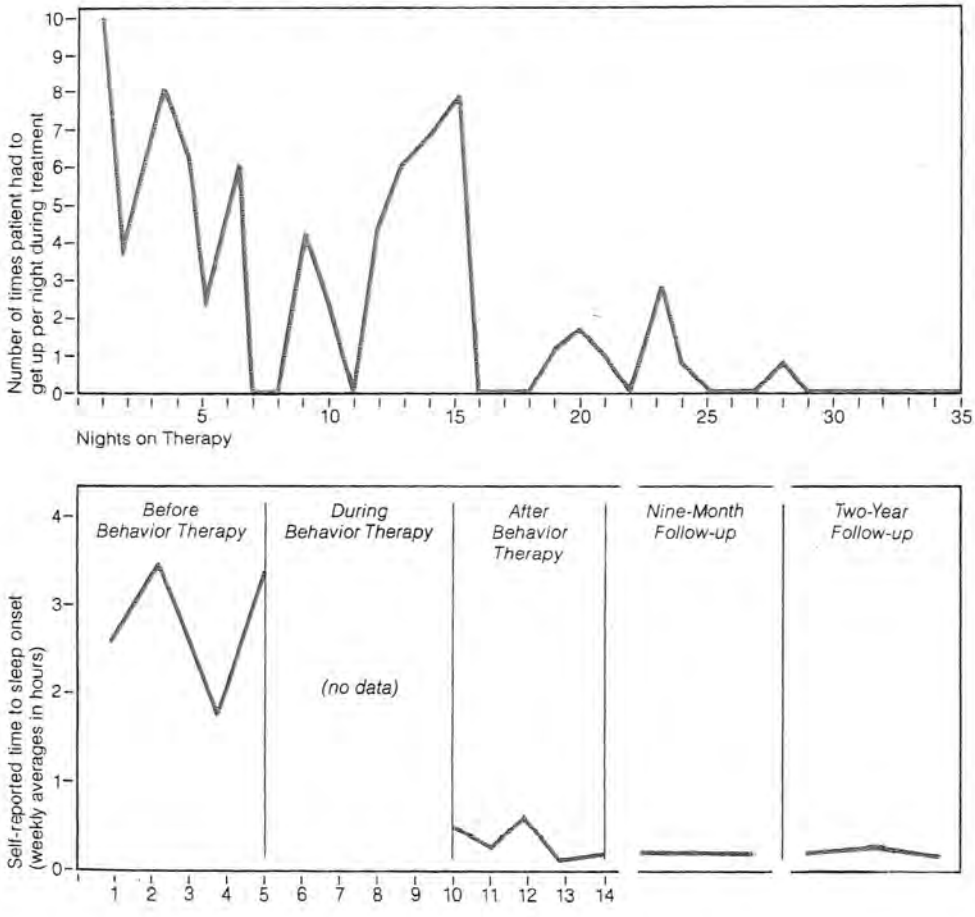
as those based on medical conditions such as sleep apneas. However, it appears that the large bulk of insomnias are related to behavioral problems. In addition, these behavioral insomnias, being essentially based on faulty learning, are particularly resistant to the customary treatments by medication and/or psychotherapy.

Conditioned insomnia:

Case History: *Mr. U. was a 19-year-old college student who usually took two to three hours to fall asleep each night. He dated his problem to the age of 13, when his parents finally divorced after a long period of continuous fighting, yelling, and occasional homicidal threats. During that period of his life, Mr. U. usually had lain awake in bed until the fighting had stopped. Insight-oriented psychotherapy for the past two years had not helped him to fall asleep more easily. Indeed, his therapist finally felt that Mr. U. was a "remarkably healthy" person, given the extensive family difficulties that had marred his childhood. Similarly, sleep in the lab turned out to be normal, except for a sleep latency of about 90 minutes each night. Nevertheless, friends corroborated his story that at home it usually took him two to three hours to fall asleep..*

When Mr. U. was asked to describe his best night of sleep during the past year, he related, somewhat perplexedly, that it had occurred on the side of a mountain. While climbing one day, he and a group of friends were overtaken by darkness before reaching the summit, and were

Figure 15. **Performance of a Sleep-Onset Insomniac (Mr. U.) Undergoing Stimulus-Control Behavior Therapy**



forced to spend the night on a very narrow ledge tied to the rocks. To his utter amazement, Mr. U. fell asleep almost immediately and spent an excellent night in this uncomfortable situation. Mere fatigue from climbing could not have played a major role in his good sleep, because he was a well-trained athlete and had often tried to improve sleep by exercising extensively. More likely, it appeared that all the stimuli surrounding his usual sleeping arrangement at home had been absent on the rocky ledge, and that this might have been related to his excellent sleep.

Mr. U. was then treated with the stimulus-control, behavior therapy to be described below. In the beginning, he had to get up eight to ten times per night. Nevertheless, within a five-week period the insomnia was relieved, and Mr. U. was able to fall asleep within 10 to 20 minutes on at least six out of seven nights. Follow-up data nine months later, and again two years later, showed that his insomnia had disappeared except for occasional stress-related poor sleep.

How can Mr. U.'s inability to fall asleep be understood? Assume that a person who is already sleeping somewhat marginally is put under severe stress (eg, the death of a loved one, the divorce of one's parents). It would then seem normal not to sleep well for a period of time because of the excessive emotional turmoil. Not infrequently, by the time the stress is lifted, the whole precedure of lying down in bed, turning off the light, etc., has become associated with (conditioned to) frustration and sleeplessness. Even if the original stress is now removed, the stimuli in the bedroom have been associated long enough with frustration and tension that they trigger these emotions by themselves. Occasionally reinforced by naturally occurring poor sleep, such a conditioned insomnia can maintain itself for decades, long after the original stress is gone.

If conditioning is an important factor in a person's insomnia, almost any change in sleeping arrangements initially improves sleep. People with this problem often sleep better away from their own beds, eg, in hotels or on the couch in the living room, and they are often helped by unusual sleep arrangements such as water beds or hammocks. These people frequently sleep quite well in the clinic because the stimuli associated with poor sleep at home are not present in the lab.

An efficient way to overcome conditioned imsomnia is a procedure called "stimulus-control behavior therapy." Basically, the goal of this therapy is to associate the bedroom stimuli with rapid sleep onset again, rather than with frustration and arousal. To achieve this, the patient is told that he is "misusing" his bed if he lies in it awake and frustrated. Bootzin, the behavior therapist who initiated this approach (1973), recommends the following set of rules:

1. Lie down in bed only when sleepy.
2. Use the bed only for sleeping; do not read, watch television, or eat in bed. Sexual activity is the only exception to this rule, and on such occasions the sleep instructions are to be followed afterwards, when you intend to sleep.
3. If unable to fall asleep, get up and go into another room. Stay up until you feel really sleepy, and then return to the bedroom to sleep. Get out of bed again if sleep does not come easily. Remember, the goal of this procedure is to associate the bed with falling asleep quickly.
4. If sleep does not come, repeat step 3 as often as is necessary throughout the night.
5. Set the alarm and get up at the same time every morning regardless of how much you slept during the night. This will help the body acquire a constant sleep-wake rhythm.
6. Do not nap during the day.

If the above rules are followed, there will be almost no sleep during the first night. By the second or third night, the patient is so tired that he may fall asleep on the first or second attempt. Things then fluctuate for a few weeks, but gradually the bedroom surroundings again become associated with sleep rather than with arousal and frustration. However, most patients need a lot of encouragement during this reconditioning period. This means at least twice-weekly

therapist contacts and occasional phone calls. The daily graphing of sleep behavior also helps, because the temptation to drop out of the program is reduced when there is objective evidence of progress (see Figure 15).

In a sample of unselected insomniacs, Bootzin (personal communication) had about a 60% success rate with this method. It seems likely that a much higher success rate could be established in a sample of patients preselected for signs of conditioned insomnia.

Internal arousal (trying too hard):

Case History: *Mrs. O. was a 23-year-old mother-to-be who came to the office in a state of near exhaustion two weeks before her projected time of delivery. She claimed that she had not slept a wink for at least three weeks, and her appearance supported this claim. She stated that all her life she had looked forward to the natural birth of her first baby and had enrolled in a Lamaze class just as soon as she learned that she was pregnant. However, recently her teacher had stated that in order to be ready for birth, the prospective mothers had to get plenty of rest and sound sleep. Then, "out of the blue," this insomnia had struck Mrs. O., and she exclaimed "now the whole experience will be spoiled!"*

Although sleep is usually poor in the last trimester of pregnancy, this was obviously not the whole problem in the case of Mrs. O. Therefore, she was asked to recline on the couch and was instructed in a series of Jacobsonian relaxation exercises. These exercises had been tape recorded, and Mrs. O. fell asleep almost before the tape ended.

Mrs. O. was given the tape for home use and told to perform these relaxation exercises anytime she wanted to sleep. In addition, she was told that the deep rest that would overcome her when doing these exercises would be almost as good as true sleep in terms of preparing her for natural childbirth. Because an adequate performance at the birth of her first baby was now no longer threatened by her inability to sleep, she no longer tried so desperately to fall asleep. The insomnia, created by an inadvertent remark of the Lamaze teacher, evaporated within two nights.

Sleep is one of the few things in life that cannot be improved upon by trying harder. Rather, any attempt to force sleep will inevitably result in increased activity in the arousal system, thus abolishing sleep. Trying too hard is a common error in many insomniacs, especially because many still think that they need a good eight hours' sleep per night in order to function the next day. Some information concerning the experiments on sleep need and sleep deprivation (pages 15, 18) is often helpful to such insomniacs.

Occasional "trying too hard" is difficult to avoid. However, when this becomes a habit it can cause serious insomnia. The mechanisms involved in such cases are then quite similar to the ones discussed for conditioning, except that the association is to internal thoughts, not external stimuli. Assume that a person is not sleeping well for a valid reason. After he has been sleepless for a number of nights, sleep becomes an overriding concern, and soon a vicious circle is established. He desperately needs to sleep, tries hard to sleep, and therefore cannot sleep. Later, the mere thought of sleep may cause almost phobic reactions.

The presence of internal arousal as a contributing factor in insomnia is established if the patient complains that he can fall asleep easily when he does not want to – in lectures or in front of the TV – but cannot sleep when he tries to do so. In the laboratory, the clinician will occasionally ask such people to stay awake for 30 minutes in order to obtain a valid comparison record. So instructed, these patients often fall asleep almost immediately. After 30 minutes, they are called over the intercom and told that it is now time to sleep. This instruction immediately results in a dramatic increase in muscle tension and causes an alerting response on the EEG and, occasionally, a sleep latency of three to four hours.

To date, there is no adequate treatment for internal arousal. Electromyographic biofeedback has been advocated, as have other forms of relaxation training, such as self-hypnosis and systematic desensitization. A number of published case reports attest to the occasional efficacy of these techniques, but solid, large-scale studies are yet to be conducted.

However, it has been repeatedly demonstrated that insomniacs as a group do not show greater muscle tension than do good sleepers (Good, 1975).

One might speculate that, if relaxation techniques work in this group, they do so mainly by distracting a person's attention from trying to sleep and *not* by their inherent ability to relax the musculature. Other methods not related to relaxation seem potentially as effective, such as the proverbial counting of sheep or the very careful and painstaking drawing, in one's own mind, of all numbers between 0 and 100 on large, black sheets of imaginary paper.

The matter of trying to force sleep is basically an attitudinal problem. Therefore, it will eventually be solved by psychological methods involving attitudinal change, an area still very much in need of solid research.

What should be done when one cannot sleep? If a person is comfortable and unconcerned, relaxing in bed seems best. However, when the insomniac starts to become frustrated, angry and upset, he should get up and engage for awhile in a quiet hobby or some other pleasant, nonstimulating task instead of lying in bed. If he continues to lie in bed while frustrated, he is in danger of developing insomnia that is conditioned to the bedroom stimuli.

Should one read in bed until one gets tired? The answer seems to be "yes," if the reading material is not too stimulating and if the morning arousal time remains fixed. Without a fixed arousal time, reading in bed will eventually lead to sleep-phase changes described on page 12. In many cases, reading a mildly pleasant but unexciting book will keep patients from trying too hard to sleep, and drowsiness will finally overtake them.

It may be noted that Bootzin forbids his patients to read in bed, although reading in bed is encouraged here. The main difference lies in the purpose of the treatment: if the problem is "trying too hard," then the patient might be instructed to stay in bed and read; if the problem is a conditioned association between bed and frustration, then the preferred therapy might be to suggest that the patient get out of the bedroom and go elsewhere to read.

Bedtime rituals such as brushing teeth and saying prayers seem to work on a similar principle. Good sleepers often become drowsy just as soon as they complete the routine. The close temporal association of bedtime rituals with frustration and arousal in insomniacs, however, is counterproductive, and poor sleepers should be asked to develop entirely new bedtime rituals.

Disturbance of the sleep-wake rhythm: The massive effects of irregular bedtimes or of staying in bed too long have been discussed on pages 10-12. It is obvious that for weak sleepers regular sleep-wake patterns are mandatory. In particularly susceptible patients, irregular or excessively long time spent in bed may result in a variety of physiological and endocrine disturbances that may make sleeping difficult. Therefore, disturbances of the sleep-wake rhythm might well have been discussed under sleep disorders secondary to medical problems. They are included here mainly because their treatment (ie, regular and limited time in bed) involves behavioral methods. Pure admonitions to regularize time spent in bed are usually as ineffective as orders to stop smoking or stop overeating. Rather, the same techniques useful for the treatment of overeating or smoking can be used as well for regularizing time in bed. These techniques involve such strategies as reinforcement of scheduling, aversive conditioning for oversleeping, group support, and graphing of sleep-wake behavior.

Self-image; sleep phobias: Most human beings occasionally have poor nights of sleep. For good sleepers, these nights are merely unpleasant irritations, not worthy of much thought. They know that they will sleep well soon. For persons who have labeled themselves as insom-

niacs, however, a poor night of sleep reaffirms this label. In their minds it demonstrates once more that they are inferior, and it raises again the spectre of the repetition of many consecutive, sleepless nights that they have experienced. Knowing how painful that would be creates fear and panic in the poor sleeper, leading to a self-fulfilling prophecy. Just as the "dry" alcoholic cannot take that first drink for fear of starting another alcoholic bout, so the insomniac cannot afford one sleepless night for fear of starting another series of sleepless nights. Elaborate precautions are therefore taken, a phobia of sleepless nights develops, and the problem becomes aggravated by the very means that were designed to avoid it.

Phobias are often treated either with systematic desensitization or with implosive therapy. In the former, relaxation and the presentation of a graduated hierarchy are used to help a patient face the thought of a sleepless night without fear and arousal. In implosive therapy, the patient is usually kept in the phobic situation until all signs of autonomic arousal vanish. Unfortunately, behavioral therapists have been extremely slow in exploring behavioral treatment of insomnia, and much basic research in this area still needs to be done.

The various behavioral mechanisms discussed in this section are best understood as contributing and interacting factors, not as separate disease entities. Cases of "pure" conditioned insomnia or "pure" sleep-wake rhythm disturbances are very rare. Rather, in almost every case, the behavioral factors often aggravate psychiatric problems (eg, when conditioning aggravates the insomnia of depression) or they may interact with primary sleep disturbances (eg, when an inherently weak sleep system is combined with a pattern of irregular time spent in bed). In such cases, the various factors contributing to a person's sleep problem should be assessed, especially in terms of amenability to change. If one single factor that is contributing to the total insomnia can be changed, sleep often improves and this gives the individual renewed confidence in his or her ability to sleep, thereby further improving sleep.

Parasomnias

According to Williams and Karacan (1974), parasomnias are defined as activities such as walking or urinating which, although normal for wakefulness, create problems when performed during sleep.

Most of us have experienced short periods of confusion when awakened abruptly. Such periods of disorientation occur most frequently when one is awakened from delta sleep. When partially aroused from such sleep, most of us seem quite capable of carrying out purposeful behaviors such as trips to the bathroom, adjusting bedcovers, closing windows, or turning off a bedside alarm without later remembering these acts.

Surprisingly, somnambulism, nightmares, and, to a lesser extent, enuresis, are often related to this partial arousal from delta sleep (Broughton, 1968). Rarely are they related to dreaming, as had been suspected. It seems that some especially susceptible individuals occasionally do not rise to full consciousness when disturbed during delta sleep. Rather they enter a state of confusion, indicated on the EEG by a mixture of delta (deep sleep), alpha, and beta (waking) waves. Somnambulism, nightmares, and enuresis may occur in this confusional state.

The concept that most parasomnias might be manifestations of incomplete arousal from delta sleep explains the following observations:

1. Parasomnia occurs more often in *children*, who have more delta sleep and, possibly, somewhat less-established boundaries between full wakefulness and sleep than have adults.

2. Such activities usually occur *early* in the night, when delta sleep is most prominent.

Table IV **Checklist for the Work-Up of an Insomniac**

Questions	Actions
A. Are there possible medical reasons for the insomnia?	
1. Could endocrine dysfunction, pain, neurological problems, allergies, or other problems cause the poor sleep?	*thorough medical work-up*
2. Could a problem such as sleep apnea, nocturnal myoclonus, or sleep epilepsy be present?	*interview bed partner. If positive indications are found (ie, snoring, twitches), observe sleeping patient or refer to a sleep clinic. Remember that these problems are often episodic; one normal observation does not rule them out.*
3. Could the problem be related to medications or to excessive amounts of stimulants (eg, coffee, chronic use of hypnotics, or some other medications)?	*gradual withdrawal from the offending medication.*
B. Are there treatable psychiatric components?	
1. Does the insomniac suffer from any recognized psychiatric syndrome, such as depression, schizophrenia, or alcoholism?	*appropriate psychiatric treatment, both pharmacological and psychotherapeutic.*
2. Is the insomniac overstressed because of maladaptive personality traits such as low self-esteem, inability to say "no," need for excessive power, or because of excessive environmental demands?	*psychotherapy, job or marital counseling, environmental interventions.*
C. Are there treatable behavioral components?	
1. Is the patient either muscularly or psychologically tense or anxious?	*relaxation training (ie, EMG biofeedback, meditation) coupled with adequate exercise and/or tranquilizers.*
2. Does the patient show signs of learned insomnia (ie, sleeps well anywhere but in his own bed, sleep phobias, excessive arousal when thinking about sleep).	*behavior therapy*
3. Is the 24-hour rhythm disturbed or atypical?	*attempt to regularize waking and sleeping times.*
D. What else could it be?	
1. Does the problem occur every 1½ to 2 hours?	*problem might be related to REM sleep*
2. Does the patient complain of poor sleep, but his family claims the patient sleeps long and well?	*wonder about pseudoinsomnia, nonrestorative sleep, or hypochondria.*
3. Is the patient functioning well during the day, even on very little sleep?	*patient might have a healthy hyposomnia.*
4. Has the problem existed since birth?	*patient might have primary insomnia.*

3. Different forms of parasomnia often occur in the same person.

4. Patients are difficult to arouse when engaged in some form of parasomnia, possibly because they are then in a confusional state rather than in normal sleep.

5. Because they rarely fully awaken during their parasomnias, patients rarely recall these incidents in the morning.

Somnambulism (sleepwalking):

Case History: *Mr. S., a 34-year-old math teacher, sought help because of his extreme difficulties in falling asleep and because he was sleepwalking one to three times per week. Usually, these somnambulistic episodes were harmless: Mr. S. got up about one hour after he fell asleep, rummaged around for awhile, then went back to sleep. He often slept the remainder of these nights in some unpredictable location, such as the living room couch, the kitchen floor, or the bathtub. However, Mr. S.'s sleepwalking episodes were not always that benign; he occasionally urinated on the living room carpet (apparently thinking that he was in the bathroom), had severely hurt himself when stumbling over furniture, and twice had been found trying to climb out of a window in the couple's fourth-story apartment. His wife was terrified of these sleepwalking episodes because she couldn't awaken him, and she was not strong enough to control him physically.*

In the lab, Mr. S. did not sleepwalk but he showed extreme muscle tension, both during the day and when trying to sleep. He was anxious, nervous, and claimed that he was barely keeping up with his teaching job and that the students were driving him "insane." Neither tranquilizers nor sleeping pills had helped in the past. A clinical EEG and a thorough physical work-up were entirely within normal limits.

For the sake of safety, the couple was advised to move to a ground-level apartment. Mr. S. was enrolled in intensive psychotherapy and exchanged his teaching job for a less-demanding clerical one. Furthermore, he learned deep-muscle relaxation through EMG biofeedback. Finally, door locks and window locks were installed in selected locations so that Mr. S. could only sleepwalk through the bedroom, hall, and bathroom. From these areas, all potentially dangerous objects were removed. His wife kept the keys in her custody in case of fire.

Sleepwalking episodes first increased to four or five times per week as all these changes were made. However, six months later, sleepwalking episodes were down to two or three per month, and now, two years later, Mr. S. sleepwalks no more than two or three times a year. It appears that the excessive tension from teaching resulted in some psychological problem and so aroused this person that even during sleep he was highly "keyed up."

As discussed above, sleepwalking is often a disorder of arousal, an inability to awaken completely when disturbed in delta sleep. It is not the acting out of a dream, and the EEG during sleepwalking shows a mixture of delta and high-amplitude alpha waves (Jacobson et al, 1965). In this confusional state, relatively simple behavior patterns can be carried out, but the senses are dull; higher brain functions are erratic, and coordination is poor. Reports of exquisite balancing acts on roof tops and window ledges are exaggerated, and somnambulists have fallen to their deaths after mistaking a window for a door.

Somnambulism in children is quite common and of relatively little concern. Episodes of sleepwalking can even be triggered in most children by lifting them to their feet during delta sleep (Kales et al, 1966). However, there is some evidence that adult somnambulists often suffer from psychological problems amenable to psychotherapy and should be referred to a therapist for treatment. Although rare, sleepwalking may also be associated with temporal lobe epilepsy or other CNS abnormalities that are occasionally difficult to diagnose except by nasopharyngeal EEG leads after sleep deprivation (Guilleminault, personal communication).

If epilepsy seems involved, sleepwalking may be abolished with appropriate medication (phenytoin, carbamazepine). In other cases, either diazepam or imipramine has been reported as being effective against somnambulism. However, many cases are not treatable by medication. The main concern in these cases is the safety of a patient during his walks. As was noted in the case history,

this involves requiring somnambulists to sleep on the ground floor, removing potentially dangerous objects before they go to bed, locking windows and doors, and depositing the keys with someone who could open the doors in case of fire or other emergency. Some somnambulists claim success by tying a rope loosely around their waist and around a bedpost. When they start sleepwalking, the jerk from the rope awakens them fully, assuring that they will neither harm themselves nor scare others. However, others soon learn how to untie the rope before embarking on an episode of sleepwalking.

Nightmares and night terrors:

First case history: *Marc appeared to be a healthy 10-year-old who wanted to go to summer camp. However, his parents hesitated because three or four times a week Marc woke up screaming, sweating, and wildly flailing his arms. This usually happened about the time his parents were getting ready to go to bed. They never could find out what terrified their son, although occasionally he mumbled something about "monsters." Marc usually calmed down within a few minutes and rarely remembered his night terrors in the morning. The parents were mainly concerned that his bloodcurdling screams would awaken the entire camp. They had also noted that Marc's night terrors increased whenever he went through some new adjustment or stress, and they were afraid that camp might trigger such a period of increased night terrors.*

After extended psychological testing, the parents were reassured that nothing seemed dramatically wrong with Marc. It was then found empirically that 20 mg of imipramine h.s. suppressed Marc's night terrors quite effectively. This was the dose given to Marc in camp, and he had only one nightmare during the four-week camp session.

Night terrors reappeared, although with diminished frequency, when the imipramine was withdrawn in the fall. Later, Marc's enlarged adenoids were removed for reasons entirely unrelated to his nightmares. This, too, seemed to have a beneficial effect. Night terrors decreased to one or two per month, reappeared dramatically when he was transferred to a new school, but then disappeared completely.

Second case history: *Rose was a high school senior planning to go to college. However, she was afraid that her repeated nightmares might expose her to ridicule in the dorm. Two or three times per week Rose awakened early in the morning, fearful and agitated. Although she never screamed, she thrashed around in bed, moaning and groaning and awakening her sister who slept in the same room.*

Rose could remember her nightmares quite well: all of them dealt with strangers lurking behind buildings, chasing her, grabbing, and attempting to sexually assault her.

Psychological testing suggested that Rose was an easily alarmed and somewhat immature teenager starting to rebel against an excessively overprotective family situation. When asked what she would do if episodes similar to the ones in her dreams should occur in waking life, she had no answer. She vaguely mentioned that she might scream, but the men in her nightmares always went for her throat, thus making such a defense impossible.

Based on her psychological testing, Rose was referred for short-term psychotherapy. As part of this treatment, the therapist suggested that she should always carry a strong hatpin with her and quietly slip it into her hand should she notice suspicious characters. Then, in reality as well as in her dreams, she was to stab the would-be molester as soon as he covered her mouth. This would at least give her time to scream.

Rose had two more nightmares when she "forgot" that she had a hatpin. As part of assertiveness training, the hatpin scene was rehearsed repeatedly in therapy; a week later she emerged victorious from a number of her nightmares, enjoyed her victories, and the nightmares disappeared.

These case histories illustrate two very different types of frightening dreams: the night terror, usually associated with delta sleep (illustrated by Marc), and the nightmare, usually a REM dream "gone astray" (illustrated by Rose). Delta night terrors occur early in the night, are accompanied by extreme physiological arousal (ie, increased heart rate, sweating), and patients rarely remember more than fragments of their content (Fisher et al, 1973). Classical delta sleep night terrors appear to arise from the confusional states, as discussed above. They occur more frequently when a patient is under stress, apparently because stress causes more arousal from any form of sleep. However, the stress is usually not *directly* related to the content of the night terrors, although the fragments occasionally recalled in these situations obviously

are fabricated by the sleeper and therefore relate to his or her general concerns (Kahn et al, 1973).

Although delta sleep night terrors are usually disorders of incomplete arousal, they may be the first clear signs of other problems, such as temporal lobe epilepsy or sleep apnea. Indeed, the case history of Marc is quite typical for children who suffer from sleep apneas. Whether or not Marc actually did suffer from an upper airway obstruction is now impossible to ascertain, because he improved long before the time when sleep apneas were known to exist in children.

Unlike the night terrors of delta sleep, the nightmares of REM sleep are essentially frightening dreams. Because REM sleep is more abundant toward morning, REM nightmares usually occur in the early-morning hours. Physiological arousal is much lower during REM nightmares than during the delta night terrors, and REM nightmare sufferers usually have long and detailed recall of their nightmares.

It seems important that delta night terrors be carefully distinguished from REM nightmares, because treatment is quite different. Delta night terrors are often abolished by diazepam (2 mg to 5 mg for children, 5 mg to 20 mg for adults), possibly because this drug abolishes delta sleep (Fisher et al, 1973). Occasionally, when the delta night terrors are associated with epileptic phenomena, methylphenidate becomes the treatment of choice. On other occasions, these nightmares can also be abolished by eliminating the arousal stimuli (eg, Marc's swollen adenoids) that might cause distress and disturb delta sleep. Psychotherapy for delta sleep night terrors is rarely indicated.

REM nightmares are more directly related to psychological conflict and psychopathology in general than are delta night terrors (Feldman and Hersen, 1967; Hersen, 1971). Psychotherapy and behavioral treatment seem to be effective treatment methods for REM nightmares, but medication alone is rarely appropriate.

Enuresis: Bedwetting is more common than is often assumed. The incidence of enuresis in apparently healthy navy recruits was 1% to 3% in both the American and British navies during World War II.

"Primary enuresis" means that since infancy the patient has never been consistently dry for at least one period of several months. "Secondary enuresis" means that bedwetting has reappeared after at least one dry period. According to Oppel et al (1968), 10% of all children at the age of seven and 3% at the age of 12 are still primary enuretics.

Primary enuresis, often a familial problem, suggests organic or medical reasons for the enuresis, such as urethral obstructions in males and ectopic urethra in females. Psychological factors, on the other hand, are more often related to secondary enuresis, although anybody who still often wets his bed during adolescence or adulthood should be given a careful physical examination. There are cases where sleep studies may be necessary to show the relationship of nocturnal enuresis with nocturnal epilepsy, sleep apnea, or other sleep disturbances.

Although enuretic episodes can occur in any sleep stage or even when the child is awake, many enuretic episodes (such as night terrors and sleepwalking) begin in delta sleep and therefore occur relatively early in the night. According to Gastaut and Broughton (1964), enuretic episodes usually start with a series of bladder contractions during delta sleep, and they are often associated with a general body movement. The sleep pattern then changes to stage 2 or 1 during micturition, or the patient may wake up. Similar to the other disorders of arousal, bedwetting does increase in frequency when the child is under stress, possibly because psychological stress causes more arousal during sleep and, with it, more opportunity for bedwetting. However, the act of wetting the bed during delta sleep usually carries little symbolic signifi-

cance (ie, it usually does not mean that the child is particularly upset with mother or is trying to punish the parents). On the other hand, there are some rare children who consciously wet the bed while awake, and the meaning of that action might be more open to psychological interpretation.

Imipramine is quite effective in reducing enuretic episodes, especially after careful titration of the dosage to the individual child. Because patients often relapse when imipramine is discontinued, the drug alone is rarely the treatment of choice in enuresis, but its occasional use by both child and adult enuretics may allow normal social activity (eg, summer camp, visits to friends).

A number of behavioral techniques seem effective if enuresis persists into later childhood. Foremost among them is the bell-and-blanket technique, where a bell awakens the sleeper immediately after micturition. For this technique to be successful, a very sensitive adjustment of rewards, punishments, and family attitude is necessary. A qualified behavioral therapist should be consulted before embarking on conditioning a child with this method.

Table V **Procedures Used in Bladder Training**

1. Upon returning from school, have the child hold his urine as long as possible.
2. To stretch the bladder, have the child drink a lot of fluids while practicing retention.
3. After the child has held the urine as long as possible, collect it in a vessel marked in ounces.
4. Every day, record on a calendar the number of ounces of urine the child was able to retain. Indicate with a check mark if the bed was wet that morning.
5. To divert the child's attention during the training session, enlist the family in reading or playing with him.
6. Once the child has attained some bladder control, encourage him to start and stop the urine stream. Start this type of training early in the morning or just before bedtime, then extend to other times of the day.

(Adapted from Starfield B: **Clinical Pediatrics**, Vol 11, pp 343-350, June 1972).

Based on the fact that bladder capacity in enuretics is smaller than in normals, Starfield (1972) suggested "bladder training." A child is asked to hold his urine longer and longer in the hope that increased bladder capacity will carry him through the night (see Table V):

Nocturnal bruxism: Excessive tooth grinding during sleep is not only annoying to the bed partner but also often leads to severe wear on the teeth. Although tooth grinding can occur during any stage of sleep, it seems related to a general lightening of sleep, often occurring after an alerting response has been observed in the EEG (Reding et al, 1968).

To date, there is no direct treatment for tooth grinding, but some dentists prescribe rubber tooth guards to be worn at night to prevent abrasion. Daytime bruxism has been successfully treated with biofeedback, and there is some evidence that such treatment may carry over into the night.

Violent rhythmic movements during sleep: Infants, toddlers, and occasionally an older child sometimes engage in violent, rhythmic head banging or body rocking (jactatio capitis nocturna), especially just before falling asleep. This appears to be a normal "comfort habit," like thumbsucking, that is exacerbated by stress. A nine-year-old boy recently studied at the Dartmouth Sleep Clinic rocked more than 50% of his time in bed on the first night, both while awake and when deeply asleep. Rocking subsided over the next few nights in the sleep lab and it lasted no longer than five minutes at sleep onset on the third night when he was more comfortable.

If head banging or body rocking persists into later childhood, experts disagree on whether it should be treated, and if so, how. However, when the rhythmic movements persist into adulthood, a neurological consultation is indicated.

Concluding Comments

It is hoped this review will enable physicians to more effectively deal with most sleep problems. However, in especially intractable cases, referral to a sleep disorders center might be indicated. Although all-night polysomnography is expensive, the costs of a sleep evaluation must be balanced against the costs of chronic, potentially inappropriate treatments and evaluations and against the discomfort, misery, and potential health hazard of undiagnosed sleep disturbances.

In 1971, there were only three or four sleep clinics in the United States evaluating patients suffering specifically from undiagnosed sleep disorders. Now there are more than a dozen in the U.S. alone. Most are highly professional, ethical establishments attempting to apply the wide range of current knowledge to problems of human sleep. However, the apparent success of sleep disorders centers has also attracted some so-called sleep experts who possess neither training in this field nor the necessary laboratory technology to properly evaluate sleep disorders. To deal with this problem, the Association of Sleep Disorders Centers has recently been created to establish and maintain high standards in each member clinic. Before engaging in expensive tests, one might investigate whether a potential sleep clinic is affiliated with this association.

The next advances in our understanding of sleep pathology must come from those professional and ethical clinics that have enough patients, clinical resources, and technical skills to crystallize new insights into sleep pathology. Unfortunately, animal models are rarely applicable in this work. Man is often the only suitable subject, and this slows the pace of research and the types of experimentation that can be done. Still, in the future, we can envision a more complete understanding and treatment of highly specific sleep problems, whether through drugs, other medical procedures, or individualized and specific behavioral treatment.

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